Prevalence of obese dogs in a population of dogs with cancer

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Objective—To determine the body condition score (BCS) distribution for dogs examined at a teaching hospital and examine whether the BCS distribution for dogs with cancer differed significantly from the distribution for dogs without cancer.

Sample Population—1,777 dogs with cancer and 12,893 dogs without cancer.

Procedures—A retrospective prevalence case-control study was conducted that used medical records from 1999 to 2004. Information was collected on BCS (9-point system), age, breed, sex, neuter status, diagnosis, and corticosteroid administration. Body condition score at the time of examination for cancer (dogs with cancer) or first chronologic visit (dogs without cancer) was recorded. Logistic regression was used to compare BCS prevalence distributions between groups.

Results—The overall prevalence of obese dogs (BCS ≥ 7/9) was 14.8% (2,169/14,670), and the overall prevalence of overweight dogs (BCS ≥ 6/9 to < 7/9) was 21.6% (3,174/14,670). There was a significant difference in the BCS distribution between dogs with and without cancer, with a slightly lower prevalence of being overweight and obese in dogs with cancer. The prevalence of obese and overweight dogs varied with specific cancer types when compared with the prevalence for dogs without cancer.

Conclusions and Clinical Relevance—Differences in obesity prevalence among cancer types is suggestive of an incongruous effect of this variable on cancer expression or a differential effect of specific cancer types on weight status. Systematic use of BCSs will help elucidate the association between obesity and cancer development. (*Am J Vet Res* 2007;68:389–398)

Obesity is a growing area of concern for humans and companion animals. Each year, the medical community and popular press dedicate increasing amounts of time and resources to the health complications of obesity in humans, which is currently considered one of the fastest-growing epidemics throughout the world.[1] Obesity in humans is associated with an increase in risk of fatality for a number of disease states, and obesity-related deaths are expected to outnumber the causes of death for all other preventable conditions in the near future.[2] Diseases associated with obesity in humans include degenerative orthopedic disease, respiratory compromise, heart disease, hypertension, diabetes mellitus, and intervertebral disk disease.[2,3] Obesity influences the development of certain cancers, such as colorectal carcinoma,[4] postmenopausal breast adenocarcinoma,[5] esophageal-gastric adenocarcinoma,[6,7] and hepatocellular carcinoma,[8] but it does not increase the incidence of premenopausal breast cancer,[5,9] and its role in the development of prostate cancer is unclear.[10,11] The relationship between obesity and specific types of cancer in humans suggests the potential for a similar association in dogs.

Abbreviations

IGF	Insulin-like growth factor
BCS	Body condition score
TCC	Transitional cell carcinoma
VMTH	Veterinary medical teaching hospital
POR	Prevalence odds ratio

A number of mechanisms have been proposed for the role of obesity in carcinogenesis. Chronic increases in insulin concentrations, which are evident in obese humans, suppress the formation of IGF binding proteins.[12] Decreased concentrations of IGF binding proteins allow for greater circulating concentrations of free IGF. In turn, free IGF acts as a cell-growth promoter that suppresses apoptosis of normal and aberrant cells, which leads to a proposed increase in survival of tumor cells.[12] Leptin, a hormone produced by adipocytes and therefore found in greater quantities in obese patients, is a promoter of human mammary cancer cells in vitro[13] and is implicated in the development of certain hepatocellular carcinomas.[8]

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Obese dogs also have increased circulating concentrations of IGF and leptin,[14-16] although an association between these hormones and the development of cancer in dogs is not clear.

Another potential mechanism is the relationship between mutations in tumor suppressor genes (such as p53) and obesity. Mutations in p53 are found in approximately half of all cancers in humans[17] and are also found in a number of cancers in dogs, including hemangiosarcomas,[18] osteosarcomas,[19] and mammary gland tumors.[20] In p53-knockout transgenic mice, excess caloric intake and subsequent obesity accelerate tumor development and decrease lifespan, relative to results in mice with a restricted caloric intake remain lean.[21-23]

The most common measurement of obesity used in veterinary medicine is the BCS. Body condition scoring evaluates adiposity at designated body sites and scores an animal in relation to a lean musculoskeletal system.[a] There are 2 BCS scales commonly in use. One uses a 9-point scale, with 4/9 to 5/9 considered an ideal BCS. The other uses a 5-point scale, with an ideal BCS of 3/5. Both scales have similar shortcomings, such as potential differences in assignment of BCS values as a result of variation among veterinarians in scoring[24] and variations in breed conformation, but both are noninvasive, inexpensive, readily learned, and easy for owners to understand. Additionally, the ability of the 9-point scale to predict fat body mass has been validated[25] relative to more expensive laboratory measurements, such as dual-energy x-ray absorptiometry, and when used correctly, each point above 5 on the 9-point scale represents an increase of 10% to 15% over ideal body weight.[a] Obesity in dogs has been defined as a body weight > 20% over ideal[25]; therefore, an animal with a BCS ≥ 7/9 would be considered obese.

Excess body fat mass in dogs has a substantial impact on the development of degenerative orthopedic disease[26] and resistance to insulin.[27] In 1 study,[26] investigators reported an increase of 15% in mean life span for restricted-fed dogs (mean ± SEM BCS, 4.6 ± 0.19 [9-point scale]), compared with life span for control dogs (mean BCS, 6.7 ± 0.19 [9-point scale]). In a study[28] on the association between obesity and various diseases of dogs, investigators found an increased prevalence of benign and malignant neoplasms among dogs considered obese. The importance of factors such as obesity on the development of histologically and behaviorally malignant cancers remains controversial despite the prevalence of cancer among the general canine population. With approximately 40% to 50% of dogs older than 10 years of age developing 1 or more types of tumors,[29] finding a relationship between obesity and cancer in dogs could serve as a provocative impetus for the avoidance of obesity in dogs. Evaluations of this relationship have focused on specific malignant neoplasms (ie, TCC of the bladder and mammary gland carcinoma)[30-33] or have included a number of benign or ill-defined types of cancer.[28] To the authors' knowledge, there have not been any published reports in which investigators evaluated obesity prevalence in a large population of dogs with various histologically and behaviorally malignant types of cancer. The purpose of the study reported here was to determine the distribution of BCS values for dogs examined at a large veterinary hospital and to evaluate whether the distribution of BCS values for dogs with histologically and behaviorally malignant neoplasms (cancer case dogs) differed significantly from BCS values for dogs without cancer (noncancer control dogs).

