FELINE HYPERTHYROIDISM

Our mission is to redefine and elevate your pet's health through collaborative, minimally-invasive, and compassionate care that supports the well-being of your pet and peace of mind for your family.

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Feline Hyperthyroidism

Incidence: Most common endocrine disease
Diagnosis: TT4; fT4ED
Treatment: Tailored to patient
Monitoring: TT4; renal disease

Feline Hyperthyroidism

Etiology

Clinical Signs

Diagnostic Testing

Treatment Options

Dietary Option (Hill's y/d)

Renal Disease and Hyperthyroidism

Asymptomatic Cats

Feline Hyperthyroidism

Etiology

- Genetic factors
  - Decreased expression of $G_{(i)}$
  - Over expression of c-ras
- Environmental factors
  - Polybrominated diphenyl ethers (PBDEs and bisphenol A)

Endocrine-disrupting chemicals and the regulation of energy balance

Angel Nadal[1], Ivan Quesada[1], Eva Tudurí[2], Rubén Nogueiras[2,3] and Paloma Alonso-Magdalena[1]

Abstract | Energy balance involves the adjustment of food intake, energy expenditure and body fat reserves through homeostatic pathways. These pathways include a multitude of biochemical reactions, as well as hormonal cues. Dysfunction of this homeostatic control system results in common metabolism-related pathologies, which include obesity and type 2 diabetes mellitus. Metabolism-disrupting chemicals (MDCs) are a particular class of endocrine-disrupting chemicals that affect energy homeostasis. MDCs affect multiple endocrine mechanisms and thus different cell types that are implicated in metabolic control. MDCs affect gene expression and the biosynthesis of key enzymes, hormones and adipokines that are essential for controlling energy homeostasis. This multifaceted spectrum of actions precludes compensatory responses and favours metabolic disorders. Herein, we review the main mechanisms used by MDCs to alter energy balance. This work should help to identify new MDCs, as well as novel targets of their action.

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The aetiologies of obesity, type 2 diabetes mellitus (T2DM) and the metabolic syndrome are multifactorial, and both genes and environment have key roles[1,2]. Environmental factors, such as food intake and exercise, are acknowledged as regulators of energy balance. However, over past years, chemical pollutants have also been demonstrated to potentially have an important role in the aetiology of metabolic diseases. Endocrine-disrupting chemicals (EDCs) are special types of pollutants defined as "an exogenous chemical, or mixture of chemicals, that interferes with any aspect of hormone action" (REF. 3). In cellular and animal models, exposure to a series of EDCs has been shown to induce obesity[4–6] and result in a predisposition to T2DM[5,7,8] and non-alcoholic fatty liver disease (NAFLD)[9,10]. The effects of EDCs on metabolism might also affect wildlife[11] and domestic pets[12].

Importantly, an increasing number of epidemiological studies have converged to establish a relationship between EDC exposures and the prevalence of obesity and T2DM (for extensive reviews, see REFS 5,10,13,14). A compilation of the epidemiological evidence published between 2011 and 2016 on the association between MDCs and obesity and T2DM is shown in Supplementary information S1 (table) and Supplementary information S2 (table), respectively. The term metabolism-disrupting chemicals (MDCs) was recently proposed to refer to any EDC that alters susceptibility to metabolic disorders and includes the terms 'obesogens', 'diabetogens' and 'diabesogens' (REF. 10). The term MDC is used throughout the rest of the article. The most common MDCs identified to date include diethylstilbestrol (DES), persistent organic pollutants (POPs), including 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), polychlorinated biphenyls (PCBs), dichlorodiphenyltrichloroethane (DDT) and its metabolites, perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and tetrabromobisphenol A (TBBPA) (BOX 1), and the non-persistent MDCs, bisphenol A (BPA) and phthalates, mainly bis(2-ethylhexyl) phthalate (DEHP) (BOX 2).

In this Review, we identify the essential elements of the complex energy balance equation required to construct a simplified model describing how MDCs alter energy homeostasis. Such a model should help to predict new MDCs, as well as to increase our understanding of their mode of action and to identify important organs and sites of action that are still unexplored. Dosage, age, gender and timing of exposure are particularly important in determining the final phenotype[5,15]. Detailed information on these parameters can be found in Supplementary information S3 (table) and Supplementary information S4 (table). Throughout this article, we have indicated doses and whether exposure was perinatal or during adulthood in most cases. This information is extremely important as EDC exposures affect the developing embryo, which results in altered embryonic organization and ultimately causes

