Exogenous thyrotoxicosis in dogs attributable to consumption of all-meat commercial dog food or treats containing excessive thyroid hormone: 14 cases (2008–2013)

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Objective—To describe findings in dogs with exogenous thyrotoxicosis attributable to consumption of commercially available dog foods or treats containing high concentrations of thyroid hormone.

Design—Retrospective and prospective case series.

Animals—14 dogs.

Procedures—Medical records were retrospectively searched to identify dogs with exogenous thyrotoxicosis attributable to dietary intake. One case was found, and subsequent cases were identified prospectively. Serum thyroid hormone concentrations were evaluated before and after feeding meat-based products suspected to contain excessive thyroid hormone was discontinued. Scintigraphy was performed to evaluate thyroid tissue in 13 of 14 dogs before and 1 of 13 dogs after discontinuation of suspect foods or treats. Seven samples of 5 commercially available products fed to 6 affected dogs were analyzed for thyroxine concentration; results were subjectively compared with findings for 10 other commercial foods and 6 beef muscle or liver samples.

Results—Total serum thyroxine concentrations were high (median, 8.8 µg/dL; range, 4.65 to 17.4 µg/dL) in all dogs at initial evaluation; scintigraphy revealed subjectively decreased thyroid gland radionuclide in 13 of 13 dogs examined. At ≥ 4 weeks after feeding of suspect food or treats was discontinued, total thyroxine concentrations were within the reference range for all dogs and signs associated with thyrotoxicosis, if present, had resolved. Analysis of tested food or treat samples revealed a median thyroxine concentration for suspect products of 1.52 µg of thyroxine/g, whereas that of unrelated commercial foods was 0.38 µg of thyroxine/g.

Conclusions and Clinical Relevance—Results indicated that thyrotoxicosis can occur secondary to consumption of meat-based products presumably contaminated by thyroid tissue, and can be reversed by identification and elimination of suspect products from the diet. (*J Am Vet Med Assoc* 2015;246:105–111)

Hyperthyroidism is uncommon in dogs.[1] Most reported cases of canine hyperthyroidism are caused by differentiated, autonomously functional thyroid gland carcinomas.[1–5] Although hyperthyroidism is almost exclusively caused by autonomously functioning thyroid adenomas in cats, few cases of hyperthyroidism secondary to functional thyroid gland adenomas have been reported in dogs.[6,7]

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Abbreviations

cTSH	Canine thyroid stimulating hormone
DICOM	Digital Imaging and Communications in Medicine
T_4	Thyroxine
T_3	Triiodothyronine

Thyrotoxicosis, regardless of its origin, causes clinical signs in dogs consistent with those reported in other species, including polyuria, polydipsia, and weight loss.[1,8,9] Thyrotoxicosis refers to the biochemical and physiologic manifestations of excessive circulating concentrations of thyroid hormone.[9] Hyperthyroidism is a term reserved for disorders that result in the overproduction of hormone by a patient's thyroid gland.[9] However, thyrotoxicosis is not always attributable to hyperthyroidism.[9,10] In people, several terms are used to describe the means by which exogenous thyrotoxicosis resulting from ingestion of excessive amounts of thyroid hormone[11] occurs, including thyrotoxicosis factitia,[12] surreptitious thyrotoxicosis, occult factitial thyrotoxicosis,[13] and thy-

reoidismus medicamentosus.[14] The term thyrotoxicosis factitia veterinarius has been used to identify cases of thyrotoxicosis secondary to the accidental dosing of human patients with veterinary (T_4) preparations.[15] Excessive thyroid hormone administration in a hypothyroid patient, a form of thyrotoxicosis factitia, is an obvious potential cause for thyrotoxicosis and should be easy to exclude via a thorough anamnesis.[1,16]

Dietary sources of excessive thyroid hormone may also occur. Numerous reports[17–20] of thyrotoxicosis secondary to consumption of ground beef contaminated with thyroid tissue, so-called hamburger thyrotoxicosis, have been reported in the human literature. Thyrotoxicosis in a dog secondary to the consumption of feces from a housemate that was receiving levothyroxine has been reported in the veterinary literature.[21] Two recent reports[22,23] of thyrotoxicosis in dogs secondary to consumption of diets containing raw meat or dried gullets, presumably contaminated with thyroid tissue, have also been published. To the authors' knowledge, there are no other published reports of thyrotoxicosis in dogs secondary to consumption of commercially available dog foods or treats. The purpose of the study reported here was to describe the clinical, laboratory, and scintigraphic findings in dogs with exogenous thyrotoxicosis attributable to consumption of commercially available dog foods or treats.