Materials and Methods

Sample population—Medical records for dogs examined at the VMTH at the University of California, Davis, from January 1999 to December 2004 were reviewed. Dogs were included when the BCS (on a 9-point scale), the complete signalment (age, breed, sex, and neuter status), and a definitive diagnosis were available as listed in the veterinary medical and administrative computer system. Diagnosis of cancer was based on cytologic or histologic examination of antemortem specimens or specimens obtained during postmortem examination. Exclusion criteria for cancer case dogs and noncancer control dogs included histologically and behaviorally benign tumors (including lipoma), a history of benign or malignant neoplasms, an undefined type of cancer, multiple types of cancer, or a corticosteroid-secreting tumor.

Procedures—The database was searched for dogs with a reported BCS. The BCS values had been assigned by use of a 9-point scale (1 = extremely thin and 9 = extremely obese) and verified by the managing clinician at the time a dog was examined at the VMTH. For dogs in which BCS was recorded, the visit identification number, patient signalment, physical examination findings, body weight, and clinical diagnosis were downloaded into a database management program.[b] A separate search of the database was conducted to identify dogs in the original group that had a history of steroid administration.

Numeric values were extracted from the physical examination field of the BCS data list by the use of a computer program.[b] The BCS at the time of initial examination (dogs with cancer) or at the first chronologic visit (noncancer control dogs) was recorded. Records of each visit were manually sorted on the basis of clinical diagnosis. Dogs were assigned to 1 of 5 age categories to assess the effect of age on BCS distribution (< 1 year old, 1 to < 5 years old, 5 to < 10 years old, 10 to < 15 years old, and ≥ 15 years old).

Dogs with recorded breed designations were assigned a numeric value of 1 to 10 on the basis of published genetic breed classifications described elsewhere.[34] Dogs from breeds without published genetic relatedness were placed in a single inclusive group (ie, unclassified breed). Mixed-breed dogs were assigned to a separate group irrespective of breed and were used as the reference group for all breed analyses.

Dogs with cancer were further allocated into 1 of 3 general cancer categories on the basis of histologic classification (sarcoma, carcinoma, or round cell tumor). Specific cytologic and histologic diagnoses of cancer were retained for further evaluation. Dogs with a reported BCS but without benign neoplasms or a history of benign or malignant tumors were designated as noncancer control dogs.

Prevalences of BCS distributions for the overall population (including dogs with a history of corticosteroid administration) on the basis of age, neuter status, and breed classification were generated by use of a database management program.[b] Analysis by use of a Kruskal-Wallis ANOVA was conducted to compare BCS distributions among genetic breed classifications and neuter statuses. Logistic regression analyses were performed by use of statistical software.[c] The regression analyses included age (4 functional polynomials), genetic breed classification, sex, and neuter status in all models.

The distribution of BCS values between dogs with cancer and noncancer control dogs and between dogs in specific cancer categories and noncancer control dogs was evaluated. Paired analyses were performed that included and excluded dogs with a history of steroid administration. The distributions of BCS values for each of the 2 most prevalent sarcomas (hemangiosarcoma and osteosarcoma), carcinomas (squamous cell carcinoma and TCC), and round cell tumors (mast cell tumor and lymphoma) were also compared with values for the noncancer control dogs. Additionally, dogs with mammary gland carcinomas were selected for additional evaluation because of published[31-33] data on obesity prevalence for this type of cancer. Lymphoma was further subcategorized into B-cell, T-cell, and undifferentiated lymphomas, and the BCS distributions of these subcategories were compared with that for the noncancer control dogs. Undifferentiated lymphomas were lymphomas whose identity could not be determined with standard immunophenotyping monoclonal antibodies used at the VMTH.

Results were reported as the POR and 95% confidence interval. For all statistical analyses, values of $P \leq 0.05$ were considered significant.

A list of referral veterinarians for dogs with B-cell lymphoma (n = 118) and a randomly generated group of noncancer control dogs (165) was created by use of a database management program.[b] Written surveys were mailed to the referral veterinarians in an attempt to collect information on body weight of each dog 6 to 12 months before it was examined at the VMTH. Veterinarians who did not respond to the mail survey were subsequently contacted by telephone in an effort to collect the requested information. Regression analysis was used to compare historical weight with weight at the time of initial examination at the VMTH. Correlation coefficient of historical weight and initial weight was reported.

Results

Sample population—From January 1999 to December 2004, 35,967 dogs were examined at the VMTH. Medical data for 29,196 dogs with a recorded BCS during this time period were retrieved from the database. Eighty-two of the 85 breeds described elsewhere,[34] plus another 76 breeds of unknown genetic relatedness, were examined at the VMTH (**Table 1**). The 3 most common breeds were German Shepherd Dog (n = 657; breed group 2), Golden Retriever (808; breed group 4), and Labrador Retriever (1,597; breed group 10).

Of the dogs whose records were reviewed, 14,670 (1,777 dogs with cancer and 12,893 noncancer control dogs) met the inclusion criteria and were further evaluated. A total of 3,203 (21.8%; 600 dogs with cancer and 2,603 noncancer control dogs) had a history of corticosteroid administration. These dogs were included in the evaluation of the overall demographics of the population (**Table 2**).

The population of 14,670 dogs comprised 1,694 (11.5%) dogs < 1 year old, 4,735 (32.3%) dogs 1 to < 5 years old, 5,273 (35.7%) dogs 5 to < 10 years old, 2,808 (19.1%) dogs 10 to < 15 years old, and 196 (1.3%) dogs ≥ 15 years old. Mean age of all dogs with cancer was 9.12 years (range, 0.53 to 18.5 years), whereas mean age of noncancer control dogs was 5.72 years (range, 0.12 to 16.7 years). The population contained 2,007 (13.7%) sexually intact males, 5,092 (34.7%) neutered males, 1,562 (10.6%) sexually intact females, and 6,009 (41.0%) spayed females.

