Correlation of Racing and Reproductive Performance in Greyhounds with Response to Thyroid Function Testing

Two-hundred eighteen greyhounds were evaluated for thyroid and adrenal function using combined TSH response and low-dose dexamethasone suppression test. Dogs with abnormal serum cortisol suppression in response to dexamethasone were eliminated from the data set. Range of basal serum thyroid hormone concentrations for euthyroid dogs with adequate response to TSH was 0.7 to 3.6 μg/dl thyroxine (T4) and 62.2 to 265.6 ng/dl triiodothyronine. Basal serum thyroid hormone concentrations and increase in serum thyroxine in response to TSH were compared with reproductive and racing performance. Poor reproductive performance was not associated with low serum T4 concentration or decreased responsiveness to TSH. There was no correlation between basal thyroid hormone concentrations or increase in T4 after TSH with racing performance. The prevalence of serum antithyroglobulin antibodies in the greyhound population surveyed was 3.6%.

Karin M. Beale, DVM
Mark S. Bloomberg, DVM, MS
James Van Gilder, BS
Bernard B. Wolfson, PhD
Karen Keisling, BS

O

From the Department of Small Animal Clinical Sciences and Center for Veterinary Sports Medicine (Bloomberg, Beale, Van Gilder, Keisling), University of Florida, Gainesville, Florida 32610-0126. Dr. Wolfson's address is Daniels Pharmaceuticals, 2527 25th Avenue, St. Petersburg, Florida 33173.

Published as Journal Series No. 261 of the College of Veterinary Medicine, University of Florida.

Introduction

Hypothyroidism is a common endocrinopathy reported in dogs.[1] Dogs with hypothyroidism can be presented with multiple systemic abnormalities including dermatological disorders, reproductive abnormalities, lethargy, mental dullness, bradycardia, and myopathies.[1] Poor racing performance and infertility in male and female greyhounds often is attributed to hypothyroidism. Diagnosis of hypothyroidism is complicated by the fact that greyhounds may have lower serum basal concentrations of thyroxine than other breeds.[2] A survey conducted in Australia involving 33 racing greyhounds suggested a clinical correlation between serum thyroxine (T4) levels and performance capacity.[3] In addition to a subjective clinical viewpoint of decreased racing performance, many with serum T4 concentrations below 1.4 μg/dl demonstrated anemia, hyperpigmentation, and poor coat quality. The majority of those dogs responded clinically with better racing performance to treatment with L-thyroxine.[3]

Hypothyroidism may result in infertility in male and female dogs.[1] Hypothyroid bitches may have irregular estrous cycles, uterine inertia, decreased litter size, and failure to conceive.[1] Chronic administration of anabolic steroids to female greyhounds to suppress estrus while racing contributes to infertility in female dogs. Hypothyroidism in male dogs may be manifested as decreased libido and sperm production.[1] Diagnosis of hypothyroidism commonly is made based on clinical signs and low basal serum thyroxine concentrations. However there are numerous variables which can affect basal serum thyroid hormone concentrations, making them an unreliable indicator of actual thyroid function.[1,4] These variables include drug therapy, hyperadrenocorticism and other systemic illnesses, age, and breed. The greyhound in particular has been noted to have lower serum T4 concentrations than other breeds.[2] In addition, most female greyhounds are treated with methyltestosterone for suppression of estrus while racing, and this also may affect total serum T4 and T3 concentrations. The TSH stimulation test which measures thyroid reserve generally is regarded as a more accurate indicator of thyroid function than basal hormone concentrations.[5] The maximum serum thyroxine increase in response to TSH occurs between five to eight hours after IV administration.[6,7] Serum thyroxine concentrations in euthyroid dogs are at least 2.5 times greater than baseline three hours after IV administration of 2.5 TSH.[7]

Administration of glucocorticoids decreases basal serum thyroid hormone concentrations.[1] Stress causes several adaptive hormone responses, most notably increased secretion of catecholamines, corticosteroids, and adrenocorticotropic hormone.[8] It is possible that in the stressed racing greyhound, serum thyroid hormone concentrations may be decreased due to high endogenous cortisol levels. Administration of levothyroxine to racing greyhounds is not an uncommon practice in the industry. Clinical signs such as poor racing performance and reproductive abnormalities could be compatible with hypothyroidism.