Materials and Methods

Case selection—Electronic medical records of Advanced Veterinary Medical Imaging from January 1, 2008, through December 31, 2009, were searched to identify dogs with a diagnosis of exogenous thyrotoxicosis attributable to dietary consumption of thyroid hormone. Only dogs receiving commercially available dog food or treats were included. This retrospective search identified 1 case. Subsequent cases were identified prospectively from January 1, 2010, through December 31, 2013, and included dogs evaluated at Advanced Veterinary Medical Imaging (n = 10), the Animal Endocrine Clinic (1), The Ohio State University Veterinary Medical Center (1), and the Veterinary Specialty Hospital (1). Dogs were included if the diagnosis was made on the basis of laboratory evaluation alone (n = 1) or by laboratory evaluation and scintigraphy of the thyroid gland (13).

Data collection and medical records review—Medical records of 1 dog with a diagnosis of exogenous thyrotoxicosis were evaluated. Data from the remaining 13 dogs were collected prospectively. Information on breed, sex, age, diet (including treats fed), results of physical examinations and laboratory tests, and treatments was recorded. Scintigraphy results were reviewed for all dogs that underwent evaluation of thyroid lobe size, shape, and symmetry as well as measurement of the thyroid gland-to-salivary gland sodium pertechnetate 99m Tc uptake ratio (n = 13). Thoracic views of all thyroid gland scintigraphy scans were evaluated for evidence of ectopic cranial mediastinal thyroid disease. Owners were contacted to confirm clinical status of dogs following discontinuation of the suspect food or treat if follow-up was incomplete in the medical record.

Scintigraphy—Thyroid gland scintigraphy was performed following published guidelines.[24] Specific scintigraphic acquisition parameters including the gamma camera used, radionuclide dose, and decisions regarding sedative use as well as imaging parameters (eg, image matrix, image counts, and image acquisition time) varied by institution. Images were saved in DICOM format, and all images were evaluated by 1 author (MRB) with dedicated DICOM viewing software.[a] Analysis of scintigraphic images, including determination of the thyroid gland-to-salivary gland sodium pertechnetate Tc 99m uptake ratio, was performed according to guidelines described elsewhere.[24]

Serum thyroid hormone concentration determinations—Serum concentrations of thyroid hormones (total T_4, free T_4, and cTSH) and autoantibodies against T_4, T_3, and thyroglobulin on initial evaluation and following diet adjustment were determined by commercial veterinary laboratories.[b–d] Serum T_4 concentrations on initial evaluation were verified for 11 of 14 dogs by either the same (6/11) or another (5/11) commercial laboratory as allowed by owner compliance.

Measurement of T_4 immunoreactivity in food samples—Owners of the prospectively identified dogs were asked to provide descriptions and, if available, samples of the meat-based dog foods or treats being fed at the time of diagnosis. Thyroxine concentrations in samples of meat-based food or treats that were being fed to affected dogs at the time of diagnosis as well as a convenience sample of 10 arbitrarily selected commercial dog foods[e–n] and 6 samples of different cuts of beef muscle (n = 5) or liver (1) purchased at a grocery store were evaluated. All commercially available dog foods or treats were maintained in sealed food storage bags at room temperature (approx 20° to 26.1°C) prior to processing. Samples of beef muscle and liver were maintained under refrigeration (1.7° to 3.3°C) prior to processing.

Extraction of T_4 from various food samples followed a modification of a described method for extraction of thyroid hormone from freeze-dried fecal samples.[25] Briefly, several grams of each sample was placed in a resealable zippered food storage bag and thoroughly pulverized with a hammer. Samples were then ground with a mortar and pestle for 5 minutes. The consistency of samples varied from a powder to a paste by the end of this process.

Three 100-mg aliquots were collected from each sample, and each was placed into a tube[o] for processing. One milliliter of 70% ethanol was added to each tube and subsequently shaken (6 m/s for 60 seconds) in a bead homogenizer.[p] Tubes were then centrifuged at 12,000 × *g* for 10 minutes, and the supernatants were collected for T_4 assay. The T_4 concentration of each sample was determined with a validated radioimmunoassay kit[26,q] in accordance with the manufacturer's instructions. Samples (25 µL) were assayed by directly adding them to the T_4 assay tubes. All samples were assayed in duplicate. Assay of 70% EtOH alone did not affect the binding of ^{125}I-T_4 to the assay tube (ie, no different from assay of the zero standard). Assay values greater than

the highest standard curve point (196 nmol/L) were assigned this value for purposes of evaluation.