Dogs with cancer included 582 dogs with sarcomas (including 72 dogs with a history of corticosteroid administration), 428 dogs with carcinomas (including 87 dogs with a history of corticosteroid administration), and 767 dogs with round cell tumors (including 441 dogs with a history of corticosteroid administration). Sarcomas included but were not limited to appendicular osteosarcoma (n = 159 dogs), hemangiosarcoma (112), hemangiopericytoma (60), fibrosarcoma (44), spindle cell sarcoma (35), neurofibrosarcoma (32), chondrosarcoma (21), myxosarcoma (11), liposarcoma (4), leiomyosarcoma (3), anaplastic sarcoma (2), mesenchymal cell sarcoma (2), angiosarcoma (2), myxofibrosarcoma (2), schwannoma (2), lymphangiosarcoma (1), myofibroblastic sarcoma (1), and synovial cell sarcoma (1). Carcinomas included but were not limited to squamous cell carcinoma (n = 74 dogs); TCC (69); anal gland adenocarcinoma (67); thyroid gland adenocarcinoma (39); mammary gland carcinoma (27); mammary gland adenocarcinoma (22); nasal carcinoma (20); nasal adenocarcinoma (12); pulmonary carcinoma (9); hepatocellular carcinoma (9); pulmonary adenocarcinoma (8); basal cell carcinoma (5); colonic adenocarcinoma (4); biliary carcinoma (3); cardiac carcinoma (3); intestinal carcinoma (3); prostatic carcinoma (3); rectal carcinoma (3); renal carcinoma (3); salivary gland carcinoma (3); aural carcinoma (2); and 1 each of bronchogenic carcinoma, gastric carcinoma, papillary carcinoma, thyroid gland carcinoma, tonsillar carcinoma, vaginal carcinoma, maxillary adenocarcinoma, and papillary adenocarcinoma. Round cell tumors included mast cell tumor (n = 391 dogs), B-cell lymphoma (118), undifferentiated lymphoma (99), T-cell lymphoma (74), histiocytic sarcoma (43), leukemia (21), multiple myeloma (10), plasmacytoma (10), and anaplastic round cell tumor (1).

Prevalence of obese dogs—Prevalence of obese dogs (BCS ≥ 7/9) in the total population was 14.8% (2,169/14,670), and the overall prevalence of overweight dogs (BCS ≥ 6/9 to < 7/9) was 21.6% (3,174/14,670), irrespective of age groups (**Figure 1**). Age had a significant influence on BCS distribution ($P < 0.001$). When dogs < 1 year old were excluded from the sample population, the overall prevalence of obese dogs was 16.5% (2,136 /12,976), and the prevalence of overweight dogs was 23.5% (3,045/12,976). Prevalence of obese dogs for each age group increased from 1.9% (33/1,694) for dogs < 1 year old to 20.2% (1,060/5,237) for dogs 5 to < 10

Table 1—Genetic breed classification and number of dogs of each breed in a sample population of 14,670 dogs examined at a VMTH between January 1999 and December 2004.*

Group	Breeds
1 (393)	Akita (86), Siberian Husky (81), Chinese Shar-Pei (74), Chow Chow (52), Alaskan Malamute (51), Shiba Inu (27), and Basenji (22)
2 (1,991)	German Shepherd Dog (657), Rottweiler (476), Boxer (343), Newfoundland (177), Bulldog (137), Mastiff (92), Bullmastiff (54), Miniature Bull Terrier (39), and French Bulldog (16)
3 (303)	Shetland Sheepdog (164), Greyhound (43), Collie (42), Irish Wolfhound (20), Borzoi (15), Belgian Tervuren (10), and Belgian Sheepdog (9)
4 (2,542)	Golden Retriever (808), American Cocker Spaniel (346), Doberman Pinscher (182), Miniature Schnauzer (172), Border Collie (171), West Highland White Terrier (108), Beagle (110), German Shorthaired Pointer (95), Cavalier King Charles Spaniel (92), Basset Hound (68), Chesapeake Bay Retriever (57), Airedale Terrier (47), Standard Schnauzer (44), Cairn Terrier (42), English Cocker Spaniel (29), Irish Setter (27), Old English Sheepdog (26), Portuguese Water Dog (25), Schipperke (22), Pointer (18), Italian Greyhound (17), Giant Schnauzer (11), Australian Terrier (10), Bloodhound (7), Welsh Springer Spaniel (5), American Water Spaniel (1), Ibizan Hound (1), and Pharaoh Hound (1)
5 (349)	Shih Tzu (178), Lhasa Apso (90), Pekingese (42), and Samoyed (39)
6 (91)	Tibetan Terrier (50), Saluki (25), and Afghan Hound (16)
7 (839)	Poodle (395), Pug (158), Great Dane (117), Bichon Frise (86), Whippet (29), Keeshond (27), Norwegian Elkhound (17), Manchester Terrier (8), and Kuvasz (2)
8 (483)	Dachshund (338) and Pomeranian (145)
9 (160)	Bernese Mountain Dog (113), St Bernard (39), and Greater Swiss Mountain Dog (8)
10 (2,178)	Labrador Retriever (1,597), Australian Shepherd (215), Chihuahua (202), Rhodesian Ridgeback (82), Soft-Coated Wheaton Terrier (35), Flat-Coated Retriever (22), Kerry Blue Terrier (10), Bedlington Terrier (7), Irish Terrier (6), and Clumber Spaniel (2)
Unclassified (2,076)†	Yorkshire Terrier (240), Staffordshire Terrier (212), Australian Cattle Dog (140), Jack Russell Terrier (133), Maltese (118), Dalmatian (114), English Springer Spaniel (113), Pembroke Welsh Corgi (82), Boston Terrier (75), Fox Terrier (62), Weimaraner (57), Vizsla (56), Brittany (54), Miniature Pinscher (53), Scottish Terrier (50), American Eskimo (45), Great Pyrenees (39), Bouvier des Flandres (35), Belgian Malinois (30), Papillon (25), Bearded Collie (20), Silky Terrier (20), Border Terrier (18), English Pointer (18), Rat Terrier (17), Gordon Setter (15), and Welsh Terrier (15)