The two main causes of hypothyroidism in dogs are lymphocytic thyroiditis and idiopathic thyroidal atrophy.[1] Lymphocytic thyroiditis has been shown to be a hereditary condition in dogs.[9] Antithyroglobulin antibodies are present in lymphocytic thyroiditis, and testing for these antibodies can be used to estimate prevalence of thyroiditis in a population of dogs. The purpose of this study was threefold: 1) to establish normal basal thyroid hormone concentrations for greyhounds, 2) to correlate athletic and reproductive performance with basal serum thyroid hormone concentrations and results of thyroid function tests, and 3) to determine prevalence of antithyroglobulin antibodies (ATA) in the greyhound population.

Materials and Methods

Serum samples were obtained from 218 healthy greyhounds in the racing industry. Investigators were unaware of the performance and reproductive histories of these animals. No dogs currently receiving thyroid hormone supplementation were included in the study. The sample population included intact male and female dogs ranging in age from 11 months to 10 years. Sample size included 97 juvenile racing dogs or dogs in training (11 to 24 months of age), 96 brood bitches (3 to 11 years of age), and 25 stud dogs (3 to 10 years of age) from six different racing greyhound farms throughout Florida. All juvenile female dogs were receiving methyltestosterone for suppression of estrus.

Each dog was weighed and 10 ml of venous blood withdrawn from the jugular vein for determination of baseline serum T3 and T4 and cortisol concentrations. A butterfly infusion catheter was placed intravenously in the cephalic vein. Following placement of the catheter, each dog received dexamethasone[a] (0.01 mg/kg body weight IV), followed by a saline flush and thyrotropin (TSH)[b] (0.1 U/kg body weight), followed by a saline flush. Total dose of TSH was no less than 2.5 U and no greater than 5.0 U per dog. Approximately 10 ml of venous blood was withdrawn from the jugular vein of each dog at four and eight hours for analysis of T3 and T4 and cortisol levels respectively. Blood samples were centrifuged within two hours of collection and serum stored at -70°C.

Serum samples were analyzed for concentrations of T3 and T4 by a commercial modified solid-phase radioimmunoassay (RIA)[c,d] that has been validated.[10] An estimation of thyroid function was made based on results of TSH stimulation testing. A dog was classified as having an adequate response to TSH if serum T4 concentration post-TSH was greater than or equal to 3.0 μg/dl. Serum samples were analyzed for ATA using an enzyme-linked immunosorbent assay (ELISA).[10] Antibody levels were reported in relative antibody units (RAU), compared to a standard positive control serum. Ten RAU is considered suggestive of thyroiditis.[10]

Serum samples were analyzed for concentrations of cortisol pre- and postdexamethasone administration by a modified solid phase RIA[e] that has been validated for the dog.[11] A postdexamethasone serum concentration of cortisol >1.0 μg/dl was considered to be abnormal and suggestive of endogenous hyperadrenocorticism. Any dog that did not suppress adequately in response to dexamethasone was excluded from the data set.

A questionnaire was sent to the participating greyhound farms to be filled out for each dog surveyed. Farm owners were unaware of thyroid function test results. Juvenile dogs were evaluated for their racing performance, and stud dogs and brood bitches were evaluated for reproductive performance [Table 1]. Dogs were classified as having poor reproductive or racing performance if owners responded with one or more positive answers to the questions asked.

Range of normal baseline thyroid hormone values were determined after eliminating any dogs

Table 1

Questions Asked of Greyhound Owners for Each Greyhound Evaluated

Questions asked regarding juvenile dogs:

1. Does the dog have poor racing performance?
2. Is the dog easily fatigued or does it have a lack of stamina?
3. Did the dog fail to make the track?

Questions asked regarding brood bitches:

1. Does the dog have irregular heat cycles?
2. Has the dog had difficulty in conceiving?
3. Has the dog had uterine inertia?

Questions asked regarding stud dogs:

1. Does the dog have decreased libido?
2. Does the dog have decreased sperm production?
3. Has the dog had difficulty in impregnating bitches?

Juvenile dogs included male and female dogs from 11 to 24 months of age. Brood bitches included female dogs from 3 to 11 years of age kept for breeding purposes, and stud dogs included male dogs from 3 to 10 years of age kept for breeding purposes.

with a decreased response to TSH from the data set. In order to correlate racing performance and reproductive performance with thyroid function, the dogs were divided into two groups: group A racing dogs (juveniles) and group B breeding animals (brood bitches and stud dogs). Dogs in group A were divided into two categories based on response to the questionnaire: dogs with poor racing performance and dogs with normal racing performance. To determine the relationship between thyroid function and racing performance, baseline T3 and T4 serum concentrations and post-TSH T4 concentrations were compared for those dogs with normal and poor racing performance by an unpaired student t test.