Statistical analysis—Data are given as median and range. A dot plot was generated for subjective comparison of immunoreactive T_4 concentrations in suspect foods and other commercially available products. Serum concentrations of total T_4 and free T_4 measured in blood samples collected from affected dogs at the time of initial evaluation were compared with serum total T_4 and free T_4 concentrations from samples obtained after discontinuation of foods or treats suspected to be the cause of thyrotoxicosis. Differences were analyzed with the Wilcoxon matched-pairs signed rank test. Statistical tests were performed with commercially available software.[r] Values of $P < 0.05$ were considered significant.

Results

Evaluation and diagnosis—Affected dogs (7 neutered males and 7 spayed females) had a median age of 10 years (range, 3 to 14 years). There were 5 mixed-breed dogs, 2 Shih Tzus, and 1 each of Border Collie, Chihuahua, Japanese Chin, Maltese, Pug, Soft Coated Wheaten Terrier, and Yorkshire Terrier. Median body weight was 10 kg (22.0 lb; range, 1.8 to 29 kg [4.0 to 63.8 lb]).

At initial evaluation, 4 dogs had no apparent clinical signs and the thyrotoxicosis was initially identified on the basis of high total T_4 concentrations (6.4 to 7.3 µg/dL; laboratory reference range, 1 to 4 µg/dL) during serum biochemical analysis as part of a routine health screening. The remaining 10 dogs had clinical signs consistent with thyrotoxicosis, including polydipsia and polyuria (n = 4 dogs), weight loss alone (2), or weight loss in combination with the following: polydipsia and polyuria (2), decreased appetite (1), or agitation (1).

Total serum T_4 concentrations were high (median, 8.8 µg/dL; range, 4.65 to 17.4 µg/dL) in all 14 dogs. Serum concentrations of free T_4, measured by equilibrium dialysis, were greater than the upper limit of the laboratory reference range in 12 of the 13 dogs that underwent the test (median, > 100 pmol/L; range, 31 to > 100 pmol/L; reference range, 8 to 48 pmol/L). Serum tests for autoantibodies against T_4 and T_3 were performed for 7 of the 14 dogs, and all had negative results. Serum tests for autoantibodies against thyroglobulin were negative in 9 of 9 dogs tested. Serum cTSH concentrations were measured in 11 of 14 dogs and were below the lower limit of the reference range in 9; values for the remaining 2 dogs (which had total serum T_4 concentrations of 5.5 and 6.7 µg/dL) were within the lower region of the reference range (0.07 and 0.08 ng/mL, respectively; reference range, 0.05 to 0.42 ng/mL).

All dogs that were evaluated by scintigraphy (n = 13) had a diffuse, bilateral, and symmetric reduction (relative to regional salivary tissue) in the uptake of sodium pertechnetate Tc 99m by eutopic, ventral cervical thyroid tissue (**Figure 1**). This reduction in radionuclide uptake was confirmed by measurement of the thyroid gland-to-salivary gland sodium pertechnetate 99m Tc uptake ratio (median, 0.26; range, 0.12 to 0.64; expected value, approx 1.0). Other than this finding, only normal areas of radionuclide uptake (ie, mandibular, parotid, and zygomatic salivary glands) and normal radionuclide distribution (ie, cardiac silhouette) were observed. No areas of abnormal radionuclide uptake from the nasal planum to the diaphragm were noted. No evidence of hyperfunctional ectopic thyroid tissue was detected in any of dogs that underwent thyroid gland scintigraphy.

Assessment of potential dietary causes of exogenous thyrotoxicosis—All 14 dogs were being fed all-meat or meat-based varieties of commercially available dog foods or treats at the time of diagnosis of exogenous thyrotoxicosis. All samples or descriptions of the suspect foods or treats provided by clients were of a similar form (sliced or rolled jerky style).