Number of dogs is indicated in parentheses. Values reported include dogs with a history of corticosteroid administration.
*The breed profiles of the 3,265 mixed-breed dogs have not been included in this table. †Includes 49 other breeds with ≤ 12 dogs/breed.

years old (Table 2). There was a subsequent decrease in prevalence of obese dogs (14.3% [28/196]) for dogs ≥ 15 years old. In comparison, the prevalence of dogs with BCS values of 4/9 to < 6/9 (ideal body weight) was highest in dogs < 1 year old (84.5% [1,431/1,694]), was decreased in dogs 1 to < 5 years old (64.6% [3,061/4,735]), and then remained stable for dogs 5 to < 10 years old (49.5% [2,594/5,237]) and dogs ≥ 15 years old (49.5% [97/196]). Prevalence of dogs with BCS 3/9 to < 4/9 (underweight) increased slightly from 4.8% (81/1,694) in dogs < 1 year old to 5.7% (159/2,808) in dogs 10 to < 15 years old. There was a subsequent increase in the prevalence of underweight dogs in dogs ≥ 15 years old (10.2% [20 of 196]). The prevalence of dogs with BCS 1/9 to < 3/9 (extremely thin) remained relatively stable for dogs < 1 year old (1.2% [20/1,694]) to dogs 10 to < 15 years old (1.5% [43/2,808]), and it then increased to 6.1% (12/196) for dogs ≥ 15 years old.

Neutered dogs were significantly ($P < 0.001$) more likely to be obese or overweight, compared with results for sexually intact dogs (Table 2). The prevalence of obese dogs increased from 7.2% (257/3,569) in sexually intact dogs to 17.2% (1,912/11,101) in neutered dogs, and the prevalence of overweight dogs increased from 14.2% (511/3,569) in sexually intact dogs to 24% (2,663/11,101) in neutered dogs.

Distribution of BCS values differed significantly ($P < 0.001$) on the basis of genetic breed classification (Table 2). Breed group 10 had the highest prevalence of obese (17.7% [378/2,176]) and overweight (25.1% [547/2,176]) dogs, whereas breed group 6 had the lowest prevalence of obese (9.9% [9/91]) and overweight (8.8% [8/91]) dogs.

All dogs with cancer—Mean ± SD BCS for dogs with cancer was 5.4 ± 1.2 (9-point scale), and mean BCS for noncancer control dogs was 5.3 ± 1.2. The BCS distribution differed significantly ($P = 0.001$) between all dogs with cancer and the noncancer control dogs. There was a significantly ($P < 0.001$) higher prevalence of corticosteroid administration for dogs with cancer (POR, 1.4), compared with that for the noncancer control dogs. There was a significantly lower prevalence of overweight (POR, 0.81; $P = 0.001$) and obese (POR, 0.8; $P = 0.002$) dogs for all dogs with cancer, compared with values for noncancer control dogs (**Table 3**). When dogs with a history of corticosteroid administration were excluded from the analysis, there was a significantly lower prevalence of overweight dogs (POR, 0.8; $P = 0.007$) and a lower prevalence of obese dogs which was not significantly different (POR, 0.85; $P = 0.07$), for dogs with cancer, compared with results for noncancer control dogs (**Table 4**).

For all dogs with cancer, there were significant confounding effects detected for age ($P < 0.001$), genetic breed classification ($P < 0.001$), and neuter status ($P = 0.01$). There was also a significantly ($P < 0.001$)

lower prevalence of breed groups 5 (POR, 0.38), 7 (POR, 0.56), and 8 (POR, 0.21), whereas there was a significantly higher prevalence of breed groups 2 (POR, 1.3; $P = 0.002$) and 9 (POR, 3.3; $P < 0.001$), relative to mixed-breed dogs. There was a significantly higher prevalence of neutered males (POR, 1.3; $P = 0.009$) and neutered females (POR, 1.3; $P = 0.01$), relative to sexually intact males for all dogs with cancer. These results were not different when dogs with a history of corticosteroid administration were excluded.

Sarcomas—A significantly lower prevalence of overweight (POR, 0.74; $P = 0.007$) and extremely thin (POR, 0.1; $P = 0.02$) dogs and a lower prevalence of obese dogs that was not significantly (POR, 0.81; $P = 0.07$) different were detected for all dogs with sarcomas (Table 3). When dogs with a history of corticosteroid administration were excluded from the analysis, there was still a significantly (POR, 0.78; $P = 0.03$) lower prevalence of overweight dogs, but the lower prevalence of extremely thin (POR, 0.15; $P = 0.07$) and obese (POR, 0.87; $P = 0.27$) dogs was not significantly different from those for noncancer control dogs (Table 4).

A significant ($P < 0.001$) confounding effect of age and genetic breed classification was detected when comparing the BCS distribution of dogs with sarcomas and noncancer control dogs for both models, whereas neuter status did not have a significant ($P = 0.1$) effect. Relative to mixed-breed dogs, there was a significantly lower prevalence of breed group 5 (POR, 0.18; $P < 0.001$), 7 (POR, 0.48; $P = 0.002$), and 8 (POR, 0.07; $P < 0.001$) for all dogs with sarcomas, whereas there was a significantly ($P < 0.001$) higher prevalence of breed groups 2 (POR, 1.6) and 9 (POR, 3.7). These results were not different when dogs with a history of steroid administration were excluded.

Carcinomas—A significantly lower prevalence of overweight (POR, 0.65; $P = 0.001$) and obese (POR, 0.74; $P = 0.03$) dogs was detected for all dogs with carcinomas (Table 3). When dogs with a history of steroid administration were excluded from the analysis, there was a significantly lower prevalence of overweight dogs (POR, 0.59; $P < 0.001$) and a lower prevalence of obese dogs that was not significantly (POR, 0.76; $P = 0.08$) different for dogs with carcinomas, relative to results for noncancer control dogs (Table 4).

Analysis revealed a significant ($P < 0.001$) confounding effect of age when comparing the BCS distribution of all dogs with carcinomas with that for noncancer control dogs. There was not a significant effect of genetic breed classification ($P = 0.7$) or neuter status

Table 2—The BCS distribution for a population of dogs examined at a VMTH on the basis of demographic characteristics.