Dogs in group B were divided into two categories based on response to the questionnaire: dogs with poor reproductive performance and dogs with normal reproductive performance. To determine the relationship between thyroid function and reproductive performance, baseline T3 and T4 serum concentrations and post-TSH T4 concentrations were compared for those dogs with normal and poor reproductive performance by an unpaired student t test. The correlation between serum T3 and T4 concentrations was determined with Pearson's correlation coefficient of determination (r^2).

Results

Twelve breeding dogs (group B) had an abnormal response to dexamethasone suppression at eight hours and were excluded from the data set. All dogs with abnormal dexamethasone suppression results were bitches ranging from 4.0 to 9.5 years; mean age was 6.0 years (SD=3.0). All juvenile dogs had a postdexamethasone serum cortisol <1.0 μg/dl. The following results pertain to the 206 dogs with adequate suppression of serum cortisol in response to dexamethasone.

Questionnaires were returned on 168 dogs (81.6%): 69 bitches, 22 studs, and 77 juvenile dogs. Eight juveniles were classified as poor racing performers, and 34 bitches and two studs were classified as having reproductive abnormalities. Two juveniles had a very elevated basal serum T3 concentration (T3 >400 ng/dl), and these T3 values were eliminated from the data set. Sixty-eight dogs had a decreased response to TSH stimulation (post-TSH T4 <3.0 μg/dl) [Table 2]. Mean basal serum thyroid hormone concentrations were greater for those dogs with a post-TSH T4 ≥ 3.0 μg/dl than for those dogs with post-TSH T4 <3.0 μg/dl [Table 2]. Range of basal thyroid hormone values for dogs with adequate response to TSH stimulation was 0.70 to 3.60 μg/dl T4 and 62.2 to 265.6 ng/dl T3. There were no significant differences using a two sample t test in basal T3, T4, or increase in T4 in response to TSH stimulation between dogs with poor and adequate racing performance [Table 3]. Basal serum T4 was significantly higher ($p \leq 0.05$) for dogs with reproductive abnormalities than for

Table 2

Mean Serum Thyroid Hormone Concentrations (SD) Pre- and Postthyrotropin (TSH*) Administration in Greyhounds with Adequate Suppression of Serum Cortisol with Dexamethasone Administration

	All Greyhounds (n=206)	Greyhounds with Adequate Response to TSH (n=138)	Greyhounds with Decreased Response to TSH (n=68)
Basal T4 (μg/dl)	1.46 (0.63)	1.68 (0.64)	1.05 (0.40)
Post-TSH T4 (μg/dl)	3.51 (1.13)	4.11 (0.79)	2.31 (0.63)
Increase in T4 post-TSH (μg/dl)	2.05 (0.81)	2.44 (0.60)	1.26 (0.55)
Basal T3 (ng/dl)	141.31 (49.64)	149.30 (44.01)	125.54 (55.39)
Post-TSH T3 (ng/dl)	201.60 (56.46)	216.50 (52.13)	172.32 (53.13)
No. females	130	96	34
No. males	76	42	34
Mean age (SD)	3.8 (2.8)	3.1 (2.5)	5.1 (2.7)

*TSH stimulation tests were performed in dogs by administering 0.1 IU/kg body weight bovine TSH intravenously. Samples were collected prior to administration of TSH and four hours later. Dogs were considered to have an adequate response to TSH if serum T4 concentrations were ≥3.0 μg/dl four hours post-TSH.

dogs with no reproductive disturbances. There was no significant difference in serum T3 or increase in serum T4 in response to TSH between the two groups of breeding animals [Table 4].

Pearson's correlation coefficient of determination was 0.024 for all 206 dogs, $r^2=0.00$ for the 138 dogs with adequate response to TSH, and $r^2=0.075$ for the dogs with a poor response to TSH. Eight had ATA levels indicative of thyroiditis (RAU ≥10) [Table 5]. All with ATA were juveniles, and only two had poor racing performance. Two with ATA had a decreased response to TSH (post-TSH T4 <3.0 μg/dl), but both of these were evaluated as having adequate racing performance.