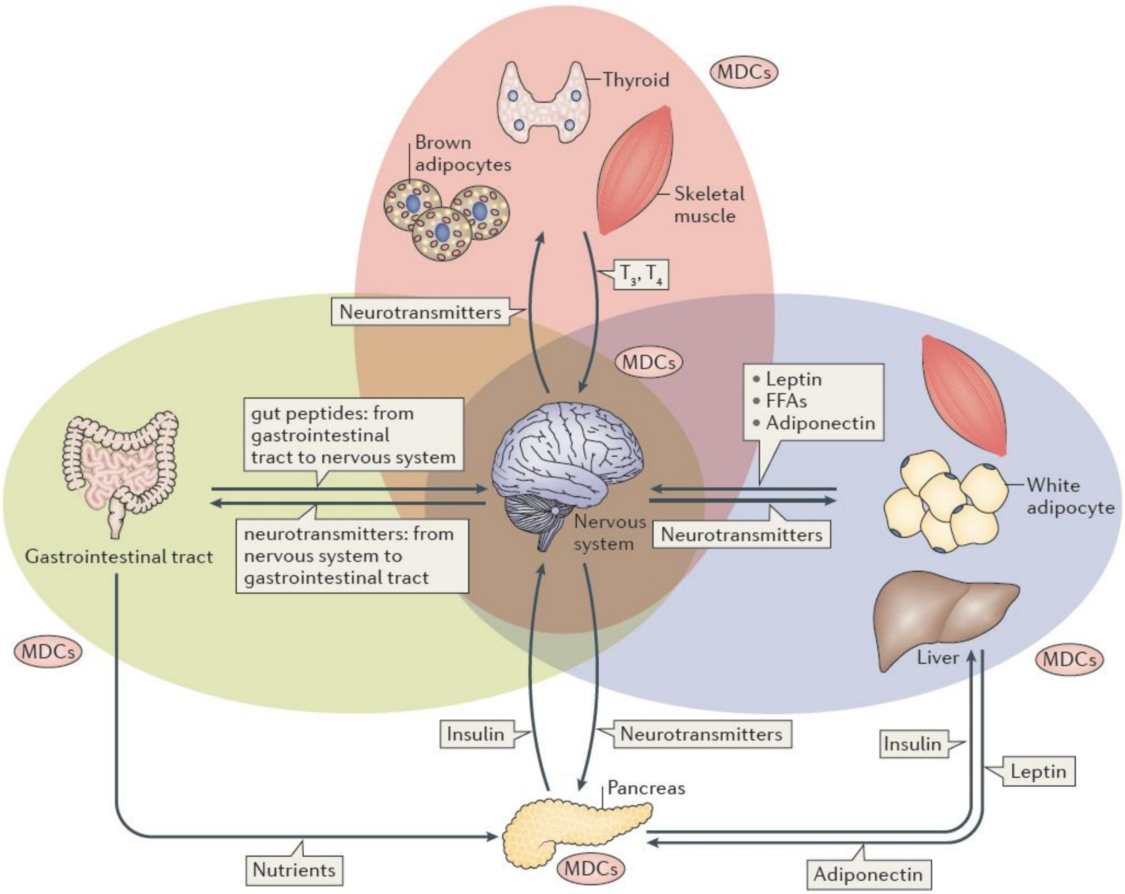
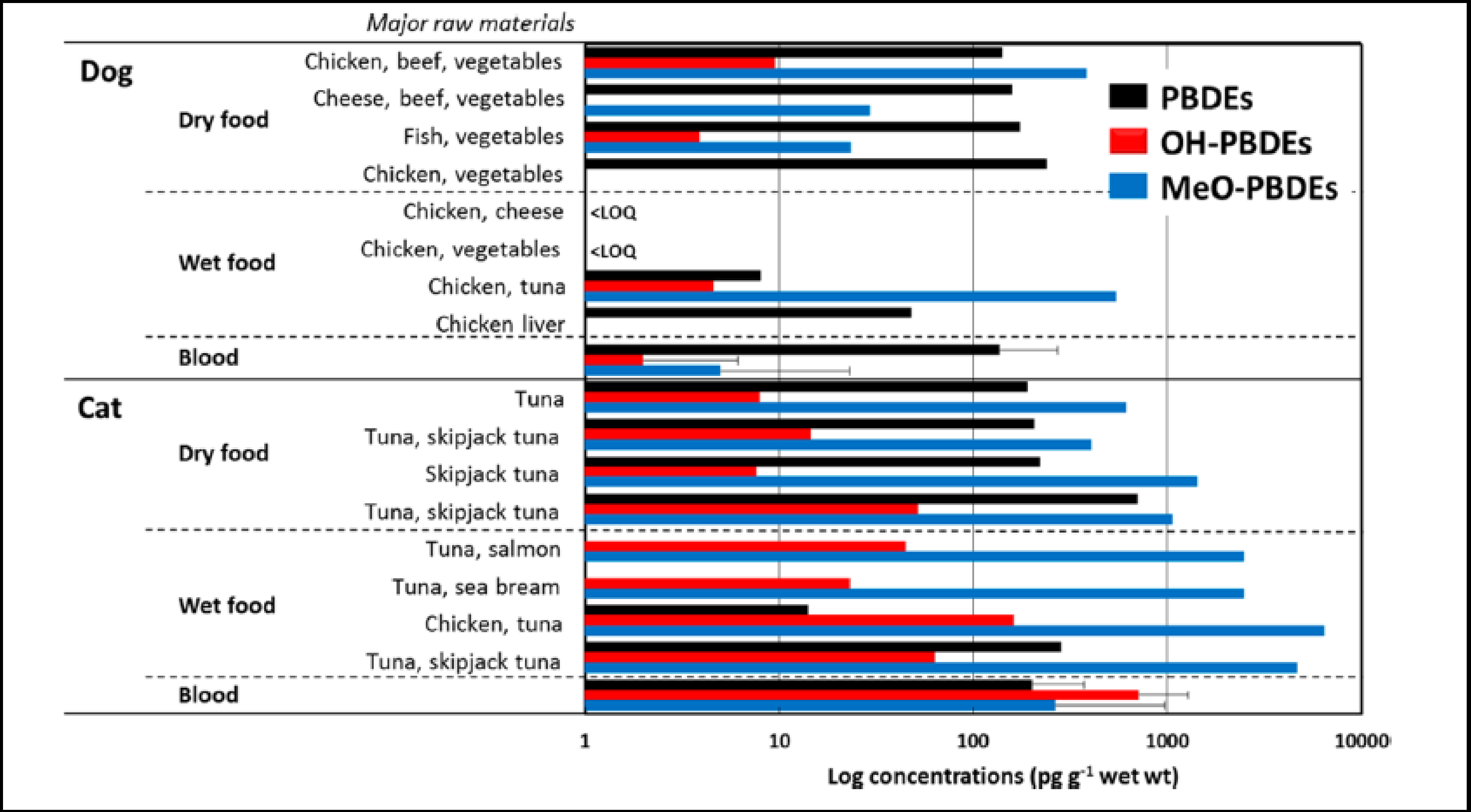
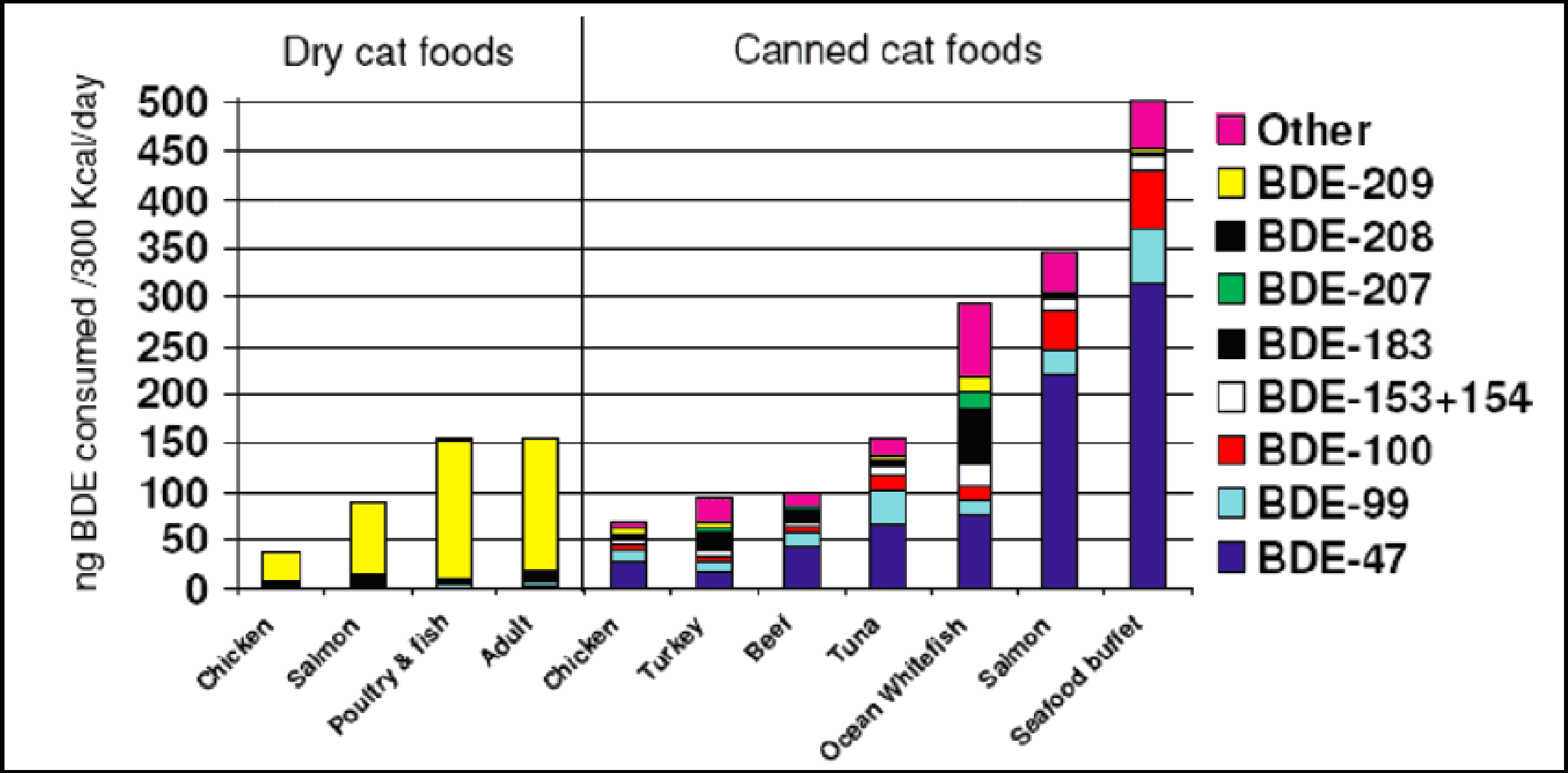


Figure 1 | **Organs targeted by metabolism-disrupting chemicals.** Energy homeostasis depends on the control of energy intake and energy expenditure. Metabolism-disrupting chemicals (MDCs) directly alter energy intake by modulating neuronal behaviour responsible for food intake. Indirectly, MDCs disrupt both epithelial transport in the intestine and the microbiota composition, which might alter serum levels of nutrients. In addition, MDCs alter the biosynthesis and probably also the release of gut peptides, which regulate food intake and energy balance. MDCs also affect insulin release from β cells, affecting both energy intake and energy storage. Energy storage might be disrupted as a consequence of the actions of MDCs on the brain, skeletal muscle, white adipose tissue and the liver. The disruption of insulin signalling in these organs or the direct action of MDCs on them disturbs the production and secretion of important signalling molecules, including free fatty acids (FFAs) and leptin, which have important roles in the endocrine pancreas and the brain. Energy output is disrupted by MDCs acting directly on the thyroid gland to modify serum levels of T_3 and T_4 and those that alter mitochondrial function in skeletal muscle and brown adipose tissue. Permission obtained from Cristina Sala-Ripoll, Alicante, Spain.

FELINE HYPERTHYROIDISM



FELINE HYPERTHYROIDISM



FELINE HYPERTHYROIDISM

Table 1. Median Concentrations (pg g^{-1} Wet Weight) And Range (Minimum to Maximum) of total PCBs, OH-PCBs, PBDEs, OH-PBDEs, MeO-PBDEs and Their Major Congeners in the Whole Blood of Pet Dogs and Cats, And Pet Food Products

	dog blood	dog dry food	dog wet food	cat blood	cat dry food	cat wet food
total PCBs[a]	<7.4 (<7.4–120)	120 (100–210)[e]	13 (<7.4–55)	48 (<7.4–260)	350 (130–1700)[e]	72 (20–350)
total OH-PCBs[b]	120 (9.4–820)	0.99 (<0.60–1.8)	0.95 (<0.60–2.1)	93 (38–290)	0.5 (<0.60–1.1)	0.86 (<0.60–3.3)
BDE47	< 4.2	< 4.2 (<4.2–42)	< 4.2	< 4.2	8.6 (<4.2–96)	4.3 (<4.2–14)
BDE209	100 (<4.2–280)	150 (130–170)	< 4.2 (<4.2–47)	160 (<4.2–490)	210 (160–510)	< 4.2 (<4.2–76)
total PBDEs[c]	100 < 4.2–300)	170 (140–240)[e]	4.2 (<4.2–48)	180 (<4.2–490)	210 (190–710)[e]	7.1 (<4.2–280)
6OH-BDE47	<1.0 (<1.0–8.0)	2.0 (<1.0–5.5)	< 1.0 (<1.0–4.6)	290 (100–1500)	4.7 (<1.0–52)	40 (19–110)
2′OH-BDE68	<1.0 (<1.0–5.6)	< 1.0 (<1.0–4.1)	< 1.0	14 (<0.60–98)	3.2 (<1.0–15)	14 (3.9–50)
total OH-PBDEs[d]	<1.0 (<1.0–14)	2.0 (<1.0–9.6)	< 1.0 (<1.0–4.6)	300 (100–1600)[f,g]	11 (7.7–52)	54 (23–160)
6MeO-BDE47	<1.0 (<1.0–65)	12 (<1.0–160)	< 1.0 (<1.0–170)	< 1.0 (<1.0–310)	380 (200–670)	1600 (1300–1900)
2′MeO-BDE68	<1.0	15 (<1.0–230)	< 1.0 (<1.0–380)	< 1.0 (<1.0–1700)	350 (200–970)	2000 (1200–4500)
total MeO-PBDEs[d]	<1.0 (<1.0–65)	26 (<1.0–380)	< 1.0 (<1.0–550)	< 1.0 (<1.0–2000)	840 (410–1400)[g]	3600 (2500–6400)[g]