		BCS* (No. of dogs)				
Characteristic	**No. (%)**	**1 to < 3**	**3 to < 4**	**4 to < 6**	**6 to < 7**	**≥ 7**
Age (y)						
< 1	1,694 (11.5)	20	81	1,431	129	33
1 to < 5	4,735 (32.3)	35	174	3,061	953	512
5 to < 10	5,237 (35.7)	50	180	2,594	1,353	1,060
10 to < 15	2,808 (19.1)	43	159	1,370	700	536
≥ 15	196 (1.3)	12	20	97	39	28
Total	**14,670 (100)**	**160**	**614**	**8,553**	**3,174**	**2,169**
Sex						
Male						
Sexually intact	2,007 (13.7)	37	125	1,404	303	138
Neutered	5,092 (34.7)	40	186	2,770	1,220	876
Female						
Sexually intact	1,562 (10.6)	24	80	1,131	208	119
Neutered	6,009 (41.0)	59	223	3,248	1,443	1,036
Both						
Sexually intact	3,569 (24.3)	61	205	2,535	511	257
Neutered	11,101 (75.7)	99	409	6,018	2,663	1,912
Breed classification group						
1	393 (2.7)	8	22	244	83	36
2	1,991 (13.6)	30	123	1,213	358	267
3	303 (2.1)	4	19	173	58	49
4	2,542 (17.3)	17	84	1,399	637	405
5	349 (2.4)	5	9	216	70	49
6	91 (0.6)	7	6	61	8	9
7	839 (5.7)	10	53	535	132	109
8	483 (3.3)	1	18	276	108	80
9	160 (1.1)	4	5	86	40	25
10	2,176 (14.8)	15	55	1,183	547	378
Unclassified	2,076 (14.2)	25	94	1,304	395	258
Mixed breed	3,265 (22.3)	34	126	1,863	738	504

Values reported include dogs with a history of steroid administration.
*Determined by use of a 9-point BCS scale (1 = extremely thin and 9 = extremely obese).

(P = 0.2) on overall BCS distribution. However, with regard to evaluation of specific characteristics, there was a significantly (P = 0.03) lower prevalence of breed group 8 relative to mixed-breed dogs (POR, 0.45), a significantly (P = 0.03) higher prevalence of neutered females (POR 1.5) relative to sexually intact males with carcinomas, and a higher prevalence of neutered males that was not significantly (POR, 1.5; P = 0.06) different. When dogs with a history of corticosteroid administration were excluded from the analysis, there was no significant difference in the prevalence of breed group 8 relative to mixed-breed dogs, whereas significance was retained for the confounding effects of age ($P < 0.001$) and the higher prevalence of neutered females (POR, 1.7; P = 0.01) relative to sexually intact males. The higher prevalence of neutered males (POR, 1.5) was not significantly (P = 0.07) different.

Round cell tumors—When all dogs with round cell tumors were evaluated, there was a significantly higher prevalence of underweight dogs (POR, 1.5; P = 0.02) and a lower prevalence of obese dogs that did not differ significantly (POR, 0.82; P = 0.06) from noncancer control dogs (Table 3). When dogs with a history of corticosteroid administration were excluded, there was no significant difference in BCS distribution between dogs with round cell tumors and noncancer control dogs (Table 4).

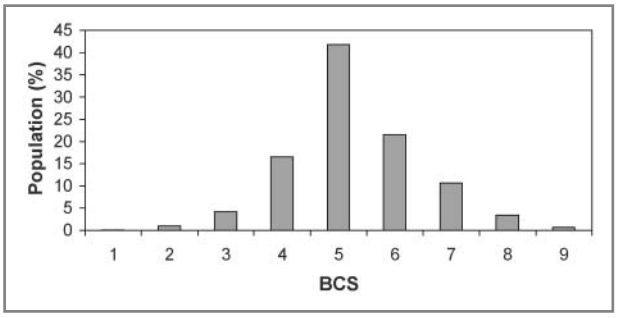

Figure 1—The BCS distribution for 14,670 dogs examined at a VMTH between January 1999 and December 2004. Values reported include those for dogs with a history of corticosteroid administration.

A significant confounding effect was detected for age ($P < 0.001$), genetic breed classification ($P < 0.001$), and neuter status (P = 0.002) when comparing the BCS distribution of all dogs with round cell tumors and noncancer control dogs. There was a significantly lower prevalence of breed groups 5 (POR, 0.35; P = 0.004), 7 (POR, 0.45; $P < 0.001$), and 8 (POR, 0.2; $P < 0.001$) in all dogs with round cell tumors, whereas there was a significantly higher prevalence of breed groups 4 (POR, 1.3; P = 0.04), 9 (POR, 3.8; $P < 0.001$), and 10 (POR, 1.3; P = 0.04). There was also a significantly (P = 0.02) higher prevalence of neutered females (POR, 1.4) relative to sexually intact males and a higher prevalence of neutered males that was not significantly (POR, 1.3; P = 0.07) different. When dogs with a history of corticosteroid administration were excluded from the analysis, significance was retained for the confounding effects of age ($P < 0.001$), the higher prevalence of neutered females (POR, 2.1; P = 0.002) relative to sexually intact males, the lower prevalence of breed groups 5 (POR, 0.24; P = 0.05) and 8 (POR, 0.17; P = 0.02), and the higher prevalence of breed groups 9 (POR, 5.6; $P < 0.001$) and 10 (POR, 1.5; P = 0.04). The higher prevalence of neutered males (POR, 2.1) relative to sexually intact males was significantly (P = 0.003) different.

Other specific types of cancer—Compared with results for noncancer control dogs, dogs with hemangiosarcoma had a significantly lower prevalence of overweight (POR, 0.53; P = 0.02) and obese (POR, 0.53; P = 0.04) dogs. When dogs with a history of steroid administration were excluded from the analysis, these results remained unchanged. There was no significant difference in BCS distribution in dogs

Table 3—Values for POR and 95% confidence interval for all dogs with cancer and dogs with sarcomas, carcinomas, or round cell tumors in a sample population examined at a VMTH.

	All dogs with cancer (n = 1,777)			Sarcomas (n = 582)			Carcinomas (n = 428)			Round cell tumors (n = 767)		
BCS*	POR	95% CI	*P* value†	POR	95% CI	*P* value†	POR	95% CI	*P* value†	POR	95% CI	*P* value†
1 to < 3	0.64	0.38–1.10	0.100	0.10	0.01–0.72	0.020	0.49	0.17–1.40	0.170	1.30	0.70–2.50	0.380
3 to < 4	1.20	0.93–1.50	0.160	0.93	0.61–1.40	0.730	1.10	0.71–1.70	0.680	1.50	1.10–2.10	0.020
4 to < 6	1.00	NA	NA	1.00	NA	NA	1.00	NA	NA	1.00	NA	NA
6 to < 7	0.81	0.71–0.92	0.001	0.75	0.6–0.92	0.007	0.65	0.50–0.84	0.001	0.94	0.78–1.10	0.490
≥ 7	0.80	0.69–0.92	0.002	0.81	0.64–1.00	0.070	0.74	0.56–0.97	0.030	0.82	0.66–1.00	0.060

†Represents comparison with values for BCS 4 to < 6; results were considered significant at $P \leq 0.05$.
CI = Confidence interval. NA = Not applicable.
See Table 2 for remainder of key.

Table 4—Values for POR and 95% confidence interval for all dogs with cancer and dogs with sarcomas, carcinomas, or round cell tumors in a sample population examined at a VMTH, excluding dogs with a history of corticosteroid administration.

	Dogs with cancer (n = 1,177)			Sarcomas (n = 510)			Carcinomas (n = 341)			Round cell tumors (n = 326)		
BCS*	POR	95% CI	*P* value†	POR	95% CI	*P* value†	POR	95% CI	*P* value†	POR	95% CI	*P* value†
1 to < 3	0.63	0.29–1.30	0.230	0.15	0.02–1.10	0.070	0.56	0.17–1.90	0.350	1.80	0.64–5.30	0.260
3 to < 4	0.94	0.68–1.30	0.710	0.93	0.58–1.50	0.750	0.95	0.57–1.60	0.840	0.89	0.46–1.70	0.720
4 to < 6	1.00	NA	NA	1.00	NA	NA	1.00	NA	NA	1.00	NA	NA
6 to < 7	0.80	0.69–0.94	0.007	0.78	0.62–0.98	0.030	0.59	0.44–0.80	< 0.001	1.10	0.82–1.40	0.640
≥ 7	0.85	0.71–1.00	0.070	0.87	0.68–1.1	0.270	0.76	0.56–1.00	0.080	0.91	0.67–1.20	0.550

See Tables 2 and 3 for key.

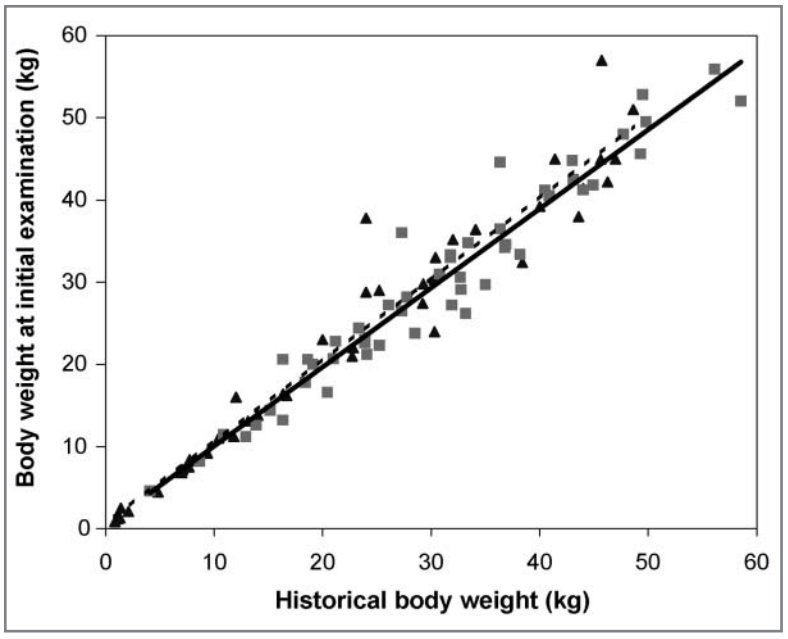

Figure 2—Comparison of historical body weight 6 to 12 months before initial examination at a VMTH and body weight at time of initial examination at the VMTH for 57 dogs with B-cell lymphoma and 50 noncancer control dogs. The solid line indicates the line of best fit for the dogs with B-cell lymphoma (squares), whereas the line of best fit for the noncancer control dogs (triangles) is indicated by the dashed line. Values were significantly ($P < 0.001$) correlated ($r^2 = 0.97$).

with osteosarcoma, squamous cell carcinoma, or TCC, relative to noncancer control dogs, regardless of whether dogs with a history of corticosteroid administration were included or excluded. Dogs with mammary gland adenocarcinoma or carcinoma had a higher but not significantly different prevalence of overweight (POR, 1.6; $P = 0.21$) and obese (POR, 1.7; $P = 0.15$) dogs. These findings changed slightly when dogs with a history of corticosteroid administration were excluded (POR, 1.6; $P = 0.17$ and POR, 1.9; $P = 0.1$ for overweight and obese dogs, respectively).

Dogs with mast cell tumors had a significantly ($P = 0.02$) higher prevalence of overweight dogs (POR, 1.3) and a significantly ($P = 0.03$) lower prevalence of underweight dogs (POR, 0.41), relative to noncancer control dogs. These findings changed slightly when dogs with a history of corticosteroid administration were excluded (POR, 1.3; $P = 0.3$ and POR, 0.4; $P = 0.03$ for overweight and underweight dogs, respectively).

Dogs with lymphoma had a significantly ($P < 0.001$) higher prevalence of underweight dogs (POR, 2.2) and a higher prevalence of extremely thin dogs (POR, 1.7) that was not significantly ($P = 0.17$) different, relative to noncancer control dogs. When dogs with a history of corticosteroid administration were excluded from the analysis, there was a significantly ($P < 0.001$) higher prevalence of underweight dogs (POR, 2.1) and a significantly ($P = 0.05$) lower prevalence of obese dogs (POR, 0.69); the higher prevalence of extremely thin dogs (POR, 1.2) was not significantly ($P = 0.59$) different. We did not detect significant differences in the BCS distribution of dogs with B-cell or T-cell lymphoma, relative to noncancer control dogs, irrespective of whether dogs with a history of corticosteroid administration were included or excluded. Dogs with undifferentiated lymphoma had a significantly ($P < 0.001$) higher prevalence of underweight dogs (POR, 3.5) and a higher but not significantly ($P = 0.09$) different prevalence of extremely thin dogs (POR, 2.5), compared with the BCS results for noncancer control dogs. When dogs with a history of corticosteroid administration were excluded from the analysis, there was still a significantly ($P < 0.001$) higher prevalence of underweight dogs (POR, 3.4), whereas there was a higher but not significantly ($P = 0.22$) different prevalence of extremely thin dogs (POR, 2.0).

Historical data on body weight—Historical body weights were obtained for 57 dogs with B-cell lymphoma and 50 noncancer control dogs. There was no significant difference (r^2, 0.97; $P < 0.001$) between body weight measured by referral veterinarians 6 to 12 months before initial examination at the VMTH and body weight at the time of initial examination of these dogs at the VMTH (**Figure 2**).

Discussion

A paucity of studies exists in the veterinary literature regarding evaluation of the prevalence of obesity of dogs in the United States by use of a consistent measurement and definition of obesity status.[28,35] In the study reported here, we defined obesity as a BCS ≥ 7/9 and found a prevalence of obese dogs of 16.5% for dogs > 1 year old. Investigators in another study[35] defined obesity as a BCS ≥ 4/5 and found that 0.9% of the dogs > 1 year old examined at a veterinary referral hospital in 1986 were obese, whereas other investigators in a 1995 survey of private practitioners[28] defined obesity as a mean BCS ≥ 4.5/5 and found that 5.1% of dogs > 1 year old were obese.

Compared with published obesity statistics for the United States, there appears to be a higher prevalence of obesity in the canine population at the VMTH in our report. Interestingly, when overweight and obese dogs > 1 year old were grouped together in our study, the sum of overweight and obese dogs was higher than that listed in the most recent US report[28] (40% vs 34.3%, respectively) but was similar to the prevalence of obese and overweight dogs reported in Australia[36] and France.[37] The higher prevalence of obese dogs in the study reported here may indicate a true increase in the prevalence of obese dogs in the United States over time that has mirrored the increase in obese humans.[1] However, it may also reflect an increased awareness of the health risks of obesity in dogs or geographic variation in obesity status. Alternatively, evaluators in the 2 studies[28,35] conducted in the United States assigned values as whole integers (ie, 1 to 5) to the dogs in their studies. The limited selection in score assignments created by eliminating half points within the 5-point scale used in those 2 studies may have resulted in deferential score assignments for dogs that were between 2 points and artificially decreased the proportion of dogs considered

obese. Because of the large number of potential evaluators at the VMTH, between-evaluator variability in score assignment could not be accounted for in the statistical model and may have altered the BCS distribution in this population. Because of limited databases on population statistics in veterinary medicine, potential geographic differences in pet characteristics, lack of standardization in recording BCS in each medical record, and lack of standard definitions of obesity, a valid comparison of patterns in obesity of dogs over time was not possible.

Not surprisingly, age, neuter status, and breed were confounders that significantly affected BCS and many cancer analyses. Gonadectomy alone has a direct effect on body fat composition and weight gain in animals,[38,39] and it is possible that increases in body weight after neutering may be compounded by increases in age or a breed propensity for obesity. The differences in BCS distribution for age and neuter status in the study reported here are consistent with reports in the United States,[28,35] United Kingdom,[40,41] Australia,[36] and France[37] and warrant early and more aggressive counseling of owners to prevent obesity in pet dogs.

Epidemiologic studies in dogs in which investigators have evaluated associations of breed and obesity have used specific breed designations[28,37,41] or breed groupings designated by kennel associations.[36] Genetic breed classification was selected for the study reported here to account for the numerous dog breeds represented at the VMTH that may have confounded BCS distributions and cancer prevalence. This breed classification scheme is a relatively new addition to veterinary epidemiological evaluations, with only 1 other report[42] in which investigators used genetic relatedness to evaluate the influence of breed on cancer incidence. Breed variability with regard to prevalence of cancer in dogs has been reported for osteosarcoma,[43] nasal carcinoma,[44,45] and lung cancer[46] but not for renal carcinoma.[47] Investigators in those reports on the relationship between breed and cancer development have used nontraditional breed classifications, such as skull shape[43-46] and body size.[47]

It is not possible to make comparisons of breed distribution among these types of cancer given the limited number of dogs with osteosarcoma, nasal carcinoma, or lung cancer in the study reported here. It should also be mentioned that the prevalence of cancer in our study may not have reflected the prevalence of cancer in the general canine population and was likely a factor of an active referral hospital. Additionally, the exclusion criteria used in this study, along with geographic location, population variation, potential changes in cancer prevalence over time, and improvements in the standard for quality of care in veterinary medicine during the past 40 years, make it difficult to make comparisons between cancer prevalence in dogs examined at this referral VMTH and other reports[28,48,49] of cancer prevalence in dogs.

A significantly lower prevalence of obese dogs was found in dogs with cancer in the study reported here, which is in contrast to expectations based on the association between obesity and a number of cancers in humans; this may have been attributable, in part, to differences in types of cancer in dogs and humans. This finding was also surprising in light of a report[28] that revealed an increased prevalence of neoplasms in obese dogs. The difference in results of that study,[28] compared with the findings for our study, may be attributed to differences in definitions of neoplasia and obesity. In that other study, investigators included a number of histologically and behaviorally benign and ill-defined types of cancer, which were excluded from our study. Also, those investigators assigned whole numbers for BCS values by use of a 5-point scale and then determined a mean score for the same dog. These differences, as well as a narrower definition of obesity than that used in our study, make it difficult to generate direct comparisons between results of that report[28] and the study reported here.

In our study, we detected a lower prevalence of overweight and obese dogs with sarcomas and carcinomas and no difference in the distribution of BCS in dogs with round cell tumors. These findings may indicate a protective effect of increased body fat mass on certain types of cancer in dogs, similar to the relationship between obesity and premenopausal breast cancer in humans.[5,9] Without accounting for the limited sample size of many veterinary epidemiologic studies and the influence of potential confounders, such as age, neuter status, or breed, the role of obesity in specific cancers in dogs may be overstated. Compared with epidemiologic studies[2,3-9] conducted on specific cancers in humans, which contain tens of thousands of people, the study reported here included 47 dogs with mammary gland cancers, 9 dogs with hepatocellular carcinoma, 4 dogs with colonic adenocarcinoma, and 3 dogs with rectal carcinomas.

Alternatively, the lower prevalence of overweight and obese dogs may indicate an indirect effect of the cancer itself (ie, cancer cachexia), which results in a negative energy balance and a higher prevalence of underweight dogs. In 1 report,[50] investigators focused on cancer cachexia as a disease syndrome in dogs examined at a referral veterinary teaching hospital. Body condition scores (based on a 9-point scale) were recorded at the time of hospital admission and used as an indicator of obesity in that study. Of the 100 dogs included in that report,[50] 55 were assigned a BCS ≥ 6/9 at the time of initial examination, and 29 of those dogs were considered obese with a BCS ≥ 7/9. The limited data on historical body weight available for that study and the study reported here make it difficult to reach direct conclusions regarding cancer cachexia or obesity as a risk factor in the development of cancer in dogs. Interestingly, although steroid administration had a significant influence on the BCS distribution of dogs included in our study, it did not appear to mask the lower prevalence of overweight and obese dogs with cancer.

The influence of obesity on tumor development in dogs has been examined.[30-33] Investigators in the earliest study[30] collected owner reports of obesity 1 year prior to diagnosis of TCC of the urinary bladder in 59 dogs and concluded that obesity significantly increased the risk for development of TCC in dogs. Investigators in the other 3 studies[31-33] evaluated the role of obesity on the development of mammary gland cancer in dogs.

Investigators in 2 of those studies[31,32] collected owner reports of obesity status when dogs were 1 year old and relied on hospital records for body weight 1 year before cancer diagnosis and at the time of cancer diagnosis to determine body weight index (comparison of body weight with the breed standard for each dog). Of the 150 dogs with mammary gland tumors evaluated in 1 study[31] and 102 dogs with mammary gland tumors or dysplasia evaluated in another study,[32] there was a significantly higher prevalence of owner-reported obesity but no difference in body weight index at any time point. The remaining study[33] revealed a similar negative finding when body weight index was evaluated at the time of diagnosis of mammary gland cancer in 99 dogs.

The aforementioned studies were limited to relatively small numbers of dogs with a single cancer type and relied on nonstandardized measurements of obesity or owner recall of past obesity. Assigning obesity on the basis of breed standards may overrepresent or underrepresent obesity in dogs that are at an ideal body condition yet are outside of the standard weight range for their particular breed. Additionally, epidemiologic studies in humans avoid the use of personal reported histories to prevent recall bias[51] or overreporting of conditions believed to increase the risk of disease. The use of the 9-point BCS scale attempted to circumvent issues associated with owner recall or comparisons with breed standards.

In the study reported here, a higher prevalence of overweight and obese dogs at the time of diagnosis was found for the 49 dogs with mammary gland cancers by use of a more standardized measure of obesity; this finding is provocative although not substantial. In our study, we did not collect body weights or BCS values 1 year before diagnosis or at 1 year of age for dogs with mammary gland cancers or TCC; therefore, comparisons for this aspect with results of other published reports was not possible.

Dogs with B-cell lymphoma were selected for collection of data on historical body weight because of the ability of that cancer to relatively rapidly progress to clinical illness.[52] There was no difference in BCS distribution of these dogs, compared with results for noncancer control dogs, and there was no difference in body weights at the time of initial examination at the VMTH, compared with body weights collected 6 to 12 months before initial examination, which suggests that there is no association between body weight status and course of cancer.

The most pronounced difference in BCS distribution was for dogs with undifferentiated lymphoma in which historical body weights were not collected. The higher prevalence of underweight and extremely thin dogs in combination with the lower prevalence of overweight dogs with this type of cancer, irrespective of corticosteroid administration, suggests a differential effect of this type of cancer on energy balance (ie, cancer cachexia) or an increased risk of development of this type of cancer in thin and underweight dogs. Historical body weight for dogs with this type of cancer would help further address this question. The unexpected finding of a higher prevalence of overweight dogs with mast cell tumors would appear to indicate a detrimental influence of obesity on the development of this type of cancer. Historical information on body weight would be useful in further evaluation of this relationship as well.

Variation in the prevalence of obese dogs based on type of cancer in the analyses suggests an incongruous relationship between obesity status and development or expression of cancer. Neither the study reported here nor the other reports on cancer in dogs answer the question as to whether obesity is related to cancer by causation or correlation. Collaborative, multiple-center prospective veterinary studies that involve large numbers of dogs would better elucidate the influence of obesity on specific types of cancer and help researchers determine whether obesity in general or at any specific life stage plays an important role in cancer development. The most pertinent information available in the veterinary literature is the mortality data from a long-term prospective study[53] on calorie restriction in 48 Labrador Retrievers, which revealed an equal distribution of cancers among control and restricted-fed dogs. The various types of cancer reported, limited sample size, and avoidance of overt obesity in the dogs of that study make it difficult to reach direct conclusions about obesity and cancer development.

Routine recording of BCS in medical records of all dogs is strongly recommended and may aid in the identification of at-risk patients prior to the development of overt obesity. Furthermore, it may help clarify interrelated effects of obesity and cancer development. Systematic use of BCS in medical records would also allow for additional prospective and retrospective studies on the general health risks of obesity in the canine population.

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