Nutrient Requirements of Dogs

Revised 1985

Subcommittee on Dog Nutrition
Committee on Animal Nutrition
Board on Agriculture
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This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

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Preface

This report is one in a series issued under the direction of the Committee on Animal Nutrition, Board on Agriculture, National Research Council. It was prepared by the Subcommittee on Dog Nutrition and replaces the 1974 edition of *Nutrient Requirements of Dogs*.

The report describes common signs of deficiency and of toxicity and discusses the basis for arriving at requirements for energy and for specific nutrients.

This new edition contains recommendations for available nutrient content of representative commercial dog foods expressed on the basis of metabolizable energy content. This change should lead to greater uniformity in the nutritional adequacy of foods with varying caloric density and facilitate meaningful comparisons of such products.

The subcommittee expresses appreciation to all individuals who contributed to this report, in particular, David H. Baker, Norlin J. Benevenga, and H. Meyer, and members of the Nutrition Task Force of the Pet Food Institute who reviewed the report and provided insightful comments and suggestions.

Review of this report was accomplished through the advice and guidance of the members of the Committee on Animal Nutrition. The subcommittee is particularly indebted to Philip Ross and Selma P. Baron of the Board on Agriculture for their valuable assistance in the preparation of the report. The subcommittee is especially grateful to James E. Corbin, who served as coordinator for the Board on Agriculture in the review of this report.

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Introduction

Canine nutrient requirements may have changed little since dogs were first domesticated. However, knowledge and understanding of nutrient requirements and their applications have changed dramatically. The objective of this revision, in keeping with guidelines of the National Research Council's Committee on Animal Nutrition, is to provide from currently available published information a summary of the minimum dietary requirements of dogs for the essential nutrients. These requirements are presented in Table 1 (see p. 44) and are expressed as amounts per kilogram of body weight per day.

Dogs require dietary sources of energy, amino acids, glucose precursors, fatty acids, minerals, vitamins, and water. Suitable dietary sources of nutrients to meet these requirements include plant, animal, and synthetic products, provided that appropriate processing procedures are followed in their preparation and that consideration is given to variations in specific nutrients available from individual sources or combinations of sources.

Food intake recommendations are based on energy content of the diet. It is therefore reasonable to conclude that nutrient content of foods should also be related to energy content of the food. Minimum contents of essential nutrients for dog foods based on requirements for growth are listed in Table 2 (see p. 44) and expressed as available nutrients in units per 1,000 kilocalories (kcal) of metabolizable energy (ME) and on a dry matter basis. Expression of nutrient requirements on these bases will simplify and expedite comparisons of nutritional value of practical diets with widely varying energy and/or moisture contents.

These recommended requirements are intended to serve as a guide to the formulation of foods and to meet the requirements of dogs of average circumstances of age, function, physiological state, and environmental condition. Special circumstances and experience may warrant modifications of requirements to provide higher or lower concentrations of individual nutrients or groups of nutrients. The reader should be aware that dietary excesses and imbalances can be as detrimental to health as dietary deficiencies.

Finally, caution is advised in the use of these requirements without demonstration of nutrient availability, because in some cases requirements have been established on the basis of studies in which nutrients were supplied by highly purified ingredients where digestibility and availability were not compromised by the interaction of dietary constituents and effects of processing. Practical diets formulated from commonly used ingredients are not free of such interactions and effects, and therefore may provide less available nutrients than the amounts measured by chemical analysis. For this reason, such diets formulated to the chemically assayed nutrient levels expressed in Table 2 may prove inadequate in meeting the nutritional needs of dogs. Table 3 (see p. 45) indicates some of the factors that may modify dietary requirements. Therefore, users are advised to obtain evidence of nutritional adequacy by direct feeding to dogs.

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Nutrient Requirements and Signs of Deficiency

ENERGY

When food is completely oxidized in a bomb calorimeter, the total combustible energy released as heat is known as gross energy (E). Gross energy values for mixed carbohydrates, fat, and protein average 4.15, 9.40, and 5.65 kcal/g, respectively. However, not all gross energy contained in food is available for metabolism. An undigested fraction is excreted in the feces. The difference between the gross energy consumed and the gross energy in the feces is referred to as *apparent digestible energy* (DE). Additional energy losses occur, namely, energy in excreted urine and combustible gases. For practical reasons, only energy in urine is subtracted from DE to determine metabolizable energy (ME) (NRC, 1981). The ME content of a food is a valid expression of the energy available to the dog and a basis for comparisons of the feeding value of various foodstuffs. ME values for most of the individual ingredients listed in Table 7 (see p. 48) have not been determined for dogs. Therefore, approximations of their ME content must be calculated. A method recommended for calculation is based on assumed apparent digestibilities of 80 percent for protein, 90 percent for ether extract, and 85 percent for nitrogen-free extract (NFE). The resulting digestion coefficients are then multiplied by gross energy values of 4.40 (5.65 – 1.25*), 9.40, and 4.15 kcal for protein, ether extract, and NFE, respectively. It is assumed that no ME was derived from crude fiber. The resulting values of 3.50, 8.46, and 3.50 kcal are reasonable estimates of the ME available to dogs from protein, fat, and carbohydrate (NFE) from feed ingredients commonly used in the manufacture of dog foods (see Chapter 4, section on Metabolizable Energy).

Expressing the energy requirements for all dogs is not a simple task. Adult size and growth rates of breeds vary greatly. Mature body weights range from 1 kg for the Chihuahua to 90 kg for the St. Bernard. From a physiological viewpoint, energy requirements of animals with widely differing weights are not directly related to body weight but are more closely related to body weight raised to some power, W^b, where W equals weight in kilograms and b is an exponent calculated from experimental data. Brody et al. (1934) found that the basal heat production of mature warm-blooded animals, ranging in size from mice to elephants, could be described by the expression $Y = 70.5\ W^{0.73}$, where Y equals kilocalories per 24 h and W equals body weight in kilograms. Kleiber (1961) argued that over such a range in body size, Brody's expression and $Y = 70\ W^{3/4}$ would not be significantly different and that the latter would be simpler to use.

More recently Heusner (1982) demonstrated that the use of the 0.75 interspecific mass exponent in Kleiber's equation is a statistical artifact. Many people assumed that the relationship between basal metabolic rate (BMR) and metabolic body weight was similar for mice and cows. Heusner argued that the theoretical exponent should be 0.67 to describe the intraspecies (e.g., dogs) relationship of energy to mass.

Requirements for Adult Maintenance

Energy requirements for maintenance of adult dogs have been studied by Cowgill (1928) and estimated by Arnold and Elvehjem (1939) from the prediction equations of Brody et al. (1934). Abrams (1962) also published estimates of maintenance energy requirements of adult male dogs that conform closely to previous values, although it is not clear how these data were derived.

* Gross energy of protein corrected for nitrogen energy loss in metabolic products.

Payne (1965), using Abrams's data, published estimates of metabolizable energy requirements for maintenance of growing, adult, pregnant, and lactating dogs.

Abrams (1976) recalculated available data related to the determination of BMR in dogs. Estimates were made of energy requirements for maintenance of adult dogs in terms of DE and expressed as joules (J) needed per day or per kilogram of body weight. Constants were determined by regression analysis and an equation for calculating kJ energy requirement was derived. Of significance is that estimates for males exceeded those for females.

Abrams (1976) reported greater metabolizable energy requirements for dogs weighing less than 20 kg than those calculated by the method of the 1974 NRC report ($132 \times BW_{kg}^{0.75}$), but his values did not differ greatly for heavier dogs. More recently, Blaza (1981) measured ME requirements for maintenance of medium and giant breeds of dogs. Great Dane and Newfoundland dogs required 1.5 and 1.3 times, respectively, the calculated ME from the equations of NRC (1974). Requirements of Labrador Retrievers were comparable to requirements predicted from NRC (1974). The Subcommittee on Dog Nutrition decided to seek available data for maintenance based on controlled feeding conditions and to develop a prediction equation using the approach advocated by Thonney et al. (1976).

Limited data were available for individual dogs weighing from 4 to 36 kg. Seven breeds (Beagle, Boxer, Labrador Retriever, Pointer, Poodle, and two types of Dachshund) were included. From visual inspection of the scatterplot it could not be concluded that either breed or sex differences affected the relationship of daily ME required for maintenance of body weight.

The data were fitted to a linear model and an allometric model. The simple linear model ($ME = b_0 + b_1W$, where b_0 is the intercept, b_1 is the slope, and W is weight in kilograms) described the within-species relationship between basal heat production and weight. The allometric model used was $ME = b_0W^{b_1}$ (where b_0 is the mass coefficient and b_1 is the exponent). The allometric approach to energy data was first used by Kleiber (1932) on data in which the lightest animal (rat) weighed close to zero compared to the heaviest (cattle). This model implies that the relationship will intersect the origin. There is no reason to use the allometric model unless a curved line that intersects the origin fits the data better than a simple straight line. Since large weight differences comparable to those in the data used by Kleiber (1932) do not exist within most species (including dogs), it is unlikely that the best model is one that requires the relationship to intersect the origin.

The results of fitting the two models to the data are shown in Figure 1. The linear equation, $ME = 144.4 + 62.2\ W$, and the allometric equation, $ME = 99.56\ W^{0.879}$, both explained 85.6 percent of the variation with a standard deviation of 260 kcal. The daily ME requirement predicted by these equations is shown in Table 5 (see p. 45) for dogs varying in weight from 1 to 60 kg. Either equation may be used.

The NRC (1974) equation ($ME = 132\ W^{0.75}$) is also shown in Figure 1, but it explains less of the variation than the allometric and linear equations actually derived from the data. It underpredicts the ME requirements of the larger dogs and, thus, supports the work of Blaza (1981). More data are needed, however, to predict accurately the energy needs of dogs greater than 35 kg mature body weight. Therefore, until more data are available, it may be advisable to determine feeding levels based on the 1974 equation predictions.

These values or any others cannot be taken as absolute ME requirements for any individual or breed of dog, since needs vary with age, activity, body condition, insulative characteristics of the hair coat, temperature, acclimatization, external environmental circumstances, and psychological temperament.

Finally, it would not appear to serve much practical purpose to further refine energy requirements of even "average" dogs, since the ME concentration of foods available to such "average" dogs is frequently either unknown or can only be calculated by methods resulting in no greater precision.

Generally, adult dogs adjust their food intake to energy requirements. Cowgill (1928) found that dogs previously adjusted to an appropriate intake of a particular diet consumed fewer grams, but a similar number of calories, when a higher-energy-density diet was offered. Durrer and Hannon (1962) reported that caloric intake varied inversely with long-term changes in environmental temperature. In July, when the mean temperature was 17°C, Beagles consumed approximately 163 kcal of ME per $W_{kg}^{0.75}$ perday, while Huskies consumed 127. In November, when $W_{kg}^{0.75}$ temperatures were -17°C, the respective daily ME intakes for Beagles and Huskies were 278 and 205 kcal per . Huskies exhibited a marked increase in hair growth during November and December, while little seasonal change in hair growth was seen in Beagles. Dogs of both breeds minimized heat loss during extremely cold weather (less than -40°C) by curling into a ball and tucking their noses and tails underneath their bodies. While Huskies showed no evidence of shivering and refused to sleep in plywood shelters, Beagles shivered and sought shelter. These data illustrate the marked effect on energy requirements imposed by the environment and the additional influence of differences in breed and behavior.

While weight changes were small, both Beagles and Huskies were heavier in the summer than in the winter.

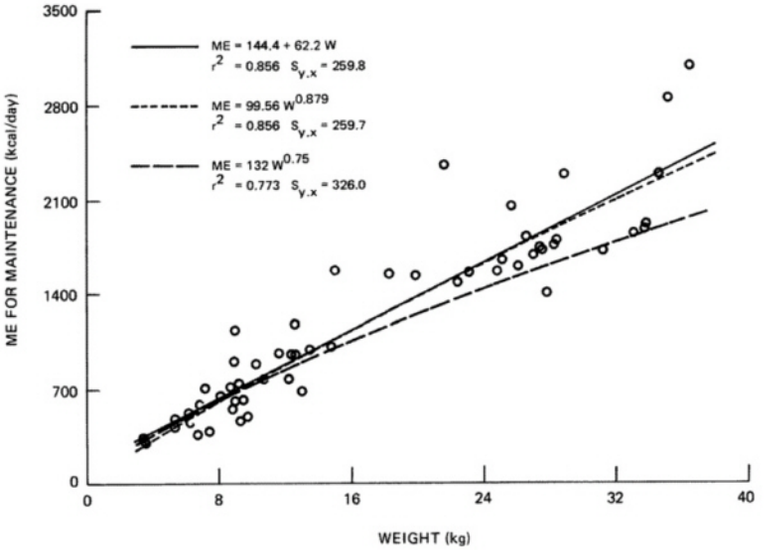

FIGURE 1 Relationship between metabolizable energy and adult body weight.

Figure 1 Relationship between metabolizable energy and adult body weight.

These data support what is increasingly recognized in practice, namely, the inability of many dogs to accurately adjust food intake to meet requirements for biological energy. When dogs are kept in a controlled environment with limited opportunity to exercise and are fed highly palatable, nutrient-dense foods, the ability to regulate food intake for optimum body weight may be compromised. Obesity, common to many pets, is the ultimate result of caloric intake in excess of metabolic requirements. Regardless of the accuracy of any prediction of energy requirements, the judgment of how much to feed must ultimately be with the individual feeding the dog. This judgment is based on weight, conformation, and general appearance of the dog in question.