Owners of the first 3 dogs identified in this series provided descriptions of the suspect products, including form, predominant color of the bag, and the store from which the product was purchased, but without sufficient brand information to ensure confident identification of the specific product involved. Owners of the remaining 11 dogs were able to provide samples of the foods or treats being fed at the time of diagnosis. Specifically, owners of the fourth through ninth dogs identified provided 7 samples of 5 brands of food or treats[s–w] that were analyzed for immunoreactive T_4 concentrations. Owners of the 10th through 14th dogs identified provided 5 samples of 4 brands of food or treats,[u,x–z] including 1 of the brands previously identified and analyzed. These last 5 samples were used to confirm the identity of the products being fed but were not analyzed because determination of the immunoreactive T_4 concentrations in these samples was not considered necessary to confirm the diagnosis of exogenous thyrotoxicosis. Five of the 8 identified products were labeled as

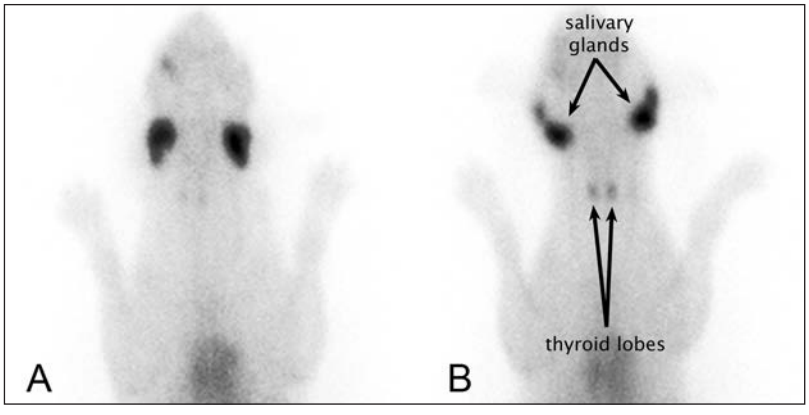

Figure 1—Dorsoventral scintigraphic images of a dog with exogenous thyrotoxicosis at the time of diagnosis (A) and 2 months after cessation of feeding a commercially available meat-based product that was suspected of containing high concentrations of thyroid hormone (B). Images were obtained following administration of sodium pertechnetate Tc 99m. A—Notice the marked, bilateral, and symmetric reduction of radionuclide uptake by the thyroid lobes relative to the parotid salivary glands, making identification of normal thyroid tissue difficult. B—The image obtained at follow-up (at which time serum concentrations of total and free T_4 were within the reference ranges) reveals increased symmetric radionuclide uptake in both lobes of the thyroid gland, compared with that in panel A.

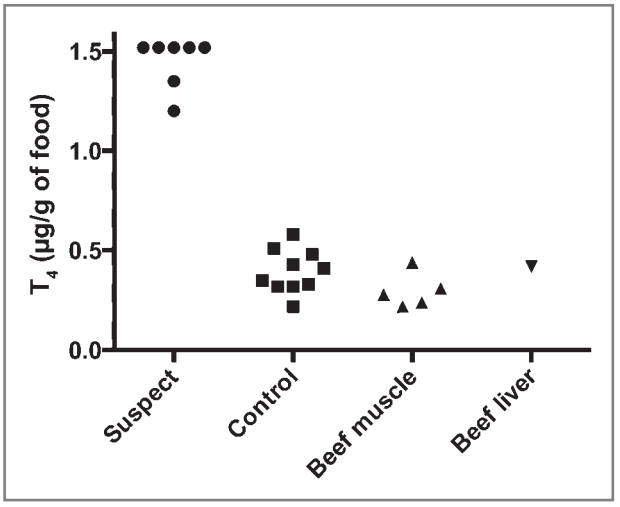

Figure 2—Thyroxine concentration (mg/g of food) in various foods or treats. Values were determined on the basis of immunoreactivity as assessed with radioimmunoassay. Suspect foods included 7 samples of 5 brands of food provided by the owners of dogs suspected to have exogenous thyrotoxicosis; control foods were arbitrarily selected commercially available (dry or jerky style) dog foods and beef muscle and liver samples that were obtained from a grocery store.

dog treats[t,u,w–y] (3 brands tested), and the other 3 were labeled as dog foods[s,v,z] (2 brands tested).

Analysis of the 7 samples of food or treats that were tested revealed that the median concentration of immunoreactive T_4 was 1.52 µg of T_4/g, whereas that for samples of commercial dog food used as controls and for beef and liver samples was 0.38 µg of T_4/g and 0.30 µg of T_4/g, respectively (**Figure 2**). Differences were not compared statistically because independence of all samples could not be confirmed.