PCB’s banned in the US in 1970’s

Feline Hyperthyroidism

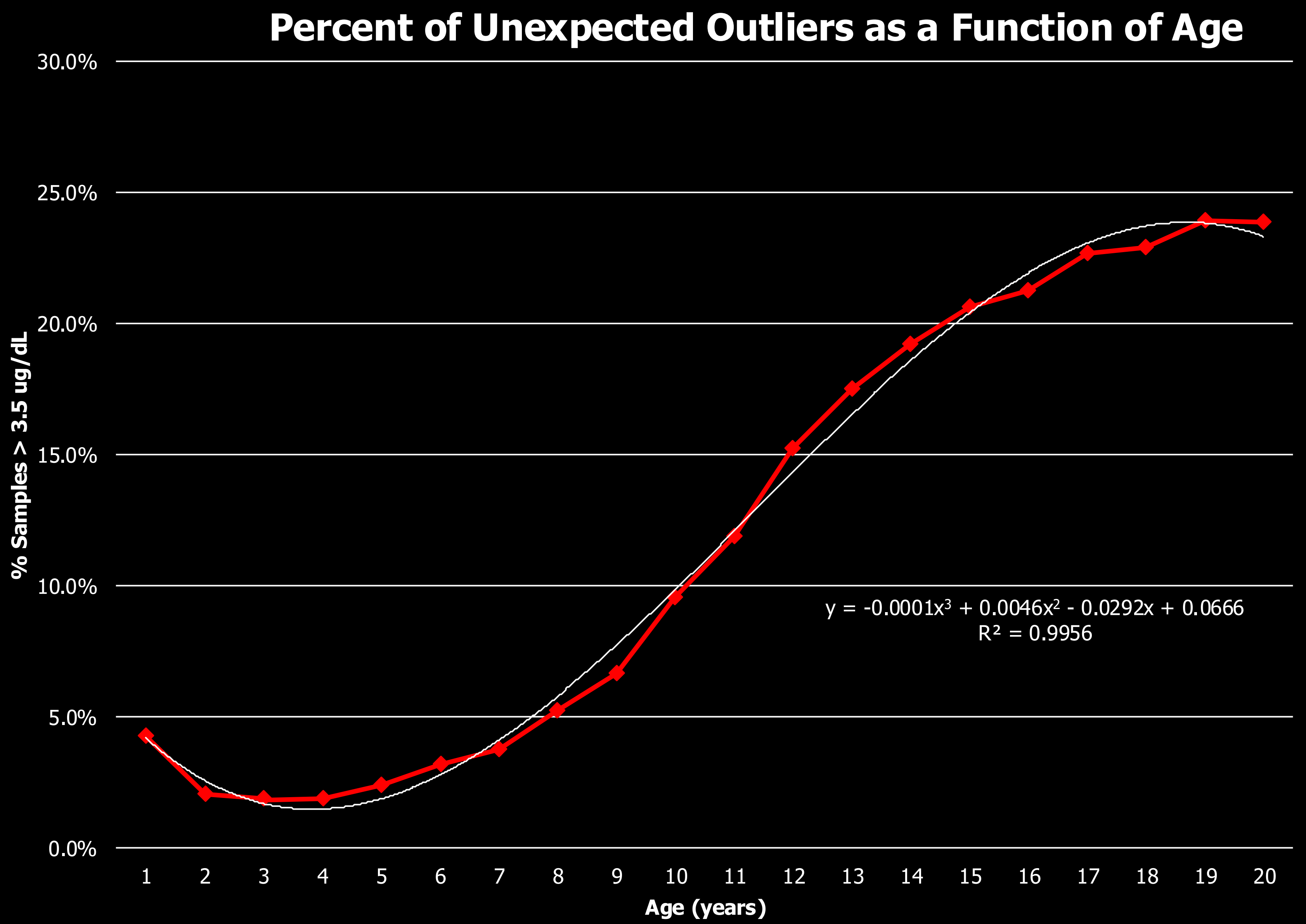
Results suggest that pet cats routinely ingest natural MeO-PBDEs in cat food products containing fish and retain their demethylated metabolites, OH-PBDEs, in the blood for a prolonged time. OH-PBDE's are known endocrine disrupters and have been associated with hyperthyroidism and acromegaly in cats.

Feline Hyperthyroidism

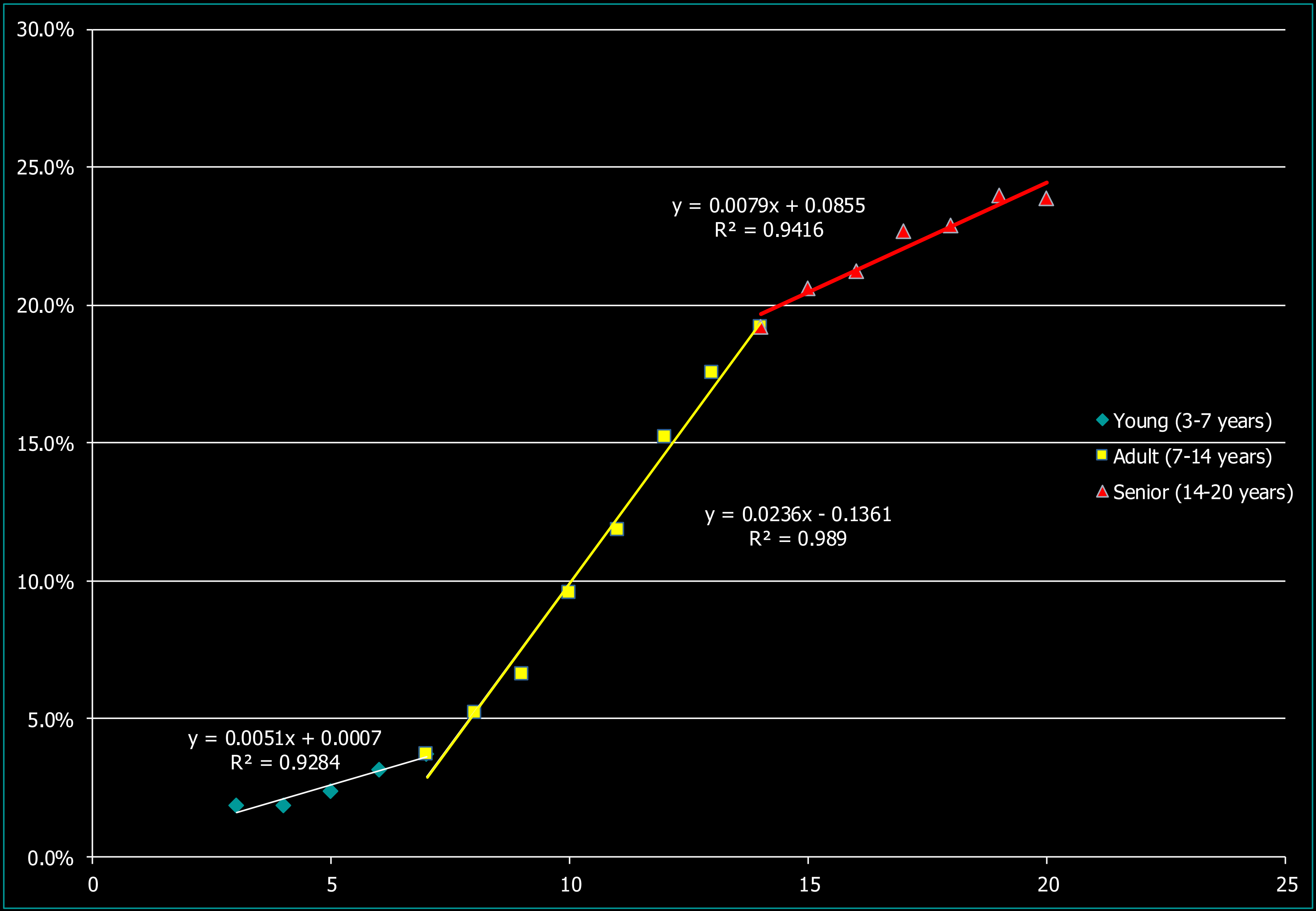
Etiology

- Increasing age
- Non pure bred cats
- Use of a litter box
- More than 50% wet food in the diet
- Exposure to fish
- Exposure to food in a can (pop-top; bisphenol A)

Risk factors for feline hyperthyroidism in the UK. J Small Anim Pract. 2009 Aug;50(8): 406-14.



Percent of Unexpected Outliers as a Function of Age
30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
% Samples > 3.5 ug/dL
y = -0.0001x³ + 0.0046x² - 0.0292x + 0.0666
R² = 0.9956
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
Age (years)



30.0%
25.0%
20.0%
15.0%
10.0%
5.0%
0.0%
0
5
10
15
20
25
y = 0.0079x + 0.0855
R² = 0.9416
y = 0.0236x - 0.1361
R² = 0.989
y = 0.0051x + 0.0007
R² = 0.9284
Young (3-7 years)
Adult (7-14 years)
Senior (14-20 years)

Feline Hyperthyroidism

Clinical signs

- Less severe than 10 - 15 years ago
- Far less clinically significant cardiac disease
- Asymptomatic cats
- Most common are GI signs
  - Vomiting, diarrhea and/or weight loss

Feline Hyperthyroidism

Clinical signs

- Palpable thyroid nodule
- Polyuria and polydipsia
- Mild weight loss
- Behavioral signs

Feline Hyperthyroidism

Diagnostic Approach

Geriatric cats
Concurrent illness and medications
Thorough physical examination

Feline Hyperthyroidism

Diagnostic Approach

Routine data base

- CBC, serum chemistry profile, UA with culture
- Thoracic radiographs
- EKG

Feline Hyperthyroidism

Diagnostic Approach

Thyroid Function Testing

- TT4
- TT3
- fT4 (equilibrium dialysis)
- TRH stimulation
- T3 suppression
- Imaging

Feline Hyperthyroidism

Diagnostic Approach

Thyroid Function Testing

- TT4
  - sensitivity 0.913
  - specificity 1.0
- fT4 (equilibrium dialysis)
  - sensitivity 0.985
  - specificity 0.937
    - * false positive rate 6 %

Feline Hyperthyroidism

Diagnostic Approach

Thyroid Function Testing

fT4 (equilibrium dialysis)

- Not as a first line test
- TT4 levels in the upper 50 % of the normal range

						Expected TT4 Concentrations		
Age (years)	# Samples	25%	Median	75%	IQR	Low	High	Outlier
<1	2,919	1.4	1.8	2.4	1	0.5	3.9	>3.9
1	3,802	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
2	6,399	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
3	7,113	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
4	8,970	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
5	12,788	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
6	15,773	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
7	22,816	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
8	29,737	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
9	33,896	1.5	1.9	2.3	0.8	0.5	3.5	>3.5
10	46,519	1.5	1.9	2.4	0.9	0.5	3.8	>3.8
11	44,303	1.6	2	2.5	0.9	0.5	3.9	>3.9
12	52,226	1.5	2	2.6	1.1	0.5	4.3	>4.3
13	55,121	1.5	2	2.7	1.2	0.5	4.5	>4.5
14	53,887	1.5	2	2.8	1.3	0.5	4.8	>4.8
15	47,844	1.5	2	3	1.5	0.5	5.3	>5.3
16	36,372	1.5	2	3	1.5	0.5	5.3	>5.3
17	25,005	1.5	2.1	3.2	1.7	0.5	5.8	>5.8
18	15,273	1.5	2	3.2	1.7	0.5	5.8	>5.8
19	7,299	1.4	2	3.3	1.9	0.5	6.2	>6.2
20	3,703	1.4	2	3.4	2	0.5	6.4	>6.4

Feline Hyperthyroidism

Diagnostic Approach

Value of cTSH

Up to 40% of normal cats have low or undetectable TSH levels

Hyperthyroid cats should have suppressed TSH

Feline Hyperthyroidism

Treatment Options

- Clinical status of the patient
- Cost concerns
- Use of chronic oral medications
- Availability of radioactive iodine
- Surgical experience

Feline Hyperthyroidism

- Treatment Options
  - Medical Management
    - Tapazole (methimazole)
      - Oral vs topical
    - Carbimazole

Feline Hyperthyroidism

Treatment Options

- Medical Management

- Tapazole (methimazole)
  - Inhibits formation of T4
  - Time to effect 7 - 14 days
  - Short term or chronic use
  - Topical applications

Feline Hyperthyroidism

Treatment Options

- Medical Management
  - Tapazole (methimazole)
    - Dose
      - 1.25 to 2.5 mg q 12 - 24 hours
      - Recheck values in 7 days
      - Monitor TT4 and renal values

Feline Hyperthyroidism

Treatment Options

- Medical Management
  - Tapazole (methimazole)
    - Side-effects
      - GI
      - Hematologic
      - Hepatic

Feline Hyperthyroidism

Treatment Options

- Medical Management
  - Tapazole (methimazole)
    - Long term monitoring
      - TT4 and CBC and chemistry values every 3 - 6 months
      - Monitor for concurrent disease

Feline Hyperthyroidism

Tapazole (transdermal)

Most studies have shown efficacy and safety

2.5 mg – 5.0 mg BID

Hoffman SB, Yoder AR, Trepanier LA: Bioavailability of transdermal methimazole in a pluronic lecithin organogel (PLO) in healthy cats. J Vet Pharmacol Ther 25:189-93, 2002

Hoffmann G, Marks SL, Taboada J, Hosgood GL, Wolfsheimer KJ: Transdermal methimazole treatment in cats with hyperthyroidism. J Feline Med Surg 5:77-82, 2003

Feline Hyperthyroidism

Treatment Options

Radioactive Iodine

Simple, safe and effective
Greater availability
No side-effects

Feline Hyperthyroidism

Radioactive Iodine

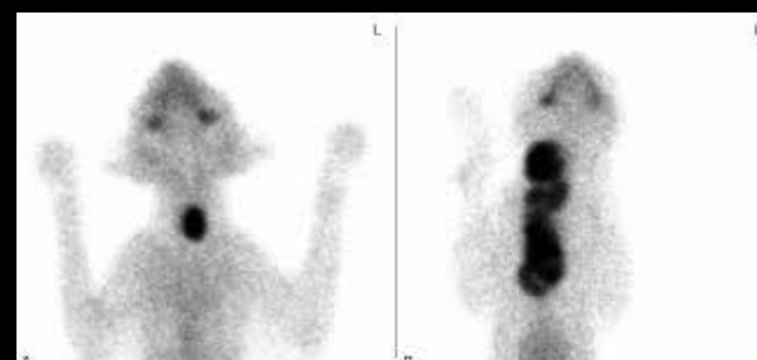
Dose: 1- 5 mCi; higher doses with carcinoma.

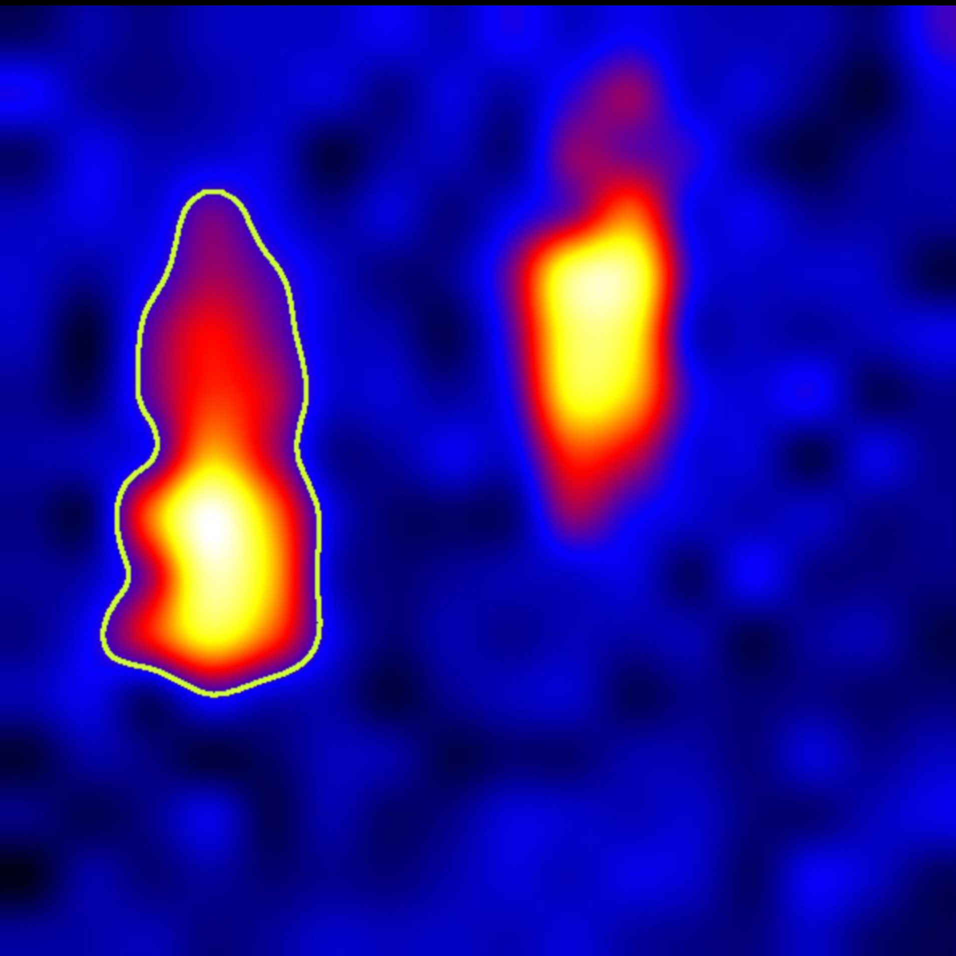
Recommendation will

suggest 1 – 2 mCi.

Effectiveness > 95 %

Median survival is 2.0 years; the percentage of cats alive after 1, 2, and 3 years of treatment is 89, 72, and 52%, respectively



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RECTOR, MEOW MEOW
SCINT46
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DR 122
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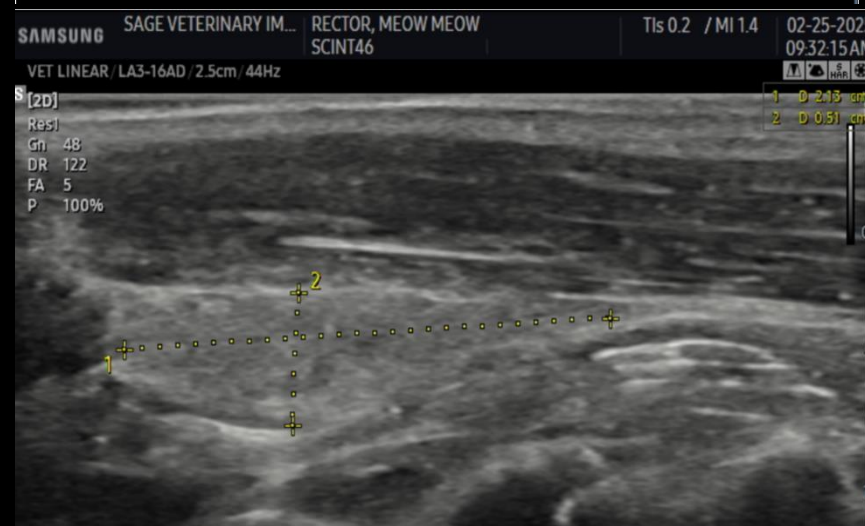
2 D 0.26 cm
R THY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SCINT46
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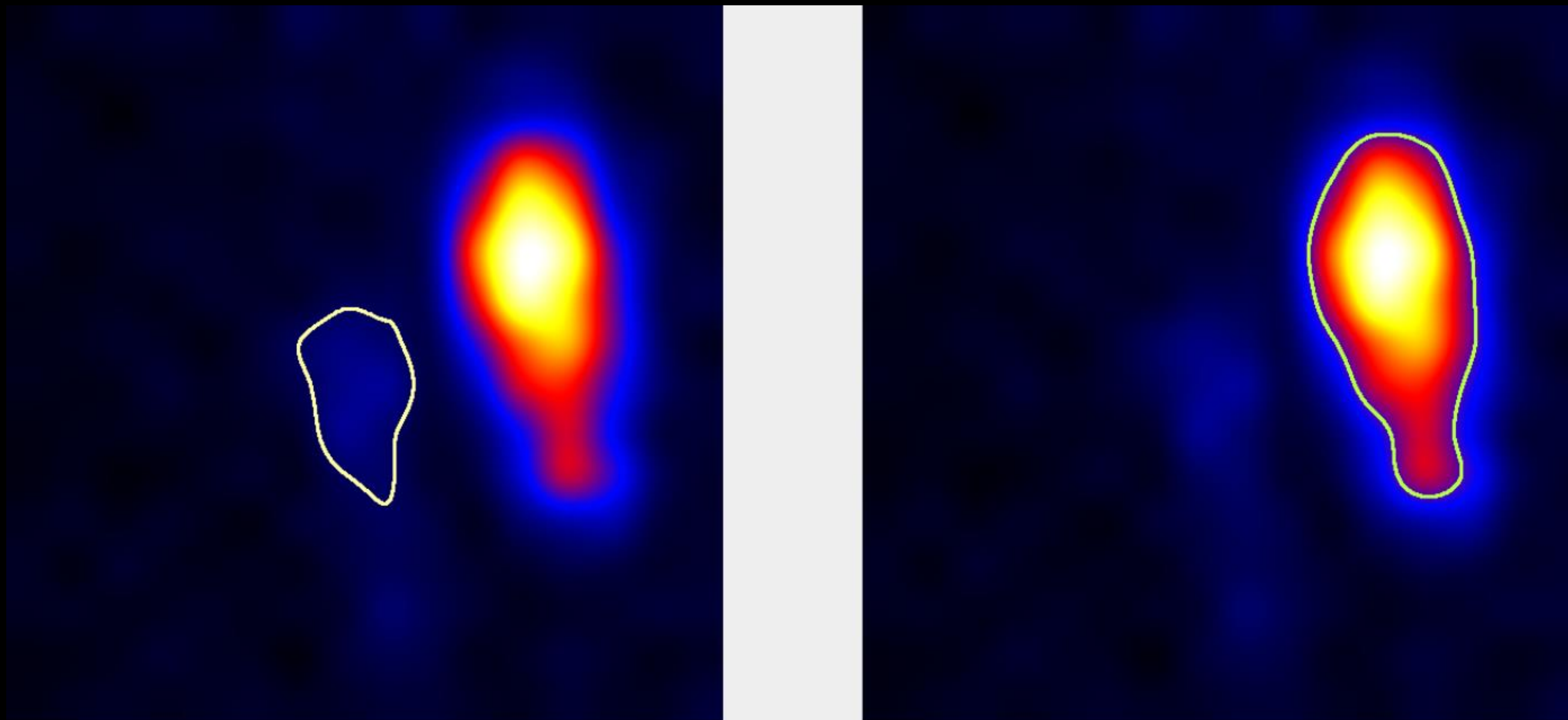
2 D 0.51 cm

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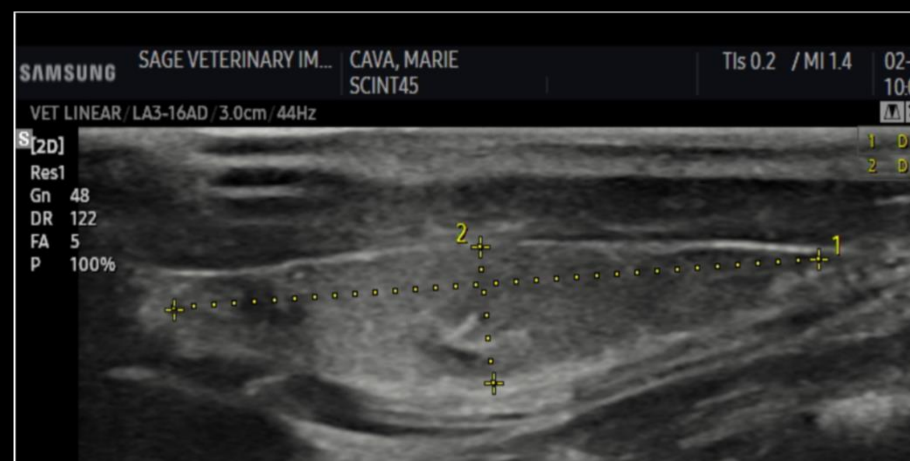
2 D 0.14 cm
1
2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Res1
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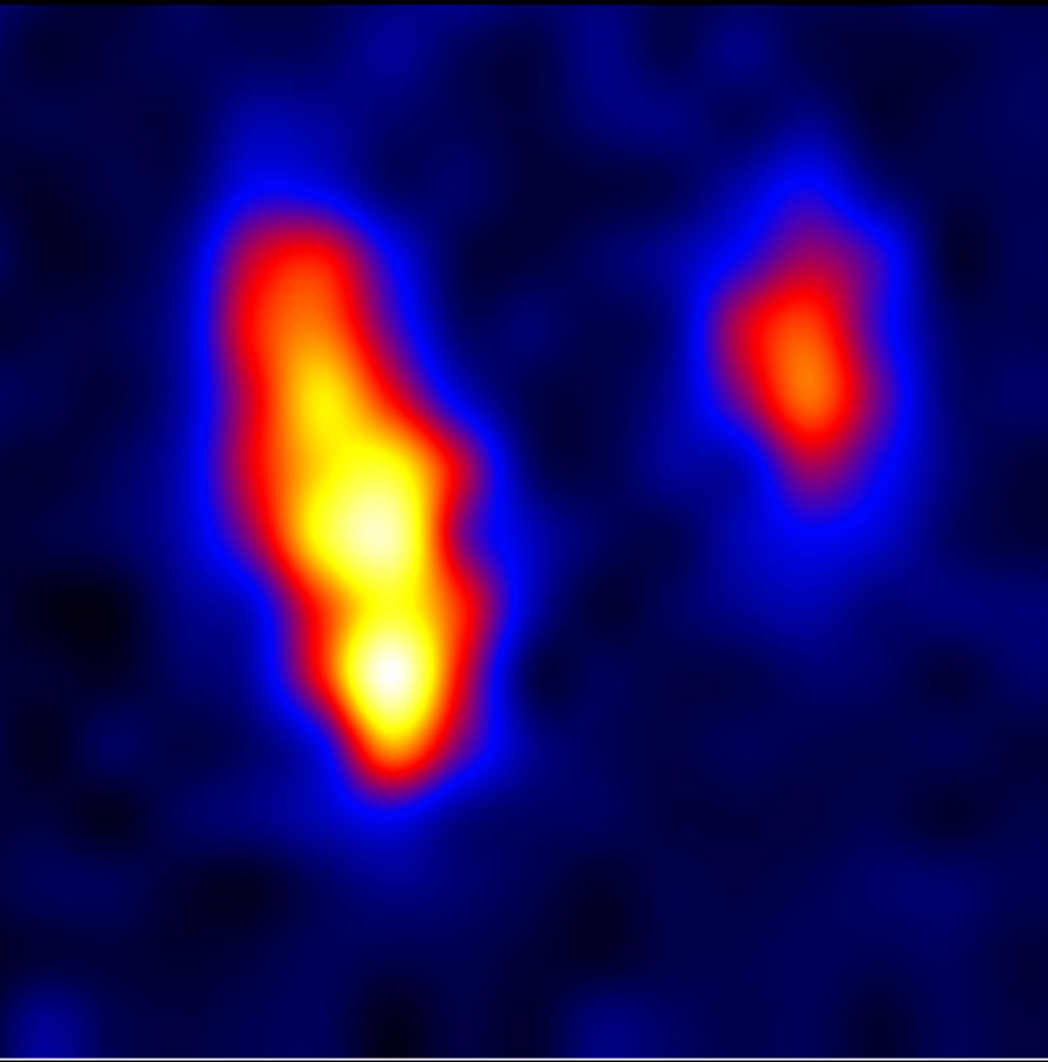
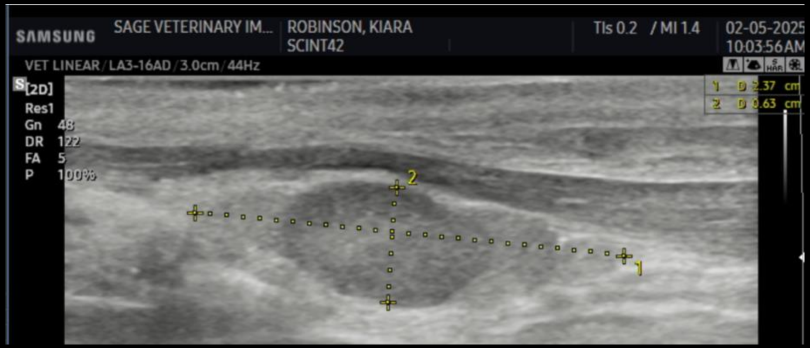
DR 122
FA 5
P 100%
2
1





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Gn 48
DR 122
FA 5
P 100%
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2 D 0.63 cm