Requirements for Growth

Growing puppies require about 2 times as much energy per unit of body weight as adult dogs of the same breed (Arnold and Elvehjem, 1939). The newly weaned dog can readily adapt to this level of feeding, particularly when the food is offered in multiple judiciously spaced meals. However, an arbitrary decrease to 1.6 times maintenance is recommended when 40 percent of adult body weight is achieved, and 1.2 times maintenance when 80 percent of adult weight is reached. This reduction compensates for the decline in energy required from weaning to adult age. Excessive nutrient intake from weaning to adolescence, resulting in maximal growth rates, is incompatible with proper skeletal development (Hedhammar et al., 1974).

Requirements for Reproduction and Lactation

Limited studies and practical experience suggest that energy requirements of the normal pregnant bitch are only slightly more than those recommended for maintenance for the first two-thirds of gestation. During the last trimester, energy requirements may increase to as much as 150 to 160 percent of preconception values (Romsos et al., 1981). Energy requirements during lactation increase greatly and are influenced by size of the litter. Bitches with large litters may require 3 or more times the maintenance energy requirement. The ability to meet these requirements may be limited by the capacity of the bitch to consume sufficient food. Foods of high

nutrient density are recommended for feeding at this time.

Requirements for Work and Adverse Environmental Conditions

The range of work performed by dogs may vary from the limited exercise characteristic of the apartment pet to the intense effort of the working sled dog. Systematic schedules for meeting such diverse requirements are impractical. Thus, it is recommended to feed to thrifty body condition. It should be pointed out, however, that while a conditioned racing Greyhound may require energy only 10 to 20 percent above that of maintenance, a sled dog working under polar conditions may require 2 to 4 times the maintenance requirement in order to avert significant weight loss (Kronfeld et al., 1977). Conversely, working dogs in hot, humid environments may require 50 to 100 percent more energy than similar dogs in less stressful circumstances (McNamara, 1971).

Signs of Deficiency

Signs of energy deficiency are frequently nonspecific, and diagnosis may be complicated by a simultaneous shortage of several nutrients. The most conspicuous and reliable sign of uncomplicated energy deficiency is a generalized loss of body weight. Under conditions of partial or complete starvation, most internal organs exhibit some atrophy. A loss of subcutaneous, mesenteric, perirenal, uterine, testicular, and retroperitoneal fat is an early sign. Low fat content of the marrow in the long bones is a good indicator of prolonged inanition. Brain size is least affected, but the size of gonads may be greatly decreased. Hypoplasia of lymph nodes, spleen, and thymus leads to a marked reduction in their size. The adrenal glands are usually enlarged. The young skeleton is extremely sensitive to energy deficiency, and growth may be slowed or stopped completely. In the adult the skeleton may become osteoporotic. Lactation and the ability to perform work are also impaired. As muscle proteins are catabolized for energy, endogenous nitrogen losses increase. Parasitism and bacterial infections frequently occur under such circumstances and may superimpose other clinical signs.

FAT

Dietary fat is a concentrated source of energy and provides essential fatty acids. These essential fatty acids serve structural functions in cell membranes and in metabolic regulation, e.g., as precursors of prostaglandins and related metabolites. Fat also serves as a carrier of fat-soluble vitamins and lends palatability and a desirable texture to dog food.

Dietary Fat Concentration

Proper formulation of diets containing fat requires an adjustment for the high energy value of fats. Because the ME concentration of digestible fat is approximately 2.25 times the ME concentration of digestible carbohydrate or protein, substitution of fat on an equal weight basis for these nutrients will increase the energy density of the diet. As a result, dogs fed high-fat diets formulated without consideration for the higher energy value of fat may exhibit nutrient deficiencies. This occurs because dogs generally respond by eating less of a high-fat diet to maintain near-normal energy intake (Cowgill, 1928). Consequently, daily intake of protein, minerals, and/or vitamins may not be adequate. The adverse consequences of improper formulation of high-fat diets on the growth rate of puppies has been demonstrated (Elvehjem and Krehl, 1947; Campbell and Phillips, 1953; Ontko et al., 1957; Crampton, 1964).

When the formulation of high-fat diets is adjusted to ensure adequate intake of protein, minerals, and vitamins, diets with wide ranges in fat concentration appear compatible with good health of dogs. (See Newberne et al., 1978, for examples of procedures for formulating high-fat diets.) Apparent digestibility of fat by dogs varies from approximately 80 to 95 percent when mixtures of glycerides from plant and animal sources are fed (James and McCay, 1950; Orr, 1965). The growth rate of Beagle puppies between 2 and 6 months of age was unaffected by feeding purified, canned diets ranging in composition from 13 to 76 percent of energy from fat and containing 20 to 25 percent of energy from protein (Romsos et al., 1976). Between 6 and 10 months of age, puppies fed the lower-fat diet (13 percent fat) gained less body weight because they gained less body fat than did puppies fed diets containing 38, 55, or 76 percent of energy from fat. Results of this study and several others (Siedler and Schweigert, 1952; Campbell and Phillips, 1953) demonstrated that puppies exhibit satisfactory growth when fed diets with rather widely varying concentrations of fat if amino acid requirements are met.

Reproductive performance of bitches fed diets varying in fat concentration has received only limited attention. Siedler and Schweigert (1954) indicated that reproductive performance was somewhat better when Cocker Spaniel bitches were fed a diet containing 7.7 percent fat rather than a diet containing either 3.7 or 11.7 percent fat. Because their diets were formulated by adding 4 and 8 percent fat to the diet containing 3.7 percent fat, the ratio of essential nutrients to ME was

progressively decreased in the higher-fat diets. This may have adversely affected performance of bitches fed the diet containing 11.7 percent fat. Ontko and Phillips (1958) fed diets containing 8 or 16 percent fat and observed satisfactory reproductive performance. Provided appropriate adjustments are made to maintain an adequate nutrient-to-energy ratio, it would appear that diets with rather widely varying concentrations of fat will also permit satisfactory reproductive performance.

Sedentary adult dogs have a greater tendency to become obese when fed high-fat diets ad libitum rather than high-carbohydrate diets. When adult female Beagles were fed ad libitum a diet containing 51 percent of energy from fat for 25 weeks, they gained twice as much body fat as dogs fed a diet containing 23 percent of energy from fat (Romsos et al., 1978). Other animals also tend to gain more body fat when fed a high-fat diet than when fed a low-fat diet (Sclafani, 1980). A slight restriction in food intake, however, will prevent development of obesity even if high-fat diets are fed.

The concentration of fat in the diet may affect work performance of dogs. Fatty acids are the primary source of energy for skeletal muscle during exhaustive exercise (Therriault et al., 1973). When adult Beagles that had been maintained in a high state of physical conditioning and had been fed a cereal-based diet with 7 percent fat were fasted for 5 days, their endurance capacity increased by 74 percent relative to their endurance in the fed state (Young, 1959). It was concluded that this improvement in work performance was mediated by an enhanced ability to mobilize body fat. Consumption of a high-fat diet rather than a high-carbohydrate diet has been shown to lengthen the time to exhaustion of Beagles on a treadmill by approximately 30 percent (Downey et al., 1980) and to cause a greater elevation in plasma-free fatty acid concentration during exercise of sled dogs (Hammel et al., 1977).

Essential Fatty Acids

Dogs, like other animals, have a dietary requirement for certain polyunsaturated fatty acids. These fatty acids have been shown to stimulate growth and cure the dermatitis characteristic of dogs fed a diet very low in fat or a diet in which the fat is completely saturated (Hansen et al., 1948, 1954; Hansen and Wiese, 1951; Wiese et al., 1965, 1966).

Members of the linoleic acid n-6 (denotes the position of the first double bond from the terminal end of the chain) family including linoleic acid, C18:2 (n-6); -linolenic acid, C18:3 (n-6); and arachidonic acid, C20:4 (n-6) all exhibit essential fatty acid activity. The shorthand terminology used denotes the number of carbon atoms in the fatty acid (followed by a colon) and the number of double bonds. Because C18:2 (n-6) can be desaturated and elongated to form C18:3 (n-6) and C20:4 (n-6) (Mead, 1980), and because the latter two fatty acids are only minor components of most natural fats, the requirement for fatty acids of the n-6 family is usually expressed as linoleic acid. Linoleic acid concentrations of a variety of ingredients used in dog foods are shown in Table 6 (see p. 46).

The minimum amount of linoleic acid (or other members of the n-6 family of fatty acids) required by the dog has not been precisely determined. The pathological and biochemical changes in the skin produced by an essential fatty acid deficiency can be reversed when 2 to 6 percent of the ME requirement is provided by linoleic acid or arachidonic acid (Hansen and Wiese, 1951; Wiese et al., 1966). One percent of the ME requirement as linoleic acid does not appear to be adequate for growing puppies (Wiese et al., 1966).

Several factors including rate of growth and concentrations of saturated fatty acids, *trans* fatty acids, and monounsaturated fatty acids in the diet influence the requirement for essential fatty acids (Mead, 1980; Holman, 1981). Beagle puppies fed a low-fat diet at 200 kcal ME per kilogram of body weight per day exhibited skin lesions within 2 to 3 months (Wiese et al., 1962). When the level of intake was reduced to 150 kcal ME per kilogram body weight per day, lesions appeared in 3 to 4 months. Puppies fed 100 kcal ME per kilogram of body weight per day did not grow, nor did they exhibit gross or histological evidence of fat deficiency during the 5-month study. Based on data obtained with rodents (Mead, 1980; Holman, 1981), one would predict that high intakes of saturated fatty acids, *trans* fatty acids, or oleic acid [C18:1 (n-9)] by dogs would compete with the metabolism of essential fatty acids and thereby increase the requirement for essential fatty acids. However, data are unavailable for estimating the extent to which these fatty acids might increase the requirement for essential fatty acids in dogs.

There is speculation that fatty acids of the -linolenate [C18:3 (n-3)] family also exhibit essential fatty acid activity in animals (Mead, 1980; Budowski, 1981). In rats C18:3 (n-3) promotes normal growth but does not prevent the skin lesions associated with lack of n-6 fatty acids (Mead, 1980). Fatty acids of the n-3 family have been suggested to serve some special function in the nervous system (Mead, 1980; Holman et al., 1982). They also are precursors of prostaglandins that may play an important role in control of blood clotting (Budowski, 1981). Data are unavailable to indicate if dogs have a requirement for fatty acids of the n-3 family.

Recommendation

It is recommended that a dog food contain at least 5 percent fat on a dry basis, including 1 percent of the diet

as linoleic acid. Because not all fats are rich in linoleic acid (Table 6), supplemental fats must be chosen judiciously when total fat is limited to 5 percent. Although these concentrations appear sufficient for normal physiological functions, higher concentrations of fat may be desirable in practical dog foods to enhance acceptability and to improve hair coat sheen. If such increases are made, the concentrations of other nutrients should be appropriately increased to maintain a satisfactory nutrient-to-energy ratio, i.e., when substantial fat supplementation is implemented, total dietary reformulation is in order to prevent nutrient imbalances from occurring.

Signs of Deficiency

Puppies fed a low-fat diet (probably less than 0.01 percent linoleic acid) but with a high energy intake per day began to show coarse, dry hair and desquamation on the ventrum after 2 to 3 months. After 4 to 5 months (6 to 7 months of age), these lesions became severe. With normal energy intake, appearance of the lesions was delayed about 1 month (Wiese et al., 1962). The earliest gross lesions appeared on the abdomen, then on the thigh, and last in the interscapular area. Histologically, the epidermis was edematous and thickened, with up to 12 layers of cells in the most severely affected areas. Keratinization was deranged and, as the deficiency advanced, parakeratosis became evident. Maturation of the epidermal cells seemed impaired. Affected areas were invaded first by mononuclear cells, followed later by polymorphonuclear neutrophils. The epidermis appeared ulcerated and was more susceptible to infection. Linoleic acid and arachidonic acid concentrations in the skin decreased markedly.

CARBOHYDRATES

All animals have a metabolic requirement for glucose to supply energy for organs, including the central nervous system, and to supply substrate for synthesis of compounds such as pentoses and glycoproteins. Provided the diet contains sufficient glucose precursors (amino acids and glycerol), the glucogenic capacity of the liver and kidneys is usually sufficient to meet the metabolic need of growing animals for glucose without inclusion of carbohydrate in the diet (Brambila and Hill, 1966; Chen et al., 1980).

Beagle puppies have been fed purified, canned diets ranging in composition from 0 to 62 percent of metabolizable energy from carbohydrate (cornstarch) and from 13 to 76 percent of metabolizable energy from fat (corn oil, tallow, and lard) to determine if dietary carbohydrate is required for growth and maintenance of normal blood glucose levels (Romsos et al., 1976). Protein (20 to 25 percent of energy) in these diets was derived from approximately equal proportions of lean beef and isolated soybean protein. Weight gain of the 2-month-old Beagles fed the carbohydrate-free diet containing 24 and 76 percent of energy from protein and fat, respectively, for 8 months was comparable to the gain of puppies fed diets containing 20 to 62 percent of energy from carbohydrate. Pups fed the carbohydrate-free diet also maintained normal plasma glucose concentrations and normal rates of glucose utilization (estimated by disappearance of [2-^{3}H] glucose) (Belo et al., 1976). These results agree with earlier reports demonstrating that growing rats (Chen et al., 1980) and chickens (Brambila and Hill, 1966) do not appear to have a dietary requirement for carbohydrate provided adequate dietary glucose precursors are available in the form of glucogenic amino acids and glycerol.