Treatment and outcome—Owners were advised to discontinue feeding of the suspect foods or treats in favor of any other commercially available kibble-style dog food. A minimum of 4 weeks after diagnosis and discontinuation of the suspect foods or treats, all 14 dogs underwent recheck examinations and blood sample collection. Total serum T_4 concentrations had decreased significantly ($P < 0.001$), and values were within the reference range for all dogs (median, 1.9 µg/dL; range, 1.0 to 4.0 µg/dL). Serum concentrations of free T_4 also decreased significantly ($P = 0.016$) for 8 dogs that had paired tests performed, and values were within the reference range for these dogs (median, 15 pmol/L; range, 8 to 30 pmol/L). In 4 dogs that had paired serum cTSH concentrations evaluated, values were within the reference range at the recheck examination. All dogs with clinical signs on initial evaluation had complete resolution of signs following discontinuation of feeding the suspect products. Repeated thyroid gland scintigraphy was performed in 1 of the 14 dogs and confirmed a bilateral, symmetric relative increase in the thyroid gland-to-salivary gland sodium pertechnetate Tc 99m uptake ratio, compared with the initial thyroid gland scintigraphy (Figure 1).

Discussion

All 14 dogs of this report developed a reversible thyrotoxicosis following consumption of various commercially available meat-based dog foods or treats that were known or suspected to contain high concentrations of T_4, presumably secondary to contamination with thyroid tissue.

Hyperthyroidism is uncommon in dogs and is almost exclusively caused by autonomously functional thyroid gland carcinomas.[2–5] Authors of previous studies have reported the prevalence of thyrotoxicosis in dogs with thyroid carcinoma as 0 of 23 (0%),[2] 6 of 29 (21%),[3] 13 of 58 (22%),[4] and 21 of 39 (54%).[5] A much smaller number of cases of hyperthyroidism in dogs have been attributed to autonomously functional thyroid gland adenomas.[6,7] Physical examination failed to identify a palpable mass in the ventral cervical area in any of the dogs in our study. However, autonomously functional thyroid nodules may result in high circulating thyroid hormone concentrations, despite small, difficult to palpate thyroid gland tumors.

Scintigraphic examination provides valuable information regarding thyroid gland anatomy and physiology and can play an integral role in the diagnosis and management of canine thyroid disease.[24,27,28] For dogs with hyperthyroidism resulting from thyroid gland neoplasia, scintigraphy is generally considered to be the imaging technique of choice for detecting and delineating all hyperfunctioning thyroid tumor tissue. Advantages of scintigraphy include its ability to differentiate bilateral versus unilateral thyroid gland disease, assess thyroid gland tumor size and uptake of radionuclide, and identify ectopic or metastatic thyroid tissue.[3,24,28,29] In dogs, autonomously functional thyroid tissue neoplasia may develop in ectopic thyroid tissue in locations ranging from the sublingual region to the heart base.[29–36] Scintigraphy was performed in 13 of the 14 dogs in our study, and all 13 dogs had a bilateral and symmetric reduction in radionuclide uptake in both eutopic cervical thyroid lobes as determined by a low thyroid gland-to-salivary gland sodium pertechnetate Tc 99m uptake ratio. No evidence for thyroid gland tumor or areas of ectopic radionuclide uptake was noted in any of the dogs. These scintigraphic findings excluded the possibility of hyperthyroidism secondary to an autonomously functional thyroid tissue tumor in either eutopic (cervical) or ectopic locations.

Bilateral and symmetrically reduced radionuclide uptake by the thyroid gland could result from primary hypothyroidism.[27,37,38] Autoimmune thyroiditis is a cause for hypothyroidism in dogs.[39] Autoantibodies against circulating T_3 or T_4 found in dogs with autoimmune thyroiditis have been reported to interfere with the immunologic assay methods used to measure thyroid hormone concentrations, leading to falsely high or low concentrations, depending on the assay method.[40,41] Seven of the 14 dogs in the present study underwent testing for autoantibodies against T_3 and T_4, but none were detected. Serum free T_4 concentrations measured by equilibrium dialysis should not be affected by the presence of autoantibodies, which cannot diffuse across the semipermeable membrane used to separate unbound or free T_4 from the much larger amount of protein-bound T_4.[42,43] Free T_4 concentrations were found to be greater than the upper limit of the laboratory reference range in 12 of 13 dogs for which

this was measured, consistent with thyrotoxicosis. In dogs with primary hypothyroidism, serum cTSH concentrations are expected to be greater than, or at the upper end of, the laboratory reference range. In contrast, cTSH concentrations were less than the lower limit of the reference range in 9 of the 11 dogs evaluated and at the low end of the reference range in the remaining 2 dogs. Hence, although not all tests were performed for all dogs, none of the available laboratory evaluations in these dogs supported the possibility of hypothyroidism or autoimmune thyroiditis, and all of the laboratory results supported the presence of thyrotoxicosis.

Concentrations of total and free serum T_4 have been shown to increase following sample storage for 5 days at high temperatures (37°C).[44] However, no evidence of delayed sample processing was noted, the high thyroid hormone concentrations found in dogs of the present study exceeded that reported with delayed sample processing, and clinical signs of thyrotoxicosis (noted in 10/14 dogs) would not have been explained by increased thyroid hormone concentrations secondary to delayed sample processing.

Hypothyroidism is a relatively common disease in dogs, and many dogs are treated by administration of thyroid hormone. Exogenous thyrotoxicosis in hypothyroid dogs has been described secondary to miscalculation of doses, miscommunication between veterinary staff and clients, or, rarely, greater absorption of exogenous thyroid hormone than expected.[39] Exogenous thyrotoxicosis attributable to this factor was easily excluded on the basis of a thorough anamnesis. No dogs in this study or their animal or human housemates were receiving exogenous thyroid hormone treatment.

Hyperthyroxinemia secondary to inadvertent consumption of raw food diets contaminated with thyroid tissue has been previously reported in dogs.[22,23] In addition, numerous reports[16–20] of thyrotoxicosis secondary to consumption of ground beef contaminated with thyroid tissue, so-called hamburger thyrotoxicosis, have been reported in the human literature.

In the dogs of this report, the diagnosis of exogenous thyrotoxicosis was supported by results of initial laboratory evaluations in all dogs, combined with scintigraphy in 13 of 14 patients. The diagnosis of thyrotoxicosis secondary to dietary consumption of exogenous thyroid hormone was further supported by resolution of high serum concentrations of total T_4 (in 14/14 dogs) and free T_4 (in 8/8 dogs) and by the resolution of clinical signs after owners stopped feeding the suspect products. This was further supported by results of analysis of 7 samples of suspect meat-based food or treats fed to 6 of the 14 dogs, which had a median concentration of 1.52 µg of immunoreactive T_4/g, whereas values for the convenience sample of other commercial dog foods (n = 10) and beef muscle or liver samples (6) were 0.38 and 0.30 µg of T_4/g, respectively. These findings also supported that consumption of the suspect foods or treats was the source for the dogs' thyrotoxicosis. Indeed, it is conceivable that the concentration of immunoreactive T_4 in the meat-based food or treats was an underestimate of the true content of thyroid hormones; much of the thyroid hormone in these products would likely be present in the form of thyroglobulin,[11] and the antibody used in the T_4 radioimmunoassay may not efficiently react with the T_4 molecules bound in thyroglobulin. Furthermore, although it would have been interesting to measure T_4 concentration in the samples of suspected foods that were not analyzed, the authors felt it was not necessary to confirm the diagnosis of exogenous thyrotoxicosis. Copies of laboratory analysis results for the suspect foods and treats as well as detailed information about the products involved, including brands, manufacturers, and lot numbers (when available), were provided to the FDA to facilitate independent evaluation. Physical samples of every suspect product provided to our hospital by clients were also forwarded to the FDA for independent analysis.

The small number of cases of exogenous thyrotoxicosis that we identified over a 5-year period suggests that the problem of contamination of commercial dog food or treat products with thyroid tissue may be sporadic. Alternatively, the number of dogs identified could be limited by the nature of the products identified as containing high concentrations of thyroid hormone (ie, most were labeled as dog treats). The risk of thyrotoxicosis caused by consumption of meat-based products contaminated with thyroid tissue should be proportional to the percentage of the dog's caloric intake of the T_4-containing product. Most (5/8) of the suspect products identified in this study were labeled as dog treats, and although the remaining 3 products were labeled as complete and balanced dog foods, the labeling on the packages strongly suggested they could be fed as a supplement to other dog foods. Thus, the T_4-containing treats or dog food products might have been consumed in insufficient quantities to induce thyrotoxicosis in many dogs. One limitation of the present study was that we were not able to obtain accurate information about the proportion of each dog's daily caloric intake from the suspect products owing to variation in the amount fed daily for many dogs. The size of a dog could also play a role in the amount of a product that would have to be consumed to increase circulating T_4 and free T_4 concentrations to a degree that would exceed the reference range, cause clinical signs, or both. In support of this is the low median body weight of the dogs in this report (10 kg), which suggests that smaller dogs were at higher risk for developing exogenous thyrotoxicosis.