Discussion

Twelve dogs failed to have adequate suppression of basal cortisol concentrations in response to a low dose of dexamethasone. This is suggestive of endogenous hyperadrenocorticism; however it also can occur in stressed animals.[12] All dogs that had a decreased serum cortisol suppression in response to dexamethasone were older females in the breeding group. Mean age of dogs with abnormal dexamethasone response tests was 6.0 years (3.0 SD); age range was 3 to 10 years. Hyperadrenocorticism usually is seen in dogs six years of age or older.[1] None of the younger racing dogs had abnormal dexamethasone suppression results. Those with abnormal dexamethasone suppression test results were excluded from the data set because hyperadrenocorticism can affect basal serum thyroid hormone concentrations.[13]

Previous reports have suggested a correlation between racing performance and basal T4; however findings in this study do not support such a correlation. Basal T4 concentration was lower in juveniles with poor performance than those with adequate performance; however the difference was not statistically significant. In addition, the difference in T4 in response to TSH was minimal between the two groups, suggesting that the thyroid reserve of the two groups was similar. In fact, only one of the eight juveniles with poor racing performance lacked a post-TSH T4 concentration ≥3.0 μg/dl. Based on this information, it would appear that hypothyroidism is an uncommon cause of poor racing performance in the greyhound. The practice of administering L-thyroxine to dogs with poor racing performance is not justified; however the effect on racing performance of administering supraphysiological doses of thyroid hormone to a dog are unknown.

The 36 dogs with poor reproductive performance had a significantly higher basal T4 concentration than those with adequate reproductive performance ($p<0.05$). The change in T4 in response to TSH and the basal T3 concentration was very similar for the

Table 3

Mean Serum Thyroid Hormone Concentrations (± SD) in Juvenile Greyhounds with Adequate and Poor Racing Performance*

Greyhounds	Serum Thyroxine (T4) (μg/dl)	Serum Triiodothyronine (T3) (ng/dl)	Increase in T4 Post-TSH (μg/dl)	Age (yrs)	No. Females	No. Males
All dogs (n=77)	1.49 (0.52)	161.87 (45.32)	2.27 (0.63)	1.18 (0.15)	36	41
Poor racing performance (n=8)	1.31 (0.61)	161.16 (20.37)	2.29 (0.46)	1.19 (0.15)	3	5
Adequate racing performance (n=69)	1.51 (0.51)	161.96 (47.47)	2.27 (0.65)	1.16 (0.11)	33	36

*Dogs' performance was based on response to a questionnaire sent to owners in which questions were asked regarding stamina, lethargy, and racing abilities. The TSH stimulation tests were performed by administering 0.1 IU/kg body weight TSH IV. Blood was collected at time 0 and 4 hours for evaluation of response to TSH.

Table 4

Mean Serum Thyroid Hormone Concentrations (SD) in Adult Greyhounds with Reproductive Performance Determined by Response to a Questionnaire*

Greyhounds	T3 (ng/dl)	T4 (μg/dl)	Change in T4 in Response to TSH (μg/dl)	Age (yrs)	No. Males	No. Females
All greyhounds used for breeding purposes (n=91)	118.80 (32.04)	1.49 (0.73)	1.94 (0.90)	5.9 (1.9)	22	69
Poor reproductive performance (n=36)	124.30 (32.19)	1.69 (0.72)	1.97 (0.89)	5.5 (2.0)	2	34
Adequate reproductive performance (n=55)	115.42 (31.71)	1.36 (0.71)	1.92 (0.92)	6.1 (1.8)	20	35

*The dogs reproductive performance was based on response to a questionnaire sent to the owners for evaluation of the dog's past and present reproductive abnormalities.

Table 5

Greyhounds with Serum Antithyroglobulin Antibodies (ATA)

Dog No.	T3 (ng/dl)	T4 (μg/dl)	ATA (RAU)	Months	Sex	Racing Performance
97	165.6	1.5	22	12	F	Adequate
157	206.5	0.7	242	15	M	Adequate
162	440.7*	2.0	3791	15	F	Adequate
163	174.1	1.5	39	15	F	Poor
170	502.2*	2.3	2223	15	M	Adequate
208	139.6	2.0	22	15	F	Adequate
209	200.8	1.6	24.5	13	M	Poor
215	128.6	1.6	290	15	F	Adequate

Eight dogs had ATA > 10 relative antibody units (RAU) which is suggestive of thyroiditis. All were juvenile racing dogs.
*Indicates probable presence of T3 autoantibodies interfering with the assay and producing erroneously elevated values

two groups. This suggests that in most instances reproductive failure in the greyhound is due to factors other than hypothyroidism. The practice of treating racing bitches with anabolic steroids for extended periods to suppress estrus certainly is a major contributing factor to the reproductive problems that are encountered in greyhounds.

Basal serum thyroxine concentrations tend to decrease as dogs age.[14] The dogs in the group with poor reproductive performance were slightly younger than those in the group with normal reproductive function. The age difference may account for the difference in basal T4. Based on this assumption, one might expect juveniles to have higher serum thyroid hormone concentrations than those in the reproductive group; however thyroid hormone concentrations were very similar for the two groups. Administration of androgens can decrease basal thyroid hormone concentrations,[1] and administration of anabolic steroids to racing bitches may have decreased basal serum thyroid hormone concentrations in the younger dogs accounting for the similarity between the two groups.

Mean basal serum T4 concentration for all the greyhounds was 1.46 μg/dl (0.63 SD). The mean falls within most published ranges for T4 in euthyroid dogs;[1,15,16] however the range of basal serum T4 in greyhounds with adequate response to TSH stimulation was 0.7 to 3.6 μg/dl (mean 1.68 μg/dl, SD=0.64). A serum T4 concentration of 0.7 μg/dl would be considered low by most laboratories. Thirty of 138 greyhounds (27%) with adequate response to TSH had a serum T4 concentration <1.2 μg/dl. The TSH response test is a measure of thyroid reserve and not an indication of the presence or lack of thyroid pathology. A dog with early thyroiditis may have a normal response to TSH. It is possible that some of the greyhounds with low serum T4 had thyroid atrophy or lymphocytic thyroiditis but still had adequate functional thyroid tissue remaining to respond to TSH. Alternatively, greyhounds may have lower serum concentrations of thyroxine than other breeds, as suggested by other investigators.[2]

The correlation coefficient of determination is indicative of the strength of a straight line relationship between a factor and a response variable. An r^2 value of 1.0 indicates a perfect positive linear relationship between the two variables, and $r^2=0$ indicates that there is no linear relationship between the variables. There was minimal linear correlation between serum T3 and T4 concentration in the greyhounds studied. There was better correlation between serum T3 and T4 concentrations in the dogs with decreased response to TSH than in those with adequate response to TSH. A dog with a poor response to TSH with a low serum T3 concentration was more likely also to have a low serum T4 than a dog with a low serum T3 concentration and an adequate response to TSH. The lack of correlation in dogs with adequate TSH responses suggests a disequilibrium in thyroid hormone production, metabolic turnover, or conversion of T4 to T3. Few dogs had serum ATA, suggesting that lymphocytic thyroiditis was uncommon in the population examined. Perhaps the disequilibrium is due to early thyroid atrophy. Alternatively, methyltestosterone received by many of the dogs may affect serum thyroid hormone protein binding or metabolic turnover of the hormones, thereby disrupting the equilibrium.

The presence of ATA is suggestive of lymphocytic thyroiditis, one of the main causes of hypothyroidism in dogs. Only 8 of 218 dogs had detectable ATA (3.6%), indicating that thyroiditis was not a common disease in the population of greyhounds examined. A survey of 1,057 hospitalized dogs without endocrine disorders revealed that 13.2% of those dogs had ATA detectable by ELISA.[17] There did not appear to be any correlation between performance and presence of ATA. Only two of the dogs with ATA had a decreased response to TSH; however this does not indicate that the remaining six did not have thyroiditis as thyroid function usually remains within normal limits in the early stages of thyroiditis in both dogs and people.[18] Borzois with early thyroiditis had normal responses to TSH stimulation.[7]

Dogs with thyroiditis occasionally develop antibodies against thyroid hormones. Autoantibodies interfere with the radioimmunoassay used and produce falsely elevated results.[19] Autoantibodies to T3 usually are associated with the presence of autoantibodies to thyroglobulin.[20] Both of the dogs with a markedly elevated basal T3 (dog Nos. 162 and 170) also had antithyroglobulin antibodies; therefore it is likely that marked elevations in measured T3 were due to autoantibody interference in the assay.

In conclusion, poor reproductive performance was not associated with low serum thyroxine concentrations or decreased serum T4 increase in response to TSH, and there does not appear to be a correlation between basal serum thyroid hormone concentrations or TSH response and racing performance in the greyhound. The range of serum thyroxine concentrations for greyhounds with adequate response to TSH was 0.7 to 3.6 μg/dl. Twenty-seven percent of these dogs had a basal serum T4 <1.2 μg/dl. The prevalence of lymphocytic thyroiditis based on serum ATA is low in greyhound dogs.

[a] Azium, Schering Corp., Kenilworth, NJ
[b] Thyrotropic hormone, bovine origin, Sigma Chemical Co., St. Louis, MO
[c] Gamma coat 125I T3, Clinical Assays, Cambridge, MA
[d] Gamma coat 125I T4, Clinical Assays, Cambridge, MA
[e] Gamma coat 125I cortisol, Clinical Assays, Cambridge, MA

References

1. Feldman EC, Nelson RW. Hypothyroidism. In: Canine and feline endocrinology and reproduction. Philadelphia: WB Saunders, 1987:55–90.
2. Rosychuck RAW, Freshman JL, Olson PN, *et al.* Serum concentration of thyroxine and 3,5,3'-triiodothyronine in dogs before and after administration of freshly reconstituted or previously frozen thyrotropin releasing hormone. Am J Vet Res 1988;49:1722–5.
3. Taylor JR, Hauler AD. Endocrinology. In: Proceedings refresher course on greyhounds. The University of Sydney, Australia, 1983;64:449–66.
4. Ferguson DC. The effect of non-thyroidal factors on thyroid function tests in dogs. Comp Cont Ed 1988:10:1365–77.
5. Beale KM. Current diagnostic techniques for evaluating thyroid function in the dog. Vet Clin North Am 1990, in press.
6. Wheeler SL, Husted PW, Rosychuck RAW, *et al.* Serum concentrations of thyroxine and 3,5,3'-triiodothyronine before and after intravenous of intramuscular thyrotropin administration in dogs. Am J Vet Res 1985;46:2605–8.
7. Oliver JW, Waldrop V. Sampling protocol for thyrotropin stimulation test in the dog. J Am Vet Med Assoc 1983;182:486–9.
8. Delitala G, Tomasi P, Virdis R. Prolactin, growth hormone and thyrotropin-thyroid hormone secretion during stress states in man. Baillere's Clin Endocrinol Metab 1987;1:391–414.
9. Conaway DH, Padgett GA, Bunton TE, *et al.* Clinical and histological features of primary, progressive, familial thyroiditis in a colony of borzoi dogs. Vet Pathol 1985;22:439–46.
10. Beale KM, Halliwell REW, Chen CL. Prevalence of antithyroglobulin antibodies detected by enzyme-linked immunosorbent assay of canine serum. J Am Vet Med Assoc 1990;196:745–8.
11. Moriello KA, Halliwell REW, Oakes M. Determination of thyroxine, triiodothyronine, and cortisol changes during simultaneous adrenal and thyroid function tests in healthy dogs. Am J Vet Res 1987;48:458–62.
12. Chastain CB, Franklin RT, Ganjam VK, Madsen RW. Evaluation of the hypothalamic pituitary-adrenal axis in clinically stressed dogs. J Am Anim Hosp Assoc 1986;22:435–42.
13. Peterson ME, Ferguson DC, Kintzer PP, Drucker WD. Effects of spontaneous hyperadrenocorticism on serum thyroid hormone concentrations in the dog. Am J Vet Res 1984;45:2034–8.
14. Weller RE, Kinnas TC, Stevens D. Basal serum thyroxine concentration in euthyroid, hypothyroid and aged dogs. Proc 12th Sci Meet ACVIM 1984;31.
15. Kirk RW, ed. Current veterinary therapy X. Philadelphia: WB Saunders, 1989:i.
16. Chester DK. Endocrine dermatoses. In: Nesbitt G, ed. Dermatology. New York: Churchill Livingstone, 1987:159–87.
17. Haines DM, Lording PM, Penhale WJ. Survey of thyroglobulin autoantibodies in dogs. Am J Vet Res 1984;45:1493–7.
18. Gosselin S, Capen CC, Martin SL. Animal model of a human disease: lymphocytic thyroiditis in the dog. Am J Pathol 1978;19:285–8.
19. Young DW, Sartin JL, Kemppainen RJ. Abnormal canine triiodothyronine-binding factor characterized as a possible triiodothyronine autoantibody. Am J Vet Res 1985;46:1346–50.
20. Rajatanavin R, Fang SL, Pino S, *et al.* Thyroid hormone antibodies and Hashimoto's thyroiditis in mongrel dogs. Endocrinology 1989;124:2535–40.