A dietary requirement for carbohydrate has been demonstrated in growing rats (Chen et al., 1980) by reducing the concentration of protein in the diet to 13 percent and by providing unesterified fatty acids, rather than glycerol-containing triglycerides, as the only nonprotein energy source. Rats fed this diet gained less weight and had depressed blood glucose concentrations. Either adding 6 percent glucose or doubling the concentration of protein in the diet to 26 percent increased their growth rate to equal the growth rate of control rats. Similar experiments have not been reported with dogs, but it seems probable that consumption of a diet devoid of carbohydrate and low in glucose precursors would also cause hypoglycemia and limit growth rate of dogs.

During gestation and lactation the metabolic requirement for glucose is increased to supply needs for fetal development and lactose synthesis, respectively. When, starting 31/2 to 4 weeks after conception, Beagle bitches were fed a diet containing 26, 74, and 0 percent of energy from protein, fat, and carbohydrate, respectively, their food intake, body weight increase, and plasma glucose concentrations during the first two trimesters of gestation were comparable to values in bitches fed a diet containing 26, 30, and 44 percent of energy from protein, fat, and carbohydrate, respectively (Romsos et al., 1981). However, during the week before whelping, bitches fed the carbohydrate-free diet developed hypoglycemia and had depressed plasma concentrations of two key glucose precursors (lactate and alanine). Total number of pups whelped by the bitches was unaffected by diet, but fewer pups from bitches fed the carbohydrate-free diet were alive at birth (63 percent) than from bitches fed the carbohydrate-containing diet (96 percent). Only 35 percent of the pups whelped by bitches fed the carbohydrate-free diet were alive at 3 days of age. The cause of death of the pups was not established, but they probably had less ability to maintain their plasma glucose concentrations immediately after delivery

than did control pups (Kliegman et al., 1980). Additionally, the lethargic condition of the hypoglycemic bitches reduced their mothering ability immediately after delivery.

The severe hypoglycemia (plasma glucose concentrations as low as 15 to 20 mg/dl) and ketosis (blood-hydroxybutyrate concentrations as high as 2.5 mM) that develop at whelping in some bitches fed a carbohydrate-free diet (Romsos et al., 1981) are not unique to dogs. Hypoglycemia and ketosis are also often observed during the last trimester of gestation in ewes carrying twins or triplets (Bergman, 1973). Since most of the carbohydrate consumed by ewes is fermented in the rumen, they absorb only small amounts of glucose and therefore depend on endogenous synthesis to supply their need for glucose. Under conditions of accelerated need for glucose such as occur in the last trimester of gestation, both dogs and ewes sometimes fail to meet these needs from endogenous synthesis. It is possible, based on observations with pregnant rats (Taylor et al., 1983), that bitches also require some carbohydrate in their diet during the period immediately postconception. Rats fed a carbohydrate-free diet from the day of conception exhibit an increase in early embryonic abnormalities and resorption (Taylor et al., 1983). Because the bitches were not switched to their experimental diets until 3 ½ to 4 weeks after conception (Romsos et al., 1981), effects of a carbohydrate-free diet on the early stages of their gestation remain unknown. It is recommended that diets of pregnant bitches contain some available carbohydrate for optimal reproductive performance.

Lactating Beagle bitches have also been fed a carbohydrate-free diet containing 26 percent of energy from protein and 74 percent from fat to determine if they require dietary carbohydrate for lactation (Romsos et al., 1981). To maximize the need for milk synthesis, each bitch nursed six pups, and the pups were not allowed to consume the bitches' diet during the first 4 weeks of lactation. Pups suckling bitches fed the carbohydrate-free diet grew as well as those suckling bitches fed the carbohydrate-containing diet. Likewise, plasma glucose concentrations of the lactating bitches were unaffected by the absence of carbohydrate in the diet. Because the milk of Beagles contains less than 20 percent of energy from lactose (Luick et al., 1960; Romsos et al., 1981), the need for glucose in lactating Beagles is less than the need during gestation when glucose is a major energy source for fetal development. This may explain why consumption of a carbohydrate-free diet adversely affected performance of the Beagles during gestation but not during lactation. In breeds of dogs that have larger litters and a greater milk production than Beagles it is possible that the metabolic demand for glucose could exceed the ability of the bitch to synthesize it. At least in high-producing dairy cows, hypoglycemia and ketosis have been shown to develop during peak lactation (Bergman, 1973).

Carbohydrates provide an economical source of energy in the diet of dogs. Cooked starch is well digested by adult dogs (Roseboom and Patton, 1929; Ivy et al., 1936; James and McCay, 1950; Heiman, 1959) and by growing Beagles (Romsos et al., 1976). Growing Beagles fed a purified, canned diet containing 62 percent of energy from cornstarch exhibited an apparent energy digestibility of 84 percent, whereas energy digestibility was slightly higher (87 to 89 percent) for dogs fed diets containing less starch (0 to 42 percent of metabolizable energy) and more fat (38 to 76 percent of metabolizable energy). Weight gain of Beagles fed the diet containing 62 percent cornstarch from 2 to 10 months of age was somewhat less than for dogs fed diets with less starch and more fat. This difference in weight gain occurred because dogs fed the high-starch diet gained less body fat; gain in fat-free mass was unaffected by concentration of starch in the diet. Raw starch is less well utilized by dogs than is starch that has been subjected to some dextrinization by processing (for example, cooking, baking, or toasting) (Heiman, 1959).

Utilization of simple carbohydrates such as lactose, dextrin-maltose, and sucrose has also been evaluated in dogs. Suckling puppies obviously utilize lactose, although as mentioned above, the milk of Beagles contains a relatively low percentage of energy from lactose. Sudden introduction of large amounts of lactose into the diet of adult Beagles causes diarrhea (Bennent and Coon, 1966). This response is not unique to dogs—adults of a number of other species would also exhibit diarrhea if abruptly challenged with large amounts of lactose. Diets containing as much as 49 percent by weight of sucrose support satisfactory body weight gain in immature Beagles (Milner, 1979). Young adult Beagles show a preference for sucrose-containing diets when allowed to self-select between a sucrose-supplemented diet and a control, starch-containing diet (Houpt et al., 1979). Although sucrose is well utilized by dogs in the postweaning period, there is the possibility, based on research in other species (Becker et al., 1954), that intestinal sucrase activity is low immediately after birth, in which case sucrose should not be included as a significant component in artificial milk for puppies.

Fiber is not generally considered essential for simple-stomached mammals, including dogs, although inclusion of some fiber in the diet may be beneficial. Energy density of the diet is reduced by fiber, and therefore inclusion of some fiber in the diet may contribute to the maintenance of ideal body weight in adult sedentary dogs fed ad libitum. Fiber reduces gastrointestinal transit time in simple-stomached mammals. But because

dogs have a relatively short gastrointestinal tract, ingesta transit time is relatively rapid even when a low-fiber diet is fed (Banta et al., 1979). As a result of their short gastrointestinal tract, dogs have a low colonic-rectal surface area per unit body weight. For example, Beagle dogs have only 17 cm^2 colonic-rectal mucosal area per kilogram of body weight, whereas the comparable value for pigs is 200 cm^2 per kilogram of body weight (Herschel et al., 1981). Consequently, dogs are unlikely to absorb significant amounts of energy from the fermentation of fiber that might occur during its relatively rapid transit through the lower gastrointestinal tract. Finally, although data are unavailable for the dog, it should be recognized that inclusion of large amounts of fiber in the diet may adversely affect nutrient availability.

PROTEIN AND AMINO ACIDS

Dietary protein is required to supply specific essential amino acids that cannot be synthesized in sufficient quantities by tissues to allow for optimum performance. Additionally, dietary nitrogen is required to allow for optimal biosynthesis of the dispensable amino acids and other nitrogenous compounds. Recent studies by Milner (1979 a,b) using purified diets containing 4.1 kcal ME/g have established that the following amino acids are required for optimum growth and nitrogen balance in the immature Beagle:

arginine	methionine
histidine	phenylalanine
isoleucine	threonine
leucine	tryptophan
lysine	valine

Studies of Rose and Rice (1939) established that all of the above amino acids except for arginine were also required to maintain nitrogen equilibrium in adult female dogs. However, recent studies of Burns et al. (1981) have shown that dietary arginine is required by the mature dog to maintain body weight and to prevent emesis and other signs associated with hyperammonemia.

Numerous factors may modify the percentage of protein required in the diet. In establishing this requirement, factors such as digestibility, amino acid composition, availability of the protein source, caloric density of the diet, and physiological state of the dog must be considered. The quantity, including excesses and deficiencies, of essential (indispensable) and dispensable amino acids, plus other nonspecific nitrogen sources are factors that may influence the minimal percentage of dietary protein required for optimum growth and health. Estimates of the protein requirement of the dog can also vary depending on the methods and criteria used in their derivation.

Signs of Deficiencies

Protein deficiency in the dog results in depressed food intake, severe growth retardation or weight loss, hypoproteinemia, depletion of protein reserves, muscular wasting, emaciation, and, ultimately, death (Chow et al., 1945; Allison et al., 1946; Allison and Wannemacher, 1965; Burns et al., 1982). Edema sometimes accompanies the hypoproteinemia. Generally, during limited access to protein the hair coat becomes rough and dull in appearance, antibody formation is impaired, and milk production is depressed. Although the signs of protein deficiency are nonspecific and can be created by other dietary deficiencies, including caloric restriction, these signs do indicate the severity of dietary limitations on the dog's health and performance.

Removal of a single essential amino acid results in a prompt reduction in food consumption leading to a negative nitrogen balance. Generally there is a return to normal within a few days after replacing the limiting amino acid. Prolonged deficiency of any of the essential amino acids leads to a syndrome similar to that occurring during protein deficiency. Limitation in a dietary essential amino acid tends to be reflected by lowered concentrations of the specific amino acid in the blood plasma (Longnecker and Hause, 1959). Specific signs characteristic of a deficiency of individual amino acids in the dog have not been adequately documented.

Amino acid imbalances or antagonisms are known to increase the requirements for individual amino acids (Harper and Rogers, 1965; Harper et al., 1970). Some adaptation to minor imbalances and antagonisms appears to occur. Data obtained in other species indicate that the effects of imbalances or antagonisms are greater when suboptimal dietary nitrogen is offered but of lesser importance if all amino acids are in excess in the diet.

Amino Acid Requirements

Indispensable

The dietary requirement of a particular protein or a mixture of proteins is determined by the ability of the protein (s) to meet the dog's metabolic requirements for amino acids and nitrogen. The closer the supply of the complement of amino acids to the requirement, the lower the percentage of protein required in the dog's diet (Allison et al., 1947; Kade et al., 1948; Arnold and Schad, 1954). Amino acid requirements, as a percentage of the diet, decline from birth to maturity. However, Wannemacher and McCoy (1966) have suggested that

amino acid needs of adult dogs may increase from maturity to old age. Unfortunately, the influence of age has not been adequately studied. Research with other species reveals that in estimating requirements for amino acids, consideration must be given to the energy and total protein concentration of the diet (Wretlind and Rose, 1950; Bressani and Mertz, 1958; Milner, 1981). Similarities in metabolism among mammalian species suggest that it is prudent to consider these as important dietary factors for the dog until research proves otherwise.

Arginine Arginine has been shown to elicit the release of a number of metabolic hormones including insulin, glucagon, growth hormone, and prolactin. Recent evidence also indicates that arginine can stimulate the immune response (Barbul et al., 1981). LeFebvre et al. (1976) showed that arginine stimulated the release of glucagon and gastrin from the stomach of the dog. The importance of physiological concentrations of arginine in maintaining body homeostasis needs further investigation.

Using purified L-amino acid diets, arginine has been shown to be an essential amino acid for the immature and mature dog (Ha et al., 1978; Burns et al., 1981). Consumption of a diet devoid of arginine resulted in signs of ammonia toxicity within 1 hour following voluntary consumption of a single meal. Signs of ammonia intoxication included emesis, frothing at the mouth, muscle spasms, and altered cellular metabolism. Consumption of an arginine-deficient diet was accompanied by an elevation in the concentration of the pyrimidine metabolite, orotic acid, in both blood and urine. These studies revealed that 0.56 percent dietary arginine supported optimum growth, nitrogen balance, and prevention of orotic aciduria in the immature Beagle fed an L-amino acid diet equivalent to 14 percent protein. Czarnecki and Baker (1984) reported that the dietary arginine requirement for English Pointer puppies was 0.40 percent for maximum weight gain. Consumption of equimolar quantities of ornithine failed to prevent signs of arginine deficiency, which included growth failure, emesis, hyperammonemia, and orotic aciduria. Consumption of equimolar quantities of citrulline, however, resulted in near-normal growth, but blood and urine metabolites did not parallel those in puppies fed arginine. Although the dietary arginine requirement in the immature dog appears to increase with increasing nitrogen concentration of the diet (Ha et al., 1978), the recommendations have been set at 274 mg per kilogram of body weight per day to allow for optimum growth and prevention of abnormal metabolism. This corresponds to 1.37 g/1,000 kcal ME.