Dissection of the cervical region of carcasses at a slaughterhouse may lead to the unintended inclusion of thyroid tissue in harvested meat because of the proximity of the thyroid gland to the surrounding musculature and the difficulty of separating these tissues. In August 1985, following an outbreak of thyrotoxicosis among people in Minnesota,[17] the USDA ordered that the procedure of gullet trimming for the collection of edible meat products be discontinued at all meat packing plants.[45] To our knowledge, no cases of human thyrotoxicosis secondary to consumption of commercially available meat products have been reported since the late 1980s in the United States. Currently, however, the USDA lists beef gullets and tracheas as acceptable for use in pet foods.[46] Pet food production economics often result in the use of materials for feeding pets that are different than those used for human consumption. We contacted the manufacturers of all of the foods involved in our study and shared our data and concerns

with them. Manufacturer representatives indicated they were actively investigating the problem. The presence of high T_4 concentrations in a variety of pet foods or treats sold under different labels suggests that the problem of thyroid tissue contamination of such items may be widespread and not confined to only a few products or manufacturers. A thorough history, including dietary information, is essential for evaluation of dogs with signs of hyperthyroidism in conjunction with high thyroid hormone concentrations, especially if endogenous causes and other causes of exogenous thyrotoxicosis have been ruled out. Results of the present study indicate that thyrotoxicosis attributable to dietary consumption of excessive thyroid hormone can be reversed by diet change alone.

a. OsiriX, Pixmeo, Geneva, Switzerland.
b. Antech Diagnostics, Irvine, Calif.
c. IDEXX Laboratories Inc, Westbrook, Me.
d. Diagnostic Center for Population and Animal Health, Michigan State University, Lansing, Mich.
e. Organic Chicken and Brown Rice Recipe Sticks, Trader Joe's, Monrovia, Calif.
f. Liv-A-Littles 100% Beef Protein Treats, Halo Purely for Pets, Tampa, Fla.
g. Canine R/D Dry Dog Food, Hill's Pet Product Inc, Topeka, Kan.
h. Pro Treat Raw Natural's Real Beef with Berries and Flaxseed, MiracleCorp, Dayton, Ohio.
i. Jerky Strips with Real Beef, Hill's Pet Product Inc, Topeka, Kan.
j. Roll-A-Rounds Chunky Lamb Formula, Natural Balance Pet Foods, Burbank, Calif.
k. Beef Jerky, Milo's Kitchen, Big Heart Pet Brands, San Francisco, Calif.
l. Farmers Market Chicken and Vegetables Flavored Dog Treat Strips, Plato, Fresno, Calif.
m. Chef Michael's Grilled Sirloin Steak Flavor, Nestlé Purina PetCare Co, St Louis, Mo.
n. Science Diet Light Dry Dog Food, Hill's Pet Nutrition Inc, Topeka, Kan.
o. Catalog No. 19-620C, 2 mL capacity containing 4-2.38 mm stainless steel beads, Omni International, Kennesaw, Ga.
p. Omni bead ruptor homogenizer, Omni International, Kennesaw, Ga.
q. Canine T4 Coat-A-Count, Siemens Healthcare Diagnostics, Los Angeles, Calif.
r. Prism, version 6, GraphPad Software Inc, La Jolla, Calif.
s. Natural Beef Air Dried Dog Food, The Real Meat Co, Montrose, Calif.
t. Lamb Jerky Treats, The Real Meat Co, Montrose, Calif.
u. Natural Beef Jerky Treats, The Real Meat Co, Montrose, Calif.
v. Thawed Beef Raw Dog Food, Whisker & Paws, Costa Mesa, Calif.
w. Beef Recipe Jerky Strip, Trader Joe's, Monrovia, Calif.
x. Pup-Peroni Stick Treats, Big Heart Pet Brands, San Francisco, Calif.
y. Venison Jerky Treats, The Real Meat Co, Montrose, Calif.
z. Max Meat Air Dried Dog Food, Only Nature Pet, Boulder, Colo.

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