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SCINT42
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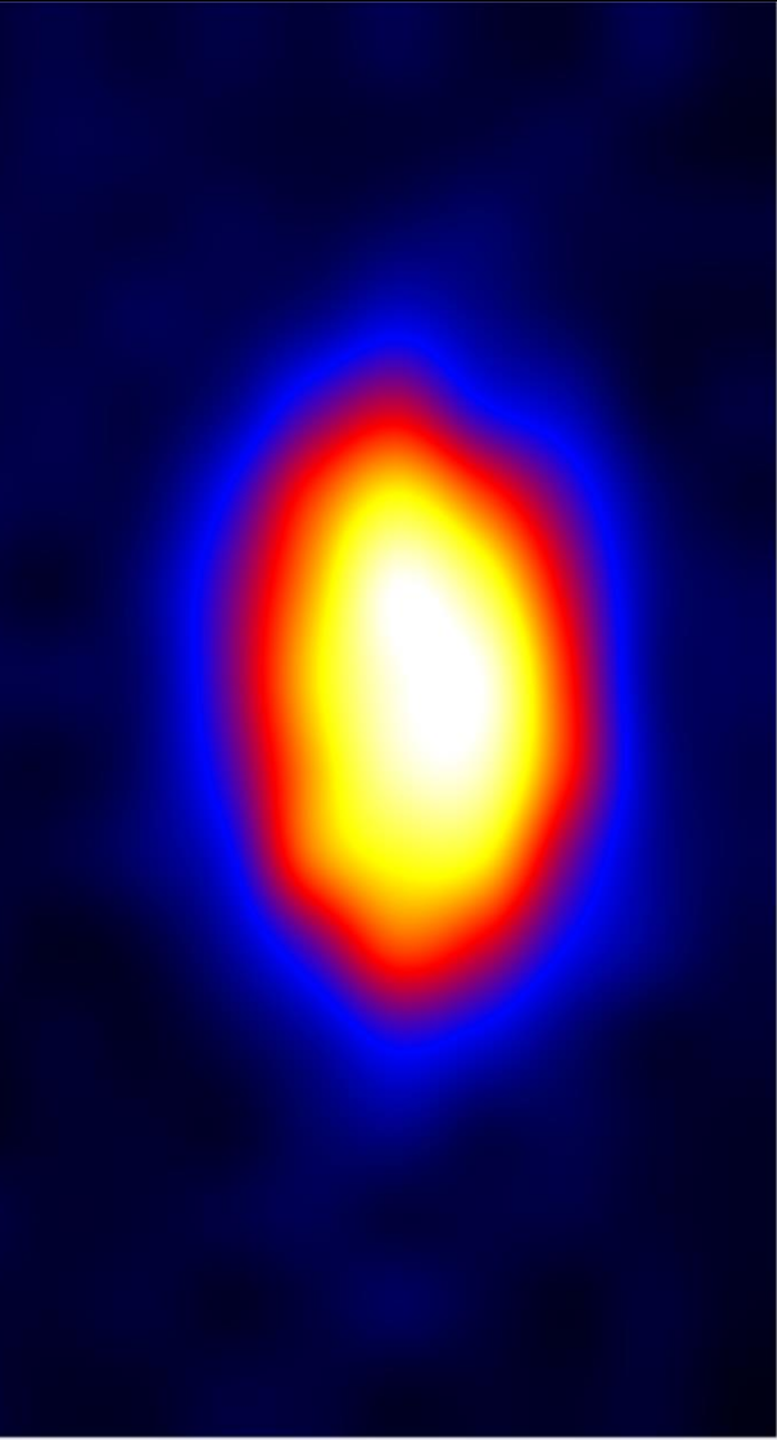

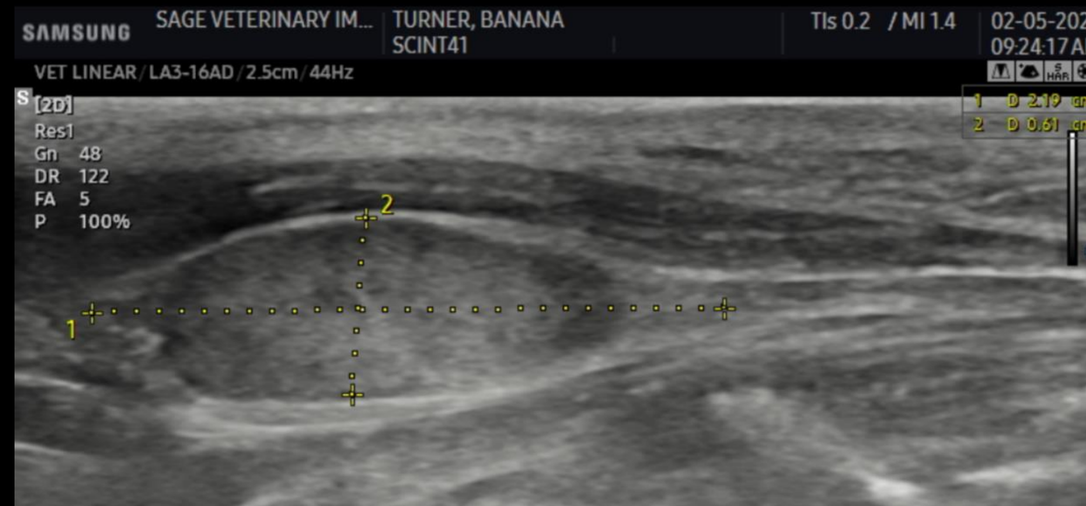

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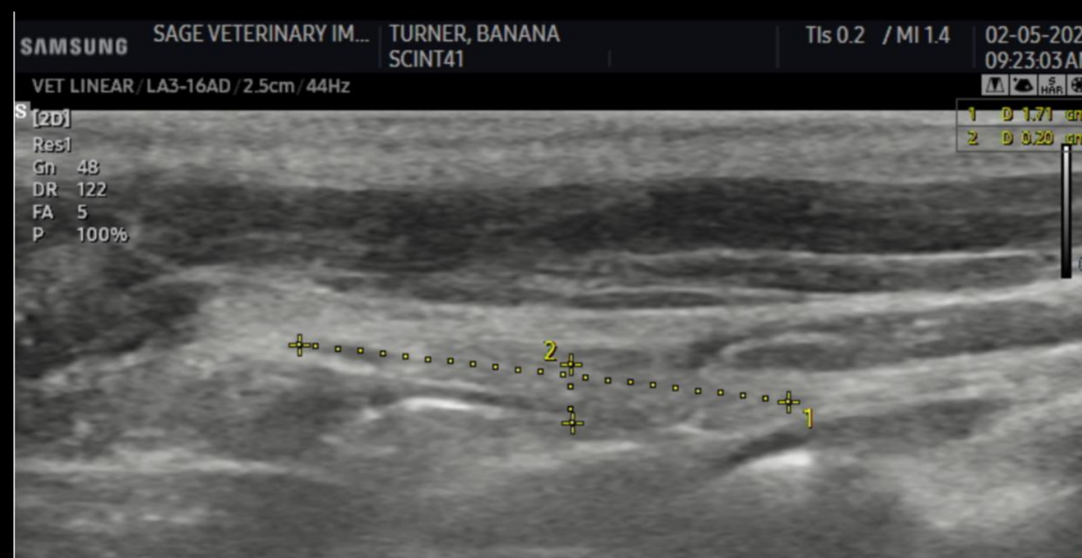
2 D 0.61 cm



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Gn 48
DR 122
FA 5
P 100%
1 D 1.71 cm
2 D 0.20 cm

Feline Hyperthyroidism

Treatment Options

Radioactive Iodine

- Hypothyroidism < 2 %
- T4 values may be low for 2-4 months post treatment
- Withdrawal times for tapazole, carbimazole, and diet

Feline Hyperthyroidism

Treatment Options

Radioactive Iodine

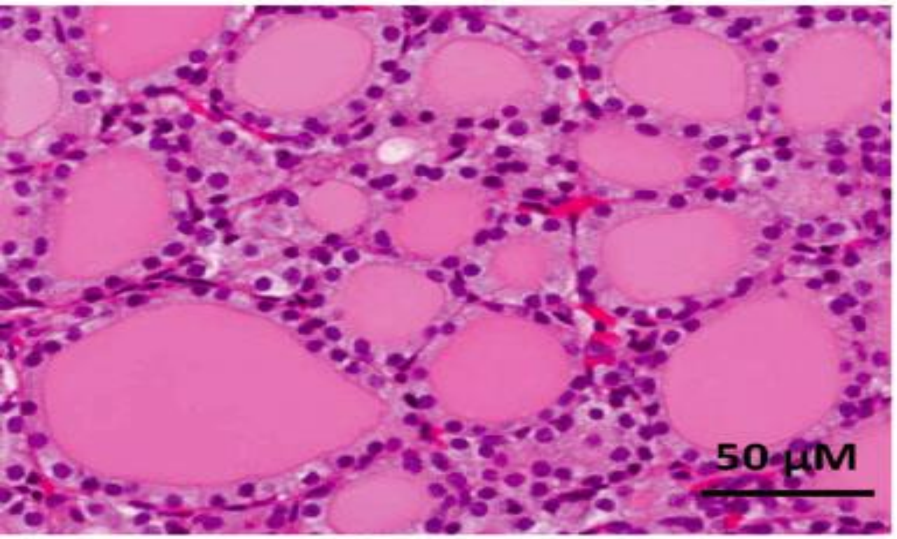
However, up to 30% (50 of 165 cats) were hypothyroid 3 months after radioactive iodine therapy in one study; of these, 56% (19 of 34 hypothyroid cats with available information) had clinical signs of hypothyroidism and 52% (23 of 44 cats) were given thyroid hormone supplementation

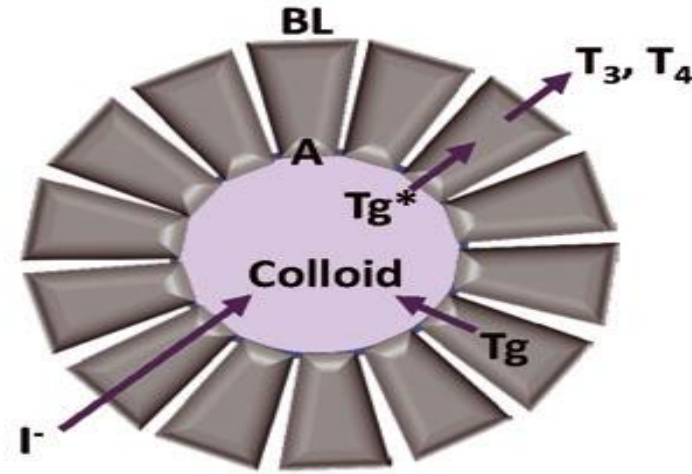
Feline Hyperthyroidism

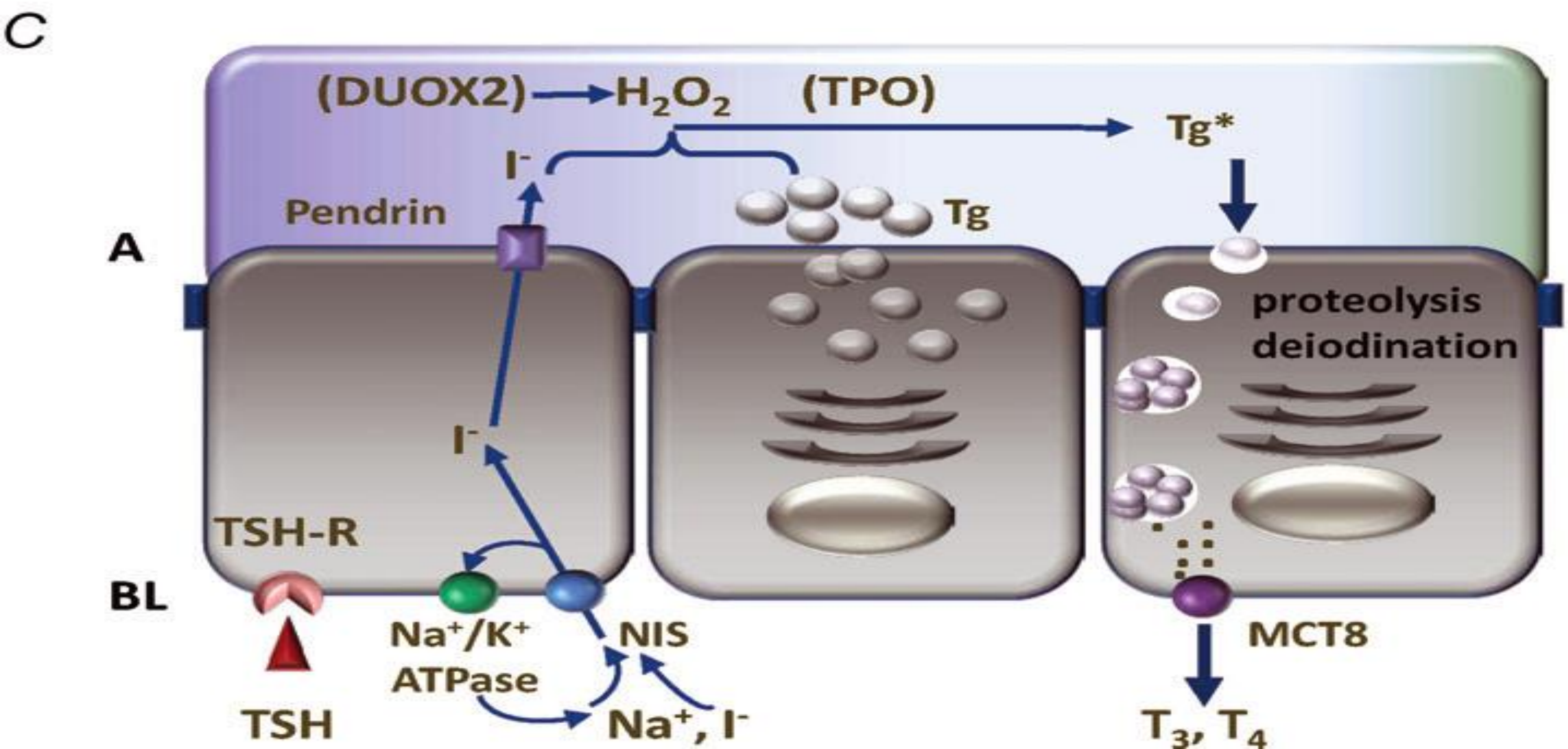
Treatment Options

- Radioactive Iodine

- Monitoring post-therapy
  - TT4 at 1, 3, 6 and 12 months
  - Complete profile and UA every 6 months
  - Thyroid supplementation timing
  - Clinical signs of hypothyroidism
    - Weight gain, lethargy and poor hair coat


50 μM


BL
T_3, T_4
A
Tg*
Colloid
Tg
I^-


C
(DUOX2) → H_2O_2
(TPO)
Tg*
I^-
Pendrin
Tg
A
proteolysis
deiodination
I^-
TSH-R
BL
Na^+/K^+
ATPase
NIS
MCT8
TSH
Na^+, I^-
T_3, T_4

Feline Hyperthyroidism

Role of iodine

What is the requirement ?

NRC = 1.4 ppm (mg I/kg diet; DM)

AAFCO = 0.35 ppm

Clinical signs of I def < 0.15-0.17 ppm

Wedekind et al = 0.46 ppm

J Amer Physio 94: 527-539, 2010

Success Rates

92%

Surgery

Radioactive Iodine

94%

82%

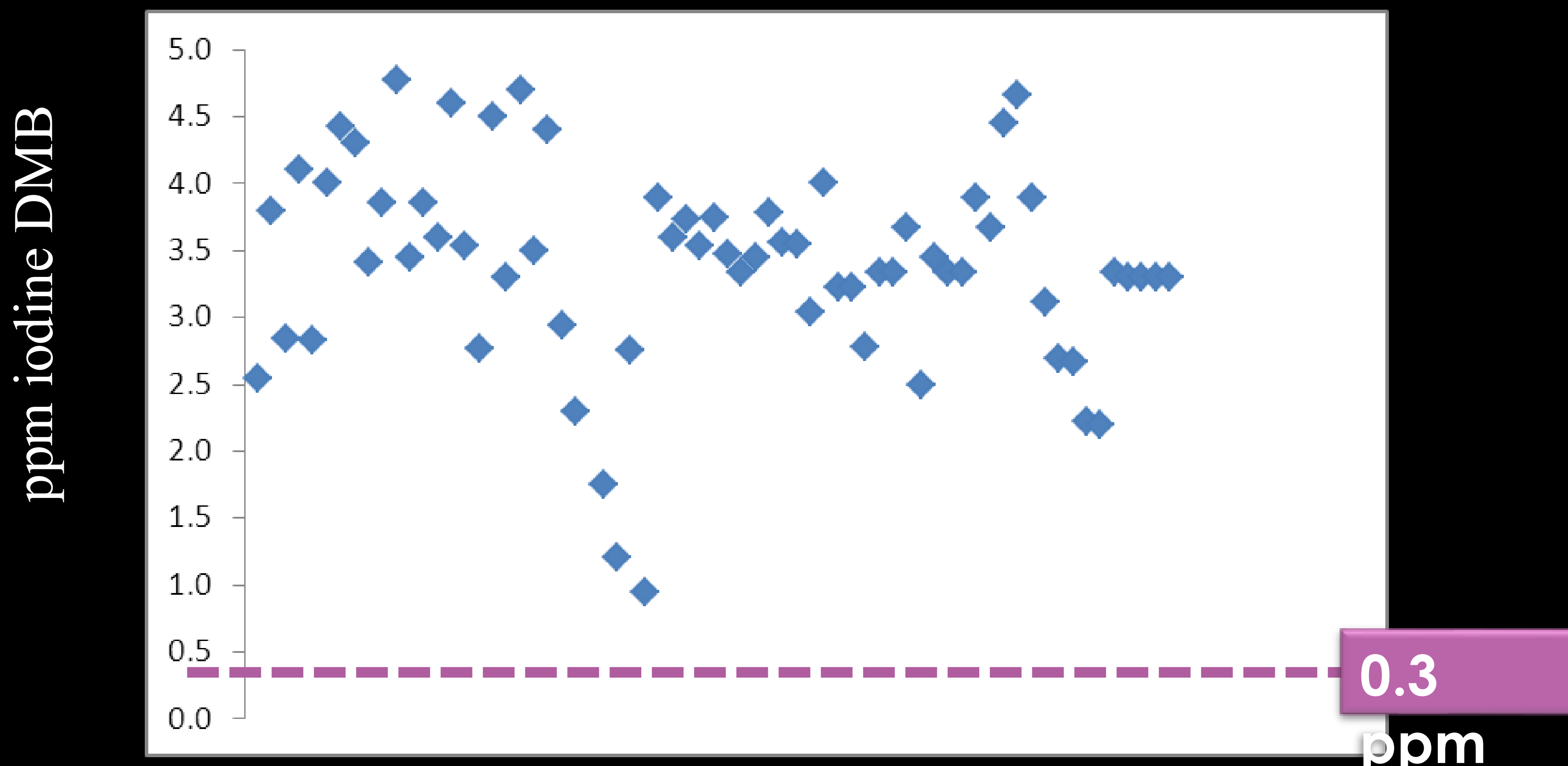
Anti-thyroid Drugs

90%

Nutritional Therapy

*Total $T_4 < 4$ µg/dl

Iodine levels of commercial cat foods



Persistent Hyperthyroidism ?

Okay to feed with y/d

- Cosequin® for Cats
- Dasuquin® for Cats
- 1-800-PetMeds® Joint Enhancer
- Epakitin®
- MiraLAX®
- Laxatone®
- Renal K+®
- C.E.T.® Enzymatic Toothpaste
- Viralys® Gel

Not okay to feed with y/d

- 1-800-PetMeds® Soft VitaChews
- Azodyl®
- GoldVet™ Potassium Citrate
- Heartgard® Chewables for Cats
- Stewart's Pet Food Flavor
- Welactin® Feline
- FortiFlora® Nutritional Supplement
- Feline Greenies® Pill Pockets®
- Felovite II® w/ Taurine
- Nutri-Cal®
- Glyco-Flex® II For Cats
- C.E.T.® Oral Hygiene Chews for Cats

Protein and Cats

Quantity – 35% DMB exceeds needs for adult/senior cats (26% DMB)

Quality - high
source – meat/vegetable
essential amino acids

Digestibility - high

Study	Protein content	Study Duration	Study population	Lean Body Mass (DEXA)
Kirk (2000)	16% calories as protein	16 weeks	10 CKD cats 9 senior cats	Lost 89.4g LBM
	24% calories as protein	16 weeks	10 CKD cats 9 senior cats	Gained 67.5 g LBM* *$p < 0.04$
Yamka (2010)	28.5% DMB	16 weeks	12 senior cats (10 ± 2 years)	Stable
Yu (2011)	28.5% DMB	30 weeks	7 CKD cats	Stable / ↑

y/d feline (canned / dry) = 34 – 36% protein DMB

Feline Hyperthyroidism

Additional Questions:

Reformulation of all feline diets so as not to exceed 0.46 ppm ?

At what age should cats be placed on iodine
"restricted" diets ?

Will this be the "cure" ??

Feline Hyperthyroidism

Renal Disease and Hyperthyroidism

- Both common in elderly cats
- Similar clinical signs
- Progression of renal disease in cats treated for hyperthyroidism
  - Rare < 2 - 5 %
  - Most cats develop clinically insignificant changes in BUN and creatinine

Feline Hyperthyroidism

Renal Disease and Hyperthyroidism

- Increasing TT4 increases GFR
- Cats with hyperthyroidism may have "falsely" lower levels of BUN and creatinine
- Sudden decreases in TT4 may result in rapidly decreasing GFR and may unmask underlying renal disease

Feline Hyperthyroidism

Renal Disease and Hyperthyroidism

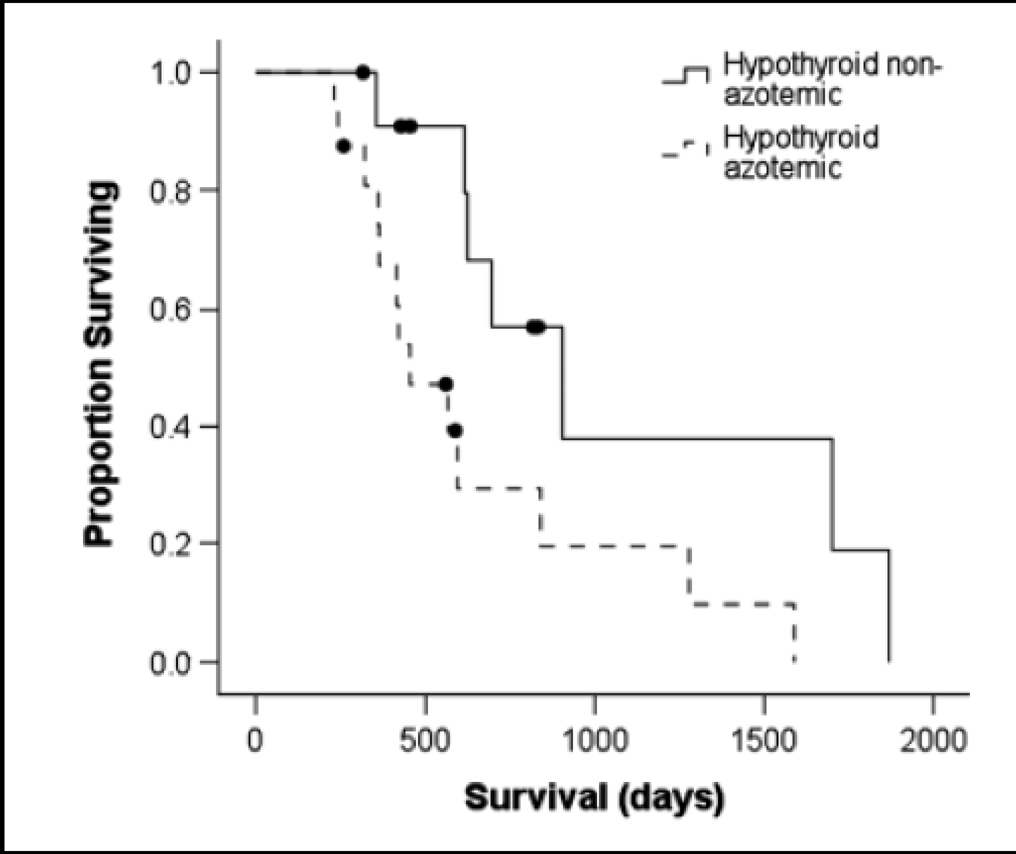
Finding a high serum SDMA concentration in a hyperthyroid cat can help predict development of azotemia after treatment. The test has high diagnostic test specificity (few false-positive results) but relatively low sensitivity (fails to predict azotemia in most hyperthyroid cats).

Evaluation of Serum Symmetric Dimethylarginine Concentration as a Marker for Masked Chronic Kidney Disease in Cats With Hyperthyroidism. J Vet Int Med 2018 Jan-Feb; 32(1): 295–304.

Feline Hyperthyroidism

Renal Disease and Hyperthyroidism

- Consider diet or low dose tapazole prior to any definitive therapy
  - Re-check TT4 and renal values in 7 days
  - If mild increase in IRIS score (less than 2) proceed with treatment
  - If more than 2 we need to re-evaluate the patient



Hypothyroid non-azotemic
Hypothyroid azotemic
Proportion Surviving
1.0
0.8
0.6
0.4
0.2
0.0
0
500
1000
1500
2000
Survival (days)

Renal Disease and Thyroid Status

	5/24	6/6	6/20	6/27	7/12
BUN	54	48	36	33	39
Creat	3.7	2.2	2.3	2.3	2.0
TT4	2.4	7.7	6	5.7	1.8

5/24 Started transition to y/d; stopped methimazole
6/6 Day 5 of y/d as sole diet
6/20 Day 19 of y/d
6/27 Day 26 of y/d
7/12 Day 41; stopped y/d and started methimazole

Renal Disease and Thyroid Status

	8/25	9/26	10/11	10/24	10/31	11/15
BUN	22	109	39	73	57	53
Creat	2.7	6.1	2.8	2.5	2.2	2.1
TT4	1.4	0.9	15.8	10.4	5.1	2.7

8/25	On Fancy Feast and methimazole
9/26	Stopped methimazole
10/11	Started y/d
10/24	Day 13 of y/d
10/31	Day 20 of y/d
11/15	Day 35 of y/d

Renal Disease and Thyroid Status
Cat # 2

	Day 1	Day 22	Day 30	Day 60
BUN	75	67	52	61
Creat	4.2	4.1	3.5	3.6
TT4	7.7	3.8	3.3	3.6

	Day 90	Day 128	Day 158
BUN	37	36	34
Creat	2.7	2.5	2.1
TT4	3.1	2.2	1.8

Renal Disease and Thyroid Status
Cat # 3

	Day 1	Day 30	Day 42	Day 56	Day 60
BUN	68	56	51	46	21
Creat	3.9	3.5	3.6	3.1	2.2
TT4	7.2	4.3	4.0	3.2	2.6

	Day 70	Day 100	Day 120	Day 150	
BUN	20	21	22	25	
Creat	1.9	2.1	2.2	2.1	
TT4	1.6	1.4	1.6	1.7	

Renal Disease and Thyroid Status
Cat # 4

	Day - 90	Day 1	Day 21	Day 35	Day 65
BUN	32	37	33	42	18
Creat	1.9	2.7	2.2	2.1	1.7
TT4	2.4	6.3	4.1	3.8	2.4

Feline Hyperthyroidism

41 cats treated to date (3 to 19 months)

35/41 (85 %) improved clinically and biochemically (weight stable or improved)

6/41 did not become euthyroid

- 3/6 – Being fed foods other than y/d
- 3/6 – Sole food was y/d (7%)

No cats became hypothyroid

Feline Hyperthyroidism

Time to Normalize TT4

Week 2	18/41	44%
Week 4	27/41	66%
Week 8	31/41	76%
Week 12	34/41	83%
Week 15	37/37	100%

Feline Hyperthyroidism

Renal Function at 16 Weeks (IRIS)

6/41 cats azotemic at Time 0

- 2 – Stage 2
  - Both normal at week 16
- 4 – Stage 3
  - 2 normal at week 16
  - 2 dropped to stage 2

Feline Hyperthyroidism

Renal Function

No decline in renal function at 16 weeks 33/35 cats.

- Two cats azotemic
  - Urethral obtruction
  - UTI, ureteral stones

Feline Hyperthyroidism

Body Weights

Increased in 31/37

Median weight gain 0.51 kg (+ 8.3%)

Decreased in 3/37

Median weight loss 0.31 kg (- 9.7%)

URI and lipidosis

Feline Hyperthyroidism

Asymptomatic Patients

- More common due to routine testing
- Presence of clinical or laboratory abnormalities
- Treat, diet or "watchful waiting"
  - Informed and observant client
  - Do no harm

Feline Hyperthyroidism

Additional Points to Ponder:

Another effective treatment option to help manage your patients with hyperthyroidism

Tailor treatment for the patient and client