Signs of ammonia intoxication in mature Pointers were prevented by feeding a purified diet containing 0.28 percent arginine (Burns et al., 1981). In the absence of other data, 21 mg per kilogram of body weight per day has been proposed as the requirement for mature dogs. These results are consistent with the concept that the mature dog has a lower dietary amino acid requirement than that of the immature dog.

Histidine Diets containing 0.11 percent or less histidine and the equivalent of 14 percent protein resulted in depressions in food intake. Analysis of growth, feed efficiency, and nitrogen retention data of Beagles fed purified L-amino acid diets revealed that 0.21 percent histidine (193 mg/d/$kg^{0.75}$) was required to meet these parameters (Burns and Milner, 1982). Therefore, the requirement for immature dogs has been given as 98 mg per kilogram of body weight per day or 0.49 g/1,000 kcal ME of dietary energy intake.

Although Rose and Rice (1939) reported that histidine was an essential amino acid for the adult dog, no data were presented. Recently Cianciaruso et al. (1981) confirmed that histidine was required in the diet of adult female dogs. Mature dogs tube-fed a purified L-amino acid diet devoid of histidine for approximately 60 days had reduced plasma and muscle histidine concentrations, decreased muscle carnosine, reduced hematocrit, depressed serum albumin, and weight loss. The minimum requirement for mature dogs has not been established but has been estimated to be 22.0 mg/kg/d by Ward (1975).

Cianciaruso et al. (1981) reported that signs of histidine deficiency may not appear for many days, but then progress rapidly. Factors that modify the onset of signs of histidine deficiency may include the release of histidine from carnosine (β-alanyl-L-histidine) and hemoglobin, a reduction in the rate of histidine degradation, and possibly enhanced histidine reabsorption in the kidney (Cianciaruso et al., 1981).

Leucine, Isoleucine, and Valine Leucine, isoleucine, and valine are classified as branched-chain amino acids. Plasma concentrations of branched-chain amino acids have been observed to increase during prolonged fasting in the dog (Brady et al., 1977). Since muscle is a site of metabolism of the branched-chain amino acids, this increase may represent decreased muscular uptake or increased muscular release of these amino acids. Branched-chain amino acids are not only substrates for protein biosynthesis, but have also been implicated in the regulation of protein turnover and energy metabolism (Adibi, 1980). Purified L-amino acid diets containing 0.41 percent isoleucine support optimum growth in immature Beagles (Milner, 1979b). Furthermore, similar studies have shown that diets containing more than

0.55 percent leucine and 0.42 percent valine are required to optimize growth and nitrogen balance in immature dogs (Milner, 1979a). Since probably all branched-chain amino acids in the dog are metabolized by a common enzyme, it is possible that an excess of one of these amino acids will increase the dietary requirements for the others. Burns et al. (1984) have recently reported that 10- to 12-week-old Beagles require 0.65 percent leucine, 0.40 percent isoleucine, and 0.43 percent valine to support optimal growth, feed efficiency, and nitrogen retention. These values and proposed recommendations correspond to 159 mg leucine, 98 mg isoleucine, and 105 mg valine per 1,000 kcal dietary metabolizable energy. Ward (1975) has estimated that the leucine, isoleucine, and valine requirements for the mature dog are 84, 48, and 60 mg/kg/d, respectively.

Lysine Milner (1979a, 1981) has shown that lysine is an essential amino acid and that the dietary requirement for maximum growth and nitrogen balance occurred in immature male Beagles fed 0.58 percent or more lysine. In the absence of other data, 280 mg per kilogram of body weight per day has been set as the lysine recommendation for growth. This requirement would be met by a diet supplying 1.40 g lysine per 1,000 kcal ME. Dogs given diets containing excess lysine (1.73 percent) had significantly lower growth rates than dogs given diets containing optimal (0.58 percent) lysine. Ward (1975) estimated the minimum lysine required to maintain nitrogen equilibrium in the adult dog is approximately 50 mg/kg/d.

Methionine Methionine was established as an essential amino acid for optimum growth and nitrogen balance in immature Beagle dogs using diets containing purified L-amino acids (Milner, 1979b; Burns and Milner, 1981; Blaza et al., 1982). These studies revealed that diets containing 4 kcal ME/g and 0.20 percent methionine and 0.15 percent cystine meet the total sulfur amino acid requirement of the immature Beagle based on growth and nitrogen balance (Burns and Milner, 1981). Expressing methionine and cystine on an isosulfurous basis, the total dietary sulfur amino acid requirement for immature Beagles fed a 16 percent L-amino acid diet was found to be 0.39 percent. The total sulfur amino acid requirement was estimated to be 373 mg/d/kg$^{0.75}$ in diets containing an estimated 4.4 kcal gross energy per gram dry weight (Burns and Milner, 1981). Support for this requirement for methionine comes from studies showing intake of diets containing more than 12 percent protein as casein did not improve the growth of the immature dog (Burns et al., 1982). Cystine could supply approximately 50 percent of this requirement. These studies also demonstrated that the dog can effectively utilize D-methionine, DL-methionine, OH-L-methionine, *N*-acetyl-L-methionine, but not *N*-acetyl-D-methionine, to replace dietary L-methionine (Burns and Milner, 1981).

Hirakawa and Baker (1984) found that the dietary sulfur amino acid requirement of English Pointer puppies for maximal growth rate and efficiency of weight gain was approximately 0.45 percent. In dogs fed diets containing limited methionine, excess cystine caused anorexia, growth depression, and severe skin lesions on the neck, tail, and foot pads.

Blaza et al. (1982) studied the sulfur amino acid requirements of growing Labrador and Beagle dogs in three experiments. In two of these experiments the diets were based on isolated soy protein supplemented with methionine, while in the third experiment a crystalline amino acid diet was used. For the isolated soy protein diet supplemented with 0.15 percent cystine, the addition of 0.39 percent methionine gave significantly lower body weight gains, nitrogen retention, and food intakes than 0.57 or 0.74 percent methionine. Intakes of 116 mg total sulfur amino acids (TSAA)/100 kcal ME were inadequate, but 154 mg TSAA/100 kcal ME appeared adequate for Labrador dogs. These studies indicated that the dog's breed may influence methionine requirements, since Labradors but not Beagles responded to increasing the methionine content from 0.36 to 0.71 percent by increased weight gains and food intakes. These data also indicate that methionine available from isolated soy preparations must be considered in evaluating the adequacy of diets.

Estimates of minimal total sulfur amino acid requirements for normal growth have ranged from 0.39 to 0.71 percent in diets of comparable caloric value. The minimal recommendation has been set at 1.06 g/1,000 kcal ME.

Methionine is often considered a limiting amino acid in many normal protein sources. Methionine supplementation is known to reduce the quantity of these protein sources required for adult dogs (Allison et al., 1947). Kade et al. (1948) reported that a daily intake of 140 mg nitrogen from casein per kilogram of body weight was adequate to support nitrogen balance in adult dogs. The quantity of nitrogen could be reduced to 90 mg per kilogram of body weight when the casein was supplemented with methionine. Arnold and Schad (1954) reported that the addition of 1 g DL-methionine to 100 g casein protein reduced the quantity of nitrogen required for nitrogen equilibrium from 139 to 102 mg per kilogram of body weight. Addition of 3 g DL-methionine reduced the quantity of nitrogen required for equilibrium to 72 mg per kilogram of body weight. These authors concluded that the sulfur amino acid requirement of the mature dog was approximately 30 mg

per kilogram of body weight per day when the sulfur amino acid component contained about 89 percent methionine and 11 percent cystine. This quantity is lower than the minimum of 43 mg/kg/d reported by Ward (1975) to maintain nitrogen equilibrium in 15-kg adult dogs and has been considered the requirement. Similar to rats, pigs, and rabbits, the mature dog (Cho et al., 1980) apparently utilizes D-methionine as efficiently as L-methionine.

Phenylalanine and Tyrosine Phenylalanine is a dietary essential for the immature dog (Milner, 1979a). Dietary phenylalanine requirements have been reported to be less than 0.58 percent when 0.35 percent tyrosine was present in the diet (Milner, 1979a). Deletion of tyrosine from purified L-amino acid diets did not significantly influence growth when excess phenylalanine (1.16 percent) was present in the diet. The percentage of the phenylalanine requirement that can be met by tyrosine remains to be determined, but it is probably similar to the 50 percent observed in other mammals. Based on data with other species, the recommendations are for 1.95 g total aromatic amino acids per 1,000 kcal ME, of which 50 percent can be supplied as tyrosine. Ward (1975) suggested that the phenylalanine requirement for maintenance of nitrogen equilibrium in the mature dog is 51 mg/kg/d or 85.8 mg/kg/d of phenylalanine and tyrosine, and this has been taken as the requirement.

Threonine The threonine requirement of the immature dog has been investigated by Milner (1979b) and Burns and Milner (1982). Studies utilizing purified L-amino acid diets containing the equivalent of 14 percent protein have revealed that 0.52 percent threonine meets the requirement for optimal growth, feed efficiency, and nitrogen retention. This intake is equivalent to 254 mg per kilogram of body weight per day, which is proposed as the requirement (1.27 g/1,000 kcal ME). Signs of neurological dysfunction and/or lameness have been reported in kittens fed a threonine-deficient or imbalanced diet (Titchenal et al., 1980). However, these signs have not been reported in dogs. The estimate of Ward (1975), that the threonine requirement to maintain nitrogen balance of the mature dog is 44 mg/kg/d, was chosen as the requirement.

Tryptophan The dietary tryptophan requirement of the immature dog has been met by supplying 145 mg/d/$kg^{0.75}$ or by supplying 0.17 percent dietary tryptophan (Burns and Milner, 1982). Czarnecki and Baker (1982) reported that the tryptophan requirement for optimal growth of weanling English Pointers 6 to 10 weeks old was at least 0.16 percent, between 0.12 and 0.16 percent for 10- to 12-week-old puppies, and ≧ 0.12 percent for 12- to 14-week old puppies. Their studies also revealed that D-tryptophan utilization was 36 ± 6 percent (X ± SD) that of L-tryptophan. Based on the above studies, the requirement for tryptophan has been placed at 82 mg per kilogram of body weight per day or 0.41 g/1,000 kcal ME of dietary energy.

The metabolism of D- and L-tryptophan by mature dogs was compared by Triebwasser et al. (1976). Kynurenic acid was shown to be a major urinary metabolite of L-tryptophan (Brown and Price, 1956; Triebwasser et al., 1976). However, unchanged D-tryptophan, D-kynurenine, and kynurenic acid were the major metabolites of D-tryptophan. The inversion of D-tryptophan to L-tryptophan via indolepyruvic acid appeared to be one of the major fates of ingested D-tryptophan in the mature dog. The minimum quantity of tryptophan that is required to maintain nitrogen equilibrium in adult dogs was estimated by Ward (1975) to be 13 mg per kilogram of body weight per day, and has been set as the daily requirement.

Protein Digestibility

The digestibility of some sources of protein has been evaluated in the dog. Hegsted et al. (1947) found that the apparent digestibility of proteins in an all-vegetable diet containing white bread, corn, rice, potatoes, lettuce, carrots, onions, tomatoes, and applesauce was 80.0 ± 7.7 percent (X ± SD). James and McCay (1950) reported that the apparent protein digestibility of commercial, dry-type food, containing both vegetable and animal proteins, ranged from 67 to 82 percent for adult dogs. Kendall and Holme (1982) reported the apparent crude protein (N × 6.25) digestibility coefficients for textured soy protein, extracted soy meal, full-fat soy flour, and micronized whole soybeans ranged from 71 to 87 percent. Moore et al. (1980) reported apparent digestibility values of soybean meal, corn, rice, and oats by mature Pointers to be in the range of 77 to 88 percent. Their data revealed that normal cooking procedures did not significantly influence the digestibility of rice, oat, or corn protein. Their data also indicated that increasing the fat content of the diet from 10 to 20 percent did not alter the digestibility of nitrogen in a corn-soybean-based diet. Studies of Burns et al. (1982) have shown that the apparent digestibilities of lactalbumin, casein, soy protein, and wheat gluten are 87, 85, 78, and 77 percent, respectively. The digestibilities of these proteins were 5 to 10 percent greater in the immature rat than observed in the immature dog.

Dispensable Amino Acids And Total Nitrogen

Requirements for Growth Voit (1881) recognized that different minima exist with respect to meeting dietary protein needs. Cathcart (1921) noted "that the search for an absolute minimum is like the search of the philosopher for absolute truth. There is not one minimum but many protein minima—[each] a resultant of many factors."

The percentage dietary protein needed should be related to the energy content of the diet (Allison, 1951). Campbell and Phillips (1953) reported that adding fat to a 19.7 percent protein diet inhibited the growth of growing puppies. Normal growth resumed when 0.3 percent methionine was added to the diet. Ontko et al. (1957) estimated the dietary protein requirements of growing Beagle, Shepherd, and Shepherd-Collie puppies fed a dry diet containing 20 or 30 percent fat supplied primarily as lard (4.02 or 4.57 kcal ME/g). They concluded on the basis of weight gain, feed efficiency, and physical condition that 25.0 percent protein was required in diets containing 20 percent fat and that 28.9 percent was required if the diet contained 30 percent fat.

Estimates of the protein requirements for growth of puppies have been reported by Heiman (1947), Gessert and Phillips (1956), Ontko et al. (1957), and Burns et al. (1982). Heiman (1947) examined the protein requirement of immature Cocker Spaniels by varying the proportion of a protein mixture containing 15 percent fish meal, 46 percent meat meal, and 39 percent soybean meal with a carbohydrate mixture containing cooked, flaked cereals. Based upon weight gain and appearance, it was concluded that 15 percent dietary protein on an air-dry basis was inadequate, but 20 percent was sufficient. Using English Setter puppies from 9 to 23 weeks of age, increasing dietary protein from 23 to 27 percent did not result in improved weight gain or appearance.

Gessert and Phillips (1956) fed 6- to 7-week-old Beagle and mongrel puppies a basal diet containing 10.6 crude protein (3.38 kcal ME/g) supplied as dried skim milk, alfalfa leaf meal, dried primary brewers' yeast, soybean meal, and yellow corn. The basal diet was supplemented with casein to obtain a final protein concentration of 12.8, 15.0, 17.2, or 19.4 percent. The basal diet was substantially improved by the addition of 2.5 percent casein (2.2 percent protein), but additional casein caused little further improvement.

The minimum percent of dietary protein required by the dog decreases with age after weaning. Data of Burns et al. (1982) indicated that the immature Beagle 8 to 10 weeks of age has a dietary protein requirement of approximately 15.0 percent when lactalbumin is the source of nitrogen and the metabolizable content of the diet is 4.2 kcal/g. However, the requirement was 11.7 percent when the Beagle puppy was 13 to 17 weeks of age. Expressed in grams of protein/d/kg$^{0.75}$, the requirement was estimated to be 14.0 and 9.3 for dogs 8 to 10 and 13 to 17 weeks old, respectively. These requirements are higher than the theoretical estimates of Miller and Payne (1963) and Payne (1965). Based on these studies, 9.5 percent protein from lactalbumin or an equivalent-quality protein source in diets containing 3.3 kcal ME/g should satisfy essential and indispensable amino acid requirements of the growing dog.

Requirements for Adult Maintenance Melnick and Cowgill (1937) studied adult dogs minimally depleted of their protein reserves and estimated that approximately 12 percent dietary protein from casein was required to maintain nitrogen equilibrium. Expressing the dietary requirement for lactalbumin, beef serum proteins, casein, and gliadin as a percentage of ME intake, values of 6.9, 8.6, 9.4, and 21.1 percent, respectively, were obtained. Kade et al. (1948) maintained nitrogen balance in adult dogs (5 to 14 kg) with 80 to 90 mg lactalbumin, 100 to 110 mg blood fibrin, or 130 to 140 mg casein per kilogram of body weight per day. These data suggest that on a dry matter basis a diet containing 6.5 percent casein or equivalent is adequate to maintain nitrogen equilibrium in the adult dog.

Protein reserves may be important in protecting the dog against a variety of stresses. Although 140 mg casein nitrogen per kilogram of body weight per day (6.5 percent of a dry-type diet) can sustain nitrogen equilibrium in a protein-depleted dog, susceptibility to the toxic effects of phosphosamides (used in cancer chemotherapy) and 2-Aminofluorene (a carcinogen) is greater than when the protein reserves are maintained by feeding 600 mg casein nitrogen per kilogram of body weight per day (16 percent dry-type diet) (Allison et al., 1954; McCoy et al., 1956).

Wannemacher and McCoy (1966) established that both 1-year-old and 12- to 13-year-old dogs can be maintained in nitrogen equilibrium with approximately 200 mg casein nitrogen per kilogram of body weight per day. Liver and muscle protein to DNA ratios reached maximal values in 1-year-olds fed 400 mg casein nitrogen per kilogram of body weight per day, while 600 mg was required in 12- to 13-year-old dogs. These results, and a significantly lower rate of incorporation of leucine into liver and muscle protein of older dogs, suggest that age may be associated with less efficient cellular protein anabolism. These studies suggest that approximately 6 percent protein as casein in a diet containing 3.5 to 4.0 kcal ME per gram should meet the needs of the older dog

for nitrogen equilibrium, but that higher concentrations may be needed to support optimum cellular integrity. Dietary protein requirements of approximately 6.0 percent are consistent with theoretical estimates made by Payne (1965).

Considerable debate remains regarding the interpretation of a value for protein requirement for nitrogen equilibrium and the potential benefits of higher intakes to maintain tissue protein stores. Kendall et al. (1982) reported that adult dogs (1 to 7 years old) ranging in body weight from 2.8 to 51.0 kg had a minimum metabolizable dietary protein (N × 6.25) requirement of 1.7 $g/kg^{0.75}$ body weight to replenish a mean daily endogenous nitrogen loss of 273 $mg/kg^{0.75}$. Additional data are needed to determine if consumption at that quantity would maintain an acceptable steady state in the adult dog.

Requirements for Reproduction and Lactation The protein and amino acid needs for reproduction and lactation have not been well defined. Additional research is needed to determine if the amino acid requirements for reproduction and lactation may exceed those for growth.

Ontko and Phillips (1958) examined the reproductive performance of mature Beagle and Cocker Spaniel bitches fed a semipurified diet containing 20 percent casein for 2.5 years. Supplementation with 10 percent fresh liver, 5 percent alcohol-extracted or untreated casein, 2 percent liver extract, or 5 percent autoclaved egg white improved the vigor of the newborn pups and reduced postnatal mortality to between 22 and 35 percent. Unfortunately in this study, a maximum of only five litters were born to any treatment, and the mortality was 62 percent in pups from bitches fed the basal diet.

Studies by Visek et al. (1976) examined the reproductive performances of Beagle bitches fed diets containing 25 to 26 percent crude protein from corn, soybean meal, meat and bone meal, fish meal, and dried skim milk with or without the addition of dried brewers' grains and yeast. Twelve of the 15 dogs maintained their nitrogen equilibrium and weight during these studies. The percent of puppies born alive in all treatments was approximately 85.

Requirements for Muscular Activity Protein requirements of working dogs have not been thoroughly evaluated. Kronfeld et al. (1977) published information indicating that consumption of a high-protein (52.8 percent), high-fat (36.7 percent) diet may offer advantages in physical performance in sled dogs compared to diets containing a high level of carbohydrates. Whether the improvements were associated with increased protein consumption or with conditioning the dogs to metabolize greater quantities of fat remains to be determined. It is clear that these dogs did acclimate to the high fat and protein diet without any apparent ill consequences. These dogs had a higher oxygen capacity and higher serum concentrations of calcium, magnesium, and albumin. Research with other species suggests that if the energy requirements are met, protein needs are not significantly greater than those for maintenance. Hard work increases caloric expenditure, reducing food intake as a consequence of fatigue (Orr, 1966). In order to encourage adequate food intake of hardworking dogs, it may be necessary to increase the palatability of the diet by adding fat and protein. It is not known whether other types of stressful conditions increase the demand for dietary protein.

MINERALS

It is common practice to include all the minerals shown to be required by other mammals in the formulation of dog diets, even though the quantitative requirements for all minerals have not been established experimentally for this species. The mineral concentrations used in dog diets are generally based on estimates extrapolated from the requirements of other species; from data obtained from studies that involve dogs and that, although not designed to establish nutrient requirements, nevertheless yielded nutritional information; or from experience with diets that have resulted in acceptable performances in dogs.

Limited controlled published data on quantitative mineral requirements of dogs is not the only complication in making an estimate of mineral requirements for dogs. The interactions between dietary mineral concentrations, availability of minerals in different compounds, and the breed of dog involved are factors that may modify individual mineral requirements. Many of the papers used as the basis for estimating the mineral requirements for dogs only contain information regarding the concentration of the mineral of interest and not a complete dietary mineral profile, which precludes the evaluation of possible interactions. In some cases the compound containing the mineral of interest is not identified, so the biological availability of the mineral cannot be determined, nor is the breed of dog or the type of diet involved in the study always stated. Perhaps the factor of most concern regarding the controlled data available for estimating the mineral requirements for dogs is that a large percentage of the research was conducted two or three decades ago. Differences in feed

manufacturing procedures or use of different ingredients may have an effect on the availability of some minerals.

Since publication of the last revision (NRC, 1974) of this report, data have not been published that would substantially alter the estimated mineral requirements of dogs. In order to be consistent throughout the present report, however, minimum mineral requirements are expressed without compensating for factors that influence mineral availability. The estimated requirements for most minerals are based on the lowest concentrations in purified diets that have resulted in acceptable performance. The requirements for most minerals in previous revisions of this report have been based on concentrations in natural ingredient diets, which are generally higher than concentrations used in purified diets.

Much work has recently been reported involving the effects of various minerals on different isolated tissues from dogs. These data provide a considerable amount of information on the role of minerals in the metabolic process, but they do not provide any information about the quantitative requirements of the intact dog. The toxic effects of feeding high concentrations of individual minerals to dogs have received some attention during the last several years. A detailed discussion of these results is beyond the scope of this report. However, results of these studies have been incorporated in a recent report, *Mineral Tolerance of Domestic Animals* (NRC, 1980).

The differences in mineral requirements for gestation, lactation, maintenance, and muscular effort have not been defined, although the dietary mineral requirements probably change with the stage of the life cycle or activity. As energy intake increases in relation to the extra demands of milk production or exercise, daily intake of minerals would be expected to increase. Therefore, the estimated mineral requirements in this report are estimated to meet the requirements of the entire life cycle of the dog.

The ash fraction represents total mineral content of dog diets. Generally, 4 to 5 percent of the dry matter as ash provides adequate amounts of minerals to meet the requirements for dogs. Frequently the ash concentration of commercial dog diets is somewhat higher (7 to 9 percent of the dry matter) because of the unavoidably high ash content of some ingredients commonly used in the formulation of dog foods. There is no apparent relationship between excess dietary ash and clinical signs of disease in dogs. High dietary ash may, however, indicate a compromise in diet quality.

Calcium and Phosphorus

Requirements

Calcium and phosphorus are considered together because of their close metabolic association. The ratio of dietary calcium and phosphorus may be as important for good nutrition but is of lesser significance than the absolute concentration of these minerals. A calcium: phosphorus ratio of 1.2:1 to 1.4:1 (by weight) in dog diets is generally considered optimal for maximum utilization. An optimal calcium: phosphorus ratio also minimizes the vitamin D requirement. The availability of calcium and phosphorus is a major factor affecting the dietary requirements of these elements (Schedle et al., 1968). Diets high in phytates or low in vitamin D adversely influence calcium absorption (Mellanby, 1920; Hoff-Jørgensen, 1946). However, vitamin D supplementation of diets low in calcium can cause pathological fractures, lameness, abnormal stance, and loss of skeletal density (Campbell, 1962).

Morgan (1934) reported that diets supplying about 0.50 percent calcium and 0.65 percent phosphorus permitted normal bone development in some dogs given adequate vitamin D. Others, mainly larger breeds, developed signs of mild rickets when the calcium intakes were between 100 and 175 mg per kilogram of body weight per day. Retention ranged between 42 and 120 mg per kilogram of body weight per day. This was lower than reported by Hoff-Jørgensen (1946), who fed diets higher in calcium to dogs weighing 0.9 to 5.3 kg. The latter retention rates of 200 to 300 mg/d are in good agreement with those obtained by Udall and McCay (1953) with young Beagles fed fresh bone.

Hoff-Jørgensen (1946) supplied each of two 30-day-old puppies with 1 g of calcium and 1 g of phosphorus daily. The amount of calcium and phosphorus retained averaged between 0.2 and 0.3 g daily through the first 200 days of age, despite an approximate sixfold increase in body weight. Retention tended to be slightly higher during the third and fourth months than at other times in the growth period. The average retention of calcium observed during the experiment was about 75 mg per kilogram of body weight per day (maximum 160 mg/kg/d). Addition of phytic acid to the diet decreased calcium absorption and retention but increased absorption and retention of phosphorus. Hoff-Jørgensen postulated that phystate caused the precipitation of calcium in the intestinal lumen as insoluble calcium phytate.

The utilization of calcium has been reported to vary from 50 to 80 percent (Morgan, 1934; McCay, 1949). Since the intestine normally controls the body status, excess calcium is excreted primarily in the feces. Jenkins

and Phillips (1960b) and Henrikson (1968) found that growing puppies required approximately 0.60 percent calcium in the diet for normal growth and for mineralization of the skeleton. Increasing the dietary fat from 2 to 3 percent did not influence the calcium requirement.

In a study of "overnutrition" and skeletal disease, Hedhammer et al. (1974) fed a diet to Great Dane puppies containing, on a dry basis, 36 percent protein, 14 percent fat, 40 percent carbohydrate, and 10 percent ash. Significant chondro-osseous changes, reflected in lameness and pain upon palpation of the skeleton, enlargement of the costochondral junctions and the epiphyseal-metaphyseal regions of long bones, hyperextension of the carpus, and sinking of the metacarpo- and metatarso-phalangeal joints were observed. Excessive intake of various nutrients may have led to these complications, since the diet contained (on a dry basis) 2.05 percent calcium, 1.44 percent phosphorus, 0.27 percent magnesium, and 4,000 IU vitamin D per kilogram—all appreciably in excess of presumed requirements.

In a study of the calcium:phosphorus ratio in relation to periodontal diseases, Henrikson (1968) fed adult Beagles a purified diet containing 0.12 percent calcium and 1.20 percent phosphorus. The progressive loss of alveolar bone was so severe that by 12 months the incisor teeth became easily detached. Histopathological examination revealed progressive parathyroid changes associated with hyperfunction. Such changes were not observed in control groups fed 0.54 percent calcium and 0.42 percent phosphorus.

The retention of phosphorus has been reported to range from 12 to 43 percent (average 23 percent) (Morgan, 1934; Hoff-Jørgensen, 1946). Jenkins and Phillips (1960a) found that a diet containing 0.33 percent dietary phosphorus provided the same amount of growth as a diet containing 0.53 percent phosphorus. Retention was 76 percent, which indicates a minimum requirement of 0.25 percent for available phosphorus. About 45 percent of the phosphorus was present as phytic phosphorus, and the calcium content was 0.60 percent. The phosphorus requirement increased by 10 to 15 percent when the calcium was increased to 0.9 or 1.2 percent. Increasing the dietary fat from 3 to 20 percent increased the phosphorus requirement about 20 percent. These observations indicate that the requirement for phosphorus would be expected to be met if the diet contained 0.5 percent of total phosphorus from other than plant sources, provided there was a desirable calcium:phosphorus ratio, and availability was 50 percent or greater.

Dogs of some types and breeds may perform satisfactorily on lower intakes of these minerals. Gershoff et al. (1958) maintained two dogs for 34 months on a purified diet that was only 0.11 percent calcium from the time they were 2 to 3 months of age. Compared to littermates fed a 0.63 percent or 1.23 percent calcium diet, no differences in fat-free bone ash or in growth rates were observed. The dietary calcium on the 0.11 percent calcium diet was 90 percent utilized compared to utilizations of 46 and 27 percent, respectively, when 0.63 or 1.23 percent calcium diets were fed. Under practical conditions, 90 percent utilization of calcium would not be expected. Gershoff et al. (1958) did not report analyses of phosphorus, but calculations based on published values show that the calcium:phosphorus ratio was about 0.2:1 on the lowest level. The authors concluded that the animals adapted to this diet. However, in view of Henrikson's (1968) studies with dogs beginning when the dogs were 1 year old, it seems probable that changes in mandibular bone would have resulted from the 0.11 percent calcium diet if it was fed over an extended period of time. Krook et al. (1971) have confirmed Henrikson's findings. Repletion with adequate calcium begins in the *laminae dura dentes* and is followed by the vertebrae and the long bones.

The salt mixture formulated by Phillips and Hart (1935) (calcium:phosphorus ratio of approximately 1.9:1.0) has been used to provide the minerals for numerous purified experimental dog diets without reported ill effects. Diets would contain about 0.5 percent calcium when this salt mixture is 4 percent of the total diet.

It is recognized that there are many breeds of dogs, that they are maintained under a wide range of environments, and are being fed a variety of foods of animal and plant origin. There also remain unknown factors that may adversely influence the utilization of minerals, including calcium and phosphorus in many natural ingredient diets. However, based on existing experimental data, it is proposed that diets containing 1.6 g calcium and 1.2 g phosphorus per 1,000 kcal ME will meet the minimum requirements of normal dogs.

Signs Of Deficiency And Imbalance

Adequate calcium and phosphorus nutrition depends on an adequate supply of available calcium and phosphorus, a suitable calcium-phosphorus ratio, and adequate vitamin D.

In dogs calcium deficiency is associated with progressive parathyroid hyperplasia (nutritional secondary hyperparathyroidism). The rate of bone loss and osteoporosis depends on the skeletal region involved. Jawbones show earliest signs, followed by other skull bones, ribs, vertebrae, and finally the long bones. Loss of calcium from the jawbones can lead to recession of alveolar bone and gingiva. Detachment of the teeth and other signs of deficiency may appear before compression

of vertebrae and fractures of long bone. With rather severe calcium deficiency, the morphologic picture is characterized by excessive bone resorption, whereas defective mineralization associated with the osteodystrophy of rickets is not readily observed except in the growing puppy.

Calcium deficiencies may result in tetany and convulsions, reproductive failures, spontaneous fractures, and altered requirements for other nutrients such as magnesium.

An uncomplicated deficiency of phosphorus seldom occurs in dogs except under experimental conditions. In young dogs, low phosphorus intake will lead to rickets, poor growth, and a depraved appetite. In adults, low phosphorus intake leads to osteomalacia. Excessive intake of phosphorus relative to calcium leads to signs of calcium deficiency.

Potassium

The potassium concentration in commercial dry dog foods typically varies between 0.70 and 0.85 percent. Ruegamer et al. (1946) fed purified low-potassium diets (5 kcal ME/g) to dogs and produced poor growth, restlessness, and paralysis of the neck and the forepart of the body. Administration of a single 3-g dose of potassium chloride by capsule and inclusion of the salt in the diet at a 0.6 percent level relieved these conditions and permitted normal growth. This amount, equivalent to that obtained from a diet containing 0.32 percent potassium, provided about 70 mg of potassium per kilogram of body weight daily when the diet was fed at the rate of 22 g per kilogram of body weight.

Serrano et al. (1964) found that feeding low-potassium diets to pregnant bitches did not affect litter size or birth weight of the puppies, although the bitches had reduced concentrations of blood potassium. In contrast to their dams, the puppies had normal blood and muscle electrolyte concentrations.

Dogs can be severely depleted of potassium in 30 days and repleted in 14 days (Abbrecht, 1972). An allowance for growth of 264 mg of potassium per kilogram of body weight per day is suggested as a minimum. This amount is considerably less than the 530 mg/kg provided by the salt mixture formulated by Phillips and Hart (1935), but that mixture was intended to provide generous amounts, and no data regarding potassium requirements were available when it was formulated. It is estimated that the potassium requirements of normal dogs will be met by providing a concentration of 1.2 g potassium per 1,000 kcal dietary metabolizable energy.

Signs of deficiency are poor growth, restlessness, muscular paralysis, a tendency to dehydration, and lesions of the heart and kidney.

Sodium and Chlorine

The general practice is to include 1 percent sodium chloride in all dry dog diets, which provides approximately 95 mg sodium and 147 mg chlorine per kilogram of body weight per day. This amount is considered the appropriate allowance in that it meets the requirements and is not excessive for normal dogs, but the minimum requirement is greatly exceeded. Some natural feedstuffs may contain enough sodium and chlorine to meet minimum requirements, while various water supplies contain ample sodium.

In experiments with dogs fed a diet containing 2 percent added sodium chloride, McCay (1949) observed greater-than-normal water intake but normal health.

Dogs fed less than 23 mg sodium per kilogram of body weight per day showed changes in concentrations of blood-pressure-regulating hormones in 3 days, whereas dogs fed 80 mg/kg did not show these changes (Bunag et al., 1966; Ganong and Boreyzka, 1967; Brubacher and Vander, 1968). However, there were no other effects detected.

Morris et al. (1976) fed a diet containing 0.0075 percent sodium to adult dogs for 23 weeks and reported no adverse effects. These results were similar to those published by Hamlin et al. (1964), which also indicated that dogs have a low sodium requirement. Based on these results, it is estimated that 0.15 g of sodium per 1,000 kcal dietary metabolizable energy is adequate for dogs. In past revisions of *Nutrient Requirements of Dogs,* requirements for sodium and chloride were expressed in terms of a requirement for NaCl. In the absence of studies establishing chloride requirements for dogs, a value of 0.23 g/1,000 kcal ME, or 1.5 times the stated requirement for sodium, was used as the chloride requirement. This value is in keeping with the Na:Cl requirement ratio for other species.

Humans with inadequate sodium chloride intake become easily fatigued. McCance (1936) and McCance and Widdowson (1944) have observed similar fatigue in dogs and decreased utilization of protein in humans and dogs during prolonged sodium chloride deficiency.

Thus, signs of deficiency are fatigue, exhaustion, inability to maintain water balance, decreased water intake, retarded growth, dryness of skin, and loss of hair.

Magnesium

Magnesium has been shown to be a dietary essential for the dog (Orent et al., 1932). However, the published experimental results regarding the quantitative dietary requirements for this mineral are inconsistent. On the basis of results obtained from studies involving graded concentrations of dietary magnesium, Bunce et al.

(1962a,b) concluded that the magnesium requirement of weanling Beagle pups fed a purified diet was 140 mg/kg, while the requirement for mature dogs ranged from 80 to 180 mg/kg. They also reported that the severity of the magnesium deficiency syndrome was increased by elevation of the dietary calcium and phosphorus concentrations from 0.6 to 0.9 percent and 0.4 to 0.9 percent, respectively. Vitale et al. (1961) fed magnesium-deficient diets to dogs and observed a series of changes that did not occur in dogs fed the basal diet with 960 mg magnesium and 5,000 mg potassium added per kilogram diet. This high magnesium concentration was presumably used to ensure an adequate intake. Kahil et al. (1966) reported dogs fed diets containing 5 mg magnesium per kilogram developed convulsive seizures and alterations in sodium and potassium transport, whereas dogs receiving 16 mg magnesium as anhydrous magnesium oxide per kilogram of body weight per day did not have any clinical signs of deficiency. Morris (1963) reported that when weanling puppies were fed diets containing 30, 100, or 320 mg magnesium per kilogram, the calcium concentrations in the aorta were 8,320; 5,450; and 980 mg/kg (dry tissue), respectively, indicating an interaction in the absorption or retention of these minerals. Romsos et al. (1976) used magnesium oxide concentrations ranging from 0.025 to 0.035 percent (250 to 350 mg/kg) in experimental purified diets without observing any clinical signs of magnesium deficiency. Based on the foregoing data, the magnesium requirements for dogs should be met by dietary concentrations of 0.11 g/1,000 kcal ME.

Signs of Deficiency

Anorexia, vomiting, decreased weight gain, and hyperextension of the front legs were observed in puppies (7 to 9 weeks of age initially) that were fed a purified diet containing less than 5 mg/kg magnesium for 3 weeks (Kahil et al., 1966). By 4 to 6 weeks the puppies fed this diet showed irritability, ataxia of hind legs, convulsive seizures, and alterations in sodium and potassium transport. Similar deficiency signs were reported in puppies fed a magnesium-deficient diet containing 0.6 percent calcium and 0.5 percent phosphorus with 8 percent fat (Bunce et al., 1962a). These authors reported that the dogs' blood serum magnesium and calcium concentrations were depressed and that their inorganic phosphorus was elevated. At necropsy, aortas of these animals contained extreme mineralized lesions, primarily calcium and phosphorus deposits. They also reported that a much longer depletion period was required to demonstrate magnesium deficiency in mature dogs than in puppies. In mature dogs there was a loss in body weight and a depression in serum magnesium but no changes in serum calcium or phosphorus. Vitale et al. (1961) recorded electrocardiographic changes in puppies fed magnesium-deficient diets that were similar to those seen in hyperkalemia. Subsequent studies in 4- to 6-month-old dogs demonstrated a relationship between magnesium and potassium deficiencies. Hyperkalemia and marked electrocardiographic changes were recorded in two dogs that received a low magnesium diet for 9 months; these changes were similar to those observed in dogs deficient in both magnesium and potassium.

Iron

Both iron and copper are essential for preventing anemia. Most of the iron is in the respiratory pigments (hemoglobin and myoglobin) and in various enzymes. The characteristic anemia associated with an iron deficiency is of a hypochromic, microcytic type. However, hypochromic anemias may also occur when the total iron content of the body is normal, indicating that factors other than total body iron are also involved (Moore, 1963). Usually, 5 to 10 percent of the oral iron intake is absorbed (Stewart and Gambino, 1961; Talwar et al., 1961; Pollack et al., 1963, 1964). However, many factors influence absorption, including the chemical form of the iron (Brown, 1963; Fritz et al., 1970), associated food proteins (Fitch et al., 1964), mineral balance of the diet, hormone balance (Cline and Berlin, 1963), freedom from intestinal abscesses (Hahn et al., 1946), vitamin stores, severity of anemia (Koepke and Stewart, 1964a,b), and diurnal variations (Goldstone et al., 1962). The gastric juice from anemic dogs contains a substance that increases the absorption of iron from the gastrointestinal tract. When the gastric juices from anemic dogs and iron were given to normal dogs, the absorption of iron was significantly increased (Koepke and Stewart, 1964a,b; Arriaga de la Cabada et al., 1969).

The iron of wheat bran has been shown to be as available for dogs as that of ferric pyrophosphate, but that of spinach is less than half as available (Frost et al., 1940). These findings conform with the relative availability of the iron in those three sources when fed to rats and suggest that availability for the rat may be used as a guide for dogs. Elvehjem et al. (1933, 1934) and Sherman et al. (1934) have shown the iron of inorganic salts, liver, heart, muscle, and soybeans to be readily available (50 percent or more utilized), while the utilization from oysters, alfalfa, spinach, blood, wheat, oats, and yeast was lower (25 percent utilized). Dogs utilize iron from porphyrin compounds, such as hemoglobin and myoglobin (Udall and McCay, 1953; Bannerman, 1965). There are variations in the efficiency with which various species utilize iron from iron-containing salts. Ferric ammonium

citrate and ferrous sulfate are highly effective for preventing anemia in a number of species (Wintrobe, 1967; Fritz et al., 1970).

Ruegamer et al. (1946) maintained normal hemoglobin in Collie puppies that received 3 mg iron as ferric pyrophosphate per kilogram of body weight per day. Other puppies, made anemic by an iron-free diet, did not recover when 0.4 mg ferric pyrophosphate per kilogram of body weight was supplied daily, but they did recover when the supplement was increased to 0.6 mg/kg (0.2 mg iron). When the supplement was increased to 1 mg, more iron was absorbed and utilized, but the percentage of utilization dropped from about 60 percent (0.6 mg per kilogram of body weight level) to about 36 percent (1 mg per kilogram of body weight level). Frost et al. (1940) also obtained 60 to 70 percent utilization of inorganic iron supplements and indicated that absorption may sometimes approach 100 percent. With intakes of 0.6 mg per kilogram of body weight per day, normal values of 100 to 200 µg iron per 100 ml plasma were found. When the smaller quantities were fed, plasma iron concentrations decreased.

On the basis of this evidence, it would seem that 1.32 mg dietary iron per kilogram of body weight per day should meet the needs of puppies, adult dogs, or anemic dogs that are synthesizing hemoglobin. The intake required for regeneration of hemoglobin is less than 0.66 mg absorbable iron per kilogram of body weight (Ruegamer et al., 1946). If a large amount of the iron came from soluble inorganic salts, the allowance might be reduced, but reduction seems inadvisable in view of lack of information about the effect of other dietary constituents on iron absorption. McCance and Widdowson (1944) found that many substances (e.g., phosphates and phytates) depress utilization of dietary iron. Likewise, iron from insoluble iron salts and certain slightly soluble sources is poorly utilized. The allowance suggested, 1.32 mg per kilogram of body weight per day, is slightly in excess of that provided by the widely used mineral mixture suggested by Phillips and Hart (1935). However, the reports of satisfactory nutrition in dogs fed the Phillips and Hart mixture have been based on refined diets rather than on mixtures of natural foodstuffs, which may contain interfering substances.

On the basis of the available experimental data, it is proposed that a concentration of 8.7 mg iron per 1,000 kcal dietary metabolizable energy will meet the minimum requirements of normal dogs.

Signs Of Deficiency

Iron is a part of the hemoglobin molecule and is essential for oxygen transport. Thus, iron-deficient dogs exhibit anemia and tissue anoxia. The mean corpuscular hemoglobin concentration and mean corpuscular volume are decreased, and the anemia may be characterized as microcytic and hypochromic. While not all hypochromic anemias are attributable to iron deficiency (Moore, 1963), serum iron of iron-deficient dogs will be depressed, and the erythropoietic system will respond quickly to iron-dextran administered orally, intramuscularly, or intraperitoneally.

Toxicity

Iron toxicity in dogs has been studied extensively (Cibis et al., 1957; Brown et al., 1959; Bronson and Sisson, 1960; D'Arcy and Howard, 1962a,b) and is associated with anorexia, weight loss, and decreased serum albumin concentration. Although some dogs have been fed as long as 18 months on diets containing 1 percent iron oxide, other iron salts have proved toxic at very low intakes (D'Arcy and Howard, 1962a). Ferrous sulfate administered orally produced gastrointestinal damage when fed in a dosage of 0.012 g per kilogram of body weight. Ferrous carbonate did not produce such changes at 1.5 g per kilogram of body weight, but did so at 3 g/kg. *Mineral Tolerance of Domestic Animals* (NRC, 1980) contains additional information regarding iron toxicity in dogs.

Copper

Copper has been shown to be a dietary essential for prevention of anemia in dogs. Linton (1934) and Frost et al. (1939) reported that during copper deficiency iron was absorbed, but hemoglobin was not formed efficiently. Hemoglobin regeneration in anemic dogs (13 kg) did not occur unless 2 mg copper per day were given. This amount of copper also met the requirement for growth.

Tinedt et al. (1979) reported a copper toxicosis in Bedlington terriers fed commercial dog diets containing 5 to 10 mg copper per kilogram of diet. Ludwig et al. (1980) studied this disease in considerable detail and concluded that it is unique to this breed of dog and is caused by a genetic abnormality. Keen et al. (1981) measured hepatic concentrations of various minerals in Beagles and reported that the concentration of copper did not change while the pups were between 8 and 193 days of age.

The copper requirement for the majority of dog breeds appears to be quite low. It is estimated that a minimum of 0.8 mg copper per 1,000 kcal ME will meet the requirements of normal dogs, provided other dietary mineral concentrations are not excessive.

Manganese

There is essentially no published information regarding the manganese requirements for dogs, nor is there any description of the deficiency signs in this species. However, this mineral is known to have a role in catalyzing several metabolic reactions in other mammalian species. It is common practice to include manganese in diets for dogs. Ingredients used in natural ingredient dog diets may contribute adequate amounts of manganese to meet requirements, and it may not be necessary to add manganese to this type of diet. Based on the requirements of other animal species, it is estimated that the minimum manganese requirement for dogs is 1.4 mg/1,000 kcal ME.

Zinc

Although zinc metabolism in dogs was studied in considerable detail as early as the 1920s (Drinker et al., 1927), few reports have been published regarding the zinc requirements of this species. A zinc deficiency was reported in dogs (breeds not indicated) by Robertson and Burns (1963) when 2 percent calcium carbonate was added to a diet containing 0.3 percent calcium and 33 mg zinc per kilogram. Differences in weight gain between animals fed this diet and those fed the diet with 200 mg zinc per kilogram from added carbonate were reported when the dogs were being studied for 3 months. After 10 months, dogs fed the diet with the lower zinc concentration only gained one-third as much weight as those fed the higher concentration. The zinc-deficiency syndrome was initially characterized by skin lesions that appeared on the abdomen and extremities, then by marked emaciation, emesis, conjunctivitis, keratitis, and general debility. On necropsy, there was gross evidence of fatty change in the liver, the gall bladder was distended, and there was evidence of kidney damage with calcium deposits in the renal pelvis. Recently, Sanecki et al. (1982) fed English Pointer pups a corn-soy-based zinc-deficient diet and reported observing within 5 weeks lesions of parakeratosis, mild hyperkeratosis, alterations in germinal epithelium, erosions, ulcerations, vesiculation, alopecia, and inflammation of the skin. These lesions were reversible by adding 200 mg zinc carbonate per kilogram to the diet, with complete remission of the external lesions in 6 weeks. These authors indicated that borderline zinc deficiencies could occur when less than 90 mg zinc per kilogram diet are included in high-calcium commercial dog foods made from natural ingredients.

Fisher (1977) fed more than 800 Beagles 32 mg/kg zinc of diet (calcium concentration not noted) and did not report any clinical signs of zinc deficiency. He reported serum zinc values decreased in dogs with hepatic disorders, hysterectomies, hypothyroidism, and infections, while there was no change in the serum zinc values due to renal disorders, castration, epilepsy, or enucleation.

Anderson and Danylchuk (1979) added 100 mg zinc oxide per kilogram of acidified drinking water and offered it to Beagles for 9 months. No pathological changes were observed. There was, however, an increase in serum zinc concentration. Baxter et al. (1970) compared radiolabeled zinc uptake by subcellular fractions in normal and infarcted myocardia in dogs. Increased radioactive zinc uptake in the infarctions suggested that zinc was mobilized for the tissue repair process.

Purified diets containing 78 percent moisture and 12 to 57 mg zinc per kilogram (as-is diet) were fed to dogs by Romsos et al. (1976) without any apparent effect on growth that could be related to zinc intake.

A concentration of 60 mg zinc per kilogram in dry natural-ingredient diets appears to meet the maintenance requirements for dogs provided calcium concentrations are not excessive. Excessive dietary calcium concentrations will decrease zinc availability, and various pathologically induced stresses will increase zinc requirements. Zinc requirements for dogs maintained on high-meat or purified diets may be slightly lower. There is no comparable experimental evidence showing the extent to which reproduction and growth influence the zinc requirements of dogs. However, optimal performance during gestation and lactation may be more readily attained when the dietary zinc concentration is increased to near 90 mg/kg (Sanecki et al., 1982), particularly when natural ingredient diets that contain high levels of phytate and calcium are involved. Based on studies with purified diets, it is estimated that the minimum zinc requirement for dogs is 9.7 mg/1,000 kcal ME. However, it is recognized that there are numerous dietary constituents that can influence zinc availability, and so for practical diets including natural feed ingredients, zinc levels approaching those suggested by Sanecki et al. (1982) or greater may be required.

Iodine

Marine and Lenhart (1909) showed that dogs require small amounts of dietary iodine for the prevention of goiter. Belshaw et al. (1975), using Beagle dogs 1 to 2 years of age and weighing 9 to 15 kg, estimated the minimum daily iodine requirement to be 140 µg per day. Serum T4 and T3 (triiodothyronine) levels were unaffected by reducing iodine intake to 90 µg per day. When iodine intake was reduced to 20 to 50 µg per day serum T4 was markedly reduced, but T3 was unaffected. Iodine

deficiency of 8 to 12 months resulted in variable patterns in thyroid histology, which related to the rate of release of radioiodine from the thyroid.

Thyroidal iodine uptake is influenced by the age of puppies (Book, 1976) and by the level of iodine in the diet (Fritz et al., 1970). The full cycle of thyroid gland accommodation to limited dietary iodine has been demonstrated in 11-month-old purebred Beagles (Norris et al., 1970). The uptake and release of ^{131}I by the thyroid gland were measured periodically for 651 days while dogs were fed a semisynthetic diet that provided 50 to 75 µg of iodine per day. During the first 268 days of restricted iodine intake, the thyroid glands became hyperplastic and hypertrophic. Hyperplasia and hypertrophy were correlated with a large increase in thyroidal uptake of test doses of ^{131}I and also with more rapid loss of ^{131}I from the gland after the point of maximum uptake. After 368 days of restricted iodine intake, the thyroid glands were involuted and had an essentially normal histological appearance. Thyroidal uptake of ^{131}I remained high, but the subsequent rate of loss of ^{131}I was drastically reduced. This correlated with the return of thyroglobulin to the gland. The proposed adequate dietary iodine concentration for normal dogs is 0.16 mg/1,000 kcal ME.

Signs Of Deficiency

Goiter is the main sign of iodine deficiency. Cretinism in dogs has been reported in localities where goiter is endemic. Myxedema appears in the skin, and skeletal deformities lead to a short, broad nose; coarse, heavy extremities, and a short body; and delayed shedding of deciduous teeth (Dammrich, 1963). Other signs of deficiency are hairlessness, dullness, apathy, drowsiness, and timidity. Excessive amounts of iodine may be toxic to dogs (Webster et al., 1966).

Selenium

The relationships between vitamin E and selenium requirements of many domestic animal species are well documented (NRC, 1983). The dietary selenium requirements of the dog, however, have not been studied in detail even though it has been reported (Fuller, 1971; Lannek and Lindberg, 1975; Van Vleet, 1980) that vitamin E or selenium administration may have a pharmacologic effect in treating a number of diseases of dogs.

Van Vleet (1975) fed two Beagle puppies a purified, Torula yeast-based diet that was deficient in vitamin E and selenium. Clinical signs of deficiency observed after the diet was consumed from 40 to 60 days included muscular weakness, subcutaneous edema, anorexia, depression, dyspnea, and eventual coma. Microscopic examination of tissues from these dogs showed extensive skeletal muscular degeneration, necrosis in the myocardium, and renal mineralization. Similar lesions were reported by Manktelow (1963) in dogs suspected to have selenium deficiency. Puppies fed the purified diet supplemented with 30 IU α-tocopherol per kilogram or 0.5 and 1.0 mg selenium per kilogram did not develop the clinical signs of deficiency (Van Vleet, 1975). At necropsy, mild skeletal myopathy was observed histologically in the dogs consuming the 0.5 mg/kg selenium but not in those fed the α-tocopherol or the 1 mg/kg dietary selenium.

Based on these results, it would appear that a dietary concentration of 0.03 mg selenium per 1,000 kcal ME meets the requirements for dogs consuming a diet with adequate vitamin E levels. A diet containing up to 0.5 mg/kg selenium may be appropriate if vitamin E concentrations are limited. On a practical basis, dogs consuming dry commercial dog foods would not become selenium-deficient because this mineral has a wide distribution in dog food ingredients (Allaway, 1973).

Fluorine

A minimum requirement for fluorine has not been established for dogs. Experimental evidence shows that more mineral is not deposited in the bones of Beagles when a low-calcium, high-phosphorus diet is supplemented with fluorine (Krook, 1969; Henrikson et al., 1970; Krook et al., 1971).

Fluorine, fed as sodium fluoride at 0.45 to 4.5 mg per kilogram of body weight per day, comparable with the quantity found in some drinking water, caused mottling of the tooth enamel during the period of calcification of permanent teeth in dogs (Biester et al., 1936).

Andreeva (1959) reported that the addition of fluorine at 20 mg per kilogram of body weight daily for 92 days to the diet of month-old pups altered serum calcium and inorganic and organic phosphorus concentrations significantly. Fluoride-chloride therapy has been reported to promote thicker trabeculae and callus formation following fractures in dogs (DeGubareff and Platt, 1969). Feeding 200 or 250 ppm of fluorine in a diet deficient in magnesium prevented aortic calcification normally found in magnesium deficiency (Bunce et al., 1962; Chiemchaisri and Phillips, 1963).

Elements Required at Trace Concentrations

A series of elements have been shown to be required by various animal species at concentrations in the microgram-per-kilogram range. An example is cobalt, which is a component of the vitamin B_{12} molecule; it is not required when adequate amounts of this vitamin are

provided. Other elements that may be required by dogs at trace concentrations include molybdenum, tin, silicon, nickel, vanadium, chromium, lead, and perhaps arsenic.

The dietary requirements of the dog for these elements have not been established. These minerals seem to be widely distributed in the ingredients used to manufacture natural ingredient dog diets, particularly at the low concentrations that appear to meet the requirements of other mammalian species. Therefore, it is highly unlikely that dogs consuming natural ingredient diets would become deficient in any of these trace minerals. Their concentrations may be of concern when dogs are to consume highly purified diets over relatively long periods.

VITAMINS

Certain vitamins have been recognized as essential nutrients for dogs for more than 60 years. Despite this long history, precise quantitative requirements have not been established for every vitamin. The recommendations made in Tables 1 and 2 are designed to provide levels that are reasonable based on research with dogs and other species and that have proven satisfactory in practice. The concentrations of most nutrients in Table 2 can be determined from the daily requirements in Table 1 by simple calculation, assuming that the 3-kg growing puppy requires 600 kcal ME per day and that the 10-kg adult dog requires 742 kcal ME for maintenance. Although the vitamin requirements for gestation, lactation, and muscular effort have not been well defined, these needs are generally related to energy intake. As energy intake increases in relation to the extra demands of milk production or exercise, daily intake of vitamins will also increase. The vitamin requirements in Table 2 should be adequate to meet the needs of the entire life cycle of the dog. Since several vitamins are rather unstable, and their destruction may be promoted by light, heat, oxidation, moisture, rancidity, or certain mineral elements, sufficient amounts should be provided to ensure that the recommended concentrations will be present when the diet is consumed. Just as important is the realization that markedly excessive intake of vitamins A and D may be harmful to dogs.

Vitamin A

The earliest studies attempting to separate the functions of vitamins A and D on bone growth were carried out in dogs more than 60 years ago (see Mellanby, 1957). The actual dietary requirement for vitamin A in dogs was investigated by Frohring (1935; 1937), who fed a vitamin A-deficient diet to Beagle puppies and calculated that 100 IU vitamin A per kilogram of body weight was lost from the liver each day during growth (3 IU = 1 µg of retinol equivalents). The minimum curative dose of vitamin A equivalents that effected a definite increase in weight was 200 IU (in the form of β-carotene) per kilogram of body weight per day. Crimm and Short (1937), using a similar vitamin A depletion technique, estimated that the minimal daily vitamin A requirement of adult dogs was 22 to 47 IU per kilogram of body weight, or between 8 and 16 µg/kg, which is the universal requirement among those species tested to date. Bradfield and Smith (1938) fed 200, 400, 1,000, or 2,000 IU vitamin A per day per kilogram body weight to growing puppies and measured weight gain and liver vitamin A concentration. To compare the vitamin A activity of carotene sources, other puppies received 200 IU vitamin A equivalents, as carotene in oil or from carrots, per kilogram of body weight per day. While increasing dietary intakes of vitamin A resulted in increases in the vitamin A levels in the liver, 200 IU were adequate to produce maximum gains and slight liver vitamin A storage. In these studies, cod liver oil and carotene appeared to be equally well utilized as sources of vitamin A activity, even though cod liver oil is not an ideal test substance because it contains a variable amount of vitamin D that may interfere with vitamin A absorption if excessive. The data confirmed the earlier observation of Turner (1934) that dietary carotene (from carrots) may be converted to vitamin A and stored in the liver of dogs.

There has never been published a careful assessment of the daily vitamin A requirement for dogs based on the liver storage of the vitamin while feeding an acceptable form of vitamin A esters under controlled dietary circumstances. However, based on available information, the daily vitamin A requirement would be met by 75 IU per kilogram of body weight for adult maintenance and 202 IU per kilogram of body weight for growing puppies. These amounts will be more than provided by a dietary concentration (dry basis) of 3,336 IU per kilogram or 1,112 µg per kilogram of a diet containing approximately 3,300 kcal ME. This is equivalent to 1,011 IU/1,000 kcal ME.

Signs Of Deficiency

Vitamin A deficiency in the dog was among the first of the vitamin deficiencies to be studied experimentally. It is seldom encountered clinically. Steenbock et al. (1921) reported that dogs deprived of fat-soluble vitamins developed an "ophthalmia." These and other workers (Stimson and Hedley, 1933; Crimm and Short, 1937; Mellanby, 1938; Russell and Morris, 1939; Singh

et al., 1965) have observed the following deficiency signs: anorexia, weight loss, ataxia, xerophthalmia, conjunctivitis, corneal opacity and ulceration, skin lesions, metaplasia of the bronchiolar epithelium, pneumonitis, and increased susceptibility to infection with associated changes in the blood leukocyte differential count. Faulty bone remodeling in the young dog resulting in overgrowth and stenosis of the neural foramina produced pressure degeneration of nerves and impaired nerve function. Mellanby (1938) established that such damage to the cochlear and vestibular divisions of the eighth cranial nerve, plus a serious labyrinthitis, may induce deafness. Similar damage may also affect function of the optic and trigeminal nerves, although this only has been observed after prolonged experimental deficiency.

Vitamin A supplements (10,000 IU per day) have been used successfully to treat specific lesions of disseminated focal hyperkeratosis in the dog (Ihrke and Goldschmidt, 1983). The dogs were otherwise healthy and without evidence of vitamin A deficiency (vitamin A not measured), indicating that the condition is probably an idiosyncrasy of individual dogs. This response to vitamin A is somewhat similar to that for certain cases of acne or ichthyosis in humans.

Hypervitaminosis A

Maddock et al. (1949), using 2-month-old Greyhound puppies, orally administered 300,000 IU vitamin A per kilogram of body weight each day except Sunday. Anorexia was first noted on the thirtieth day. Weight gains were 60 to 70 percent of controls for the first 53 days, but at this time weight declined precipitously. After 53 days a variety of clinical signs rapidly appeared. Hyperesthesia of the skin and extreme tenderness of the extremities were evident. The puppies were unwilling to stand, although no fractures were noted. The long bone epiphyseal cartilage was markedly narrower; cortices of the femur, tibia, radius, and ulna were less dense and thinner. Remodeling processes were greatly accelerated, and hemorrhage was common in these areas. Moderate exophthalmos was evident. Degenerative lesions of the media were found in arteries and veins of the myocardium, gall bladder, and urinary bladder. Serum vitamin A levels reached 8,380 to 20,400 IU/dl, compared to 660 to 1,182 IU/dl in the controls. These values may be compared with those reported by Keane et al. (1947) in 21 healthy dogs examined at a New Jersey animal hospital. The range of plasma vitamin A concentrations was 180 to 1,800 IU/dl, with a mean of 564 IU.

These observations were confirmed by Cho et al. (1975), who fed Labrador Retriever puppies a commercial diet while subjecting them to weekly injections of 100,000 IU/kg (roughly equivalent to 5,000 μg/kg/d). Only slight weight reduction was observed after 14 weeks. The puppies then received 300,000 IU/kg per day (100,000 μg/kg/d) and experienced an immediate weight loss, lethargy, anorexia and emaciation, and limb-joint pain. After 14 more weeks, fatty liver was noted at necropsy. Bone remodeling defects resulted in decreased bone length and thickness due to premature epiphyseal closure and resorption of diaphyseal bone in long bones. No degenerative vascular lesions were evident, although microhemorrhages were observed. Spontaneous bone fractures did not occur, but microcalculi were found in kidney tubules. Another group of puppies received a weekly injection of a vitamin A-D-E combination (supplying 200,000 IU vitamin A, 30,000 IU vitamin D, and 20 IU vitamin E per week) without appreciable effect other than failure to gain weight during the last 7 weeks of the 14-week study.

Wiersig and Swenson (1967) found that daily oral administration of 125,000 IU of vitamin A per kilogram of body weight to Beagle bitches on gestation days 17 to 22 produced cleft palate in the puppies.

Hendricks et al. (1947) found no adverse effects due to the continuous feeding of 10,000 IU vitamin A per kilogram of body weight to weaned Cocker Spaniel puppies for 8 to 10 months.

Vitamin A has been used pharmacologically for certain diseases in dogs presumably receiving adequate dietary levels of this nutrient. Wakerlin et al. (1942) reported marked reductions in blood pressure in dogs with experimental renal hypertension when given 200,000 IU daily per os for 3 months, followed by 400,000 IU daily for an additional 3 months. In dogs with experimental atherosclerosis, Krause and Brown (1967) found that, while atherosclerotic dogs did not show impaired glucose tolerance, oral daily supplements of 5,000 IU vitamin A increased the rate of glucose utilization. Martin (1971) found that corneal epithelial healing rate was not improved by a single oral dose of 100,000 IU vitamin A plus 25,000 IU administered topically 4 times a day as compared to untreated controls, nor did vitamin A counteract corticosteroid inhibition of epithelial healing.

Vitamin D

The dog has been utilized extensively for studies of vitamin D metabolism, and it was in this species that the separate effects of vitamin A and vitamin D on bone growth and development were identified (see Mellanby, 1957). Since that initial discovery, the dog has been a widely used model in association with the influence of this vitamin on calcium and phosphorus metabolism. Unfortunately, many vitamin D investigators have fed dogs a crude cereal-based formula that contains an incomplete

vitamin mix, no salt mix, and an extremely low calcium level and a calcium: phosphorus ratio of 1:8 (Kelly, 1967). This diet raises serious questions concerning the physiological relevance of the results and their interpretation in this species.

Vitamin D is now recognized as a hormone that can be synthesized from 7-dehydrocholesterol when the skin is exposed to ultraviolet sunlight. The product, cholecalciferol, is in turn converted to an active metabolite in a two-step process in liver and kidney. The hepatocyte converts the cholecalciferol to 25-OH cholecalciferol which is transported to the kidney and converted to $1,25(OH)_2D_3$ when the serum calcium-regulated parathyroid hormone (PTH) level is elevated or the kidney concentration of phosphate ion is low. It is thought that $1,25(OH)_2D_3$ is the metabolite that most actively induces calcium absorption in the gut (Brickman et al., 1973). The release of PTH from the parathyroid gland of the dog may be suppressed by the alternative kidney metabolite $24,25(OH)D_3$ (Canterbury et al., 1978), while $1,25(OH)_2D_3$ and $24,25(OH)_2D_3$ appear to interact with PTH at the bone level to modulate calcium resorption and homeostasis (Massry et al., 1979).

Most vitamin D research has been done with cholecalciferol (vitamin D_3), rather than ergocalciferol (vitamin D_2), utilizing rats or chickens; but this research is increasingly occurring in dogs. It appears that the above-mentioned metabolic interconversions also occur in the dog (Midgett et al., 1973).

Requirements

Requirements for vitamin D are dependent on dietary concentrations of calcium: and phosphorus, the dietary calcium: phosphorus ratio, physiological stage of development, and perhaps sex and breed. Kozelka et al. (1933) found that Collie puppies were protected from rickets by 1 to 1.3 IU vitamin D (irradiated ergosterol) per kilogram of body weight per day. Arnold and Elvehjem (1939) found calcification to be normal in a growing Airedale puppy receiving 13 IU or less of vitamin D per kilogram of body weight per day. Further studies with Great Dane puppies receiving a 1.39 percent calcium and 1.05 percent phosphorus (Ca/P = 1.32:1) diet and 12 IU or less of vitamin D per kilogram of body weight per day showed that growth and bone mineralization were normal. When part-Great Dane puppies were fed diets with a calcium:phosphorus ratio of either 1.2:1 or 2.0:1, providing 12 IU or less of vitamin D per kilogram of body weight per day, the puppy receiving a calcium:phosphorus ratio of 1.2:1 was normal throughout the 125-day trial; the puppy receiving a calcium:phosphorus ratio of 2.0:1 became severely rachitic. Fleischmann Laboratories (1944) reported that 28 IU vitamin D per kilogram of body weight daily was sufficient for Fox Terriers when using a dietary calcium:phosphorus ratio of 2.1:1. However, even with 270 IU per kilogram of body weight per day, Collies and Great Danes showed X-ray evidence of rickets. Michaud and Elvehjem (1944) concluded that, with a dietary calcium:phosphorus ratio of 1.2:1, daily intakes of 10 to 20 IU vitamin D per kilogram of body weight were adequate, even for large breeds.

Wheatley and Sher (1961), in an analysis of the lipids of dog skin, were unable to isolate 7-dehydrocholesterol (provitamin D) despite the empirical observation in dogs (McCay, 1949) that sunlight exposure minimized problems with rickets. The latter implied the probable conversion of the vitamin D precursor upon exposure to ultraviolet light. Arnold and Elvehjem (1939) have concluded that dogs use orally administered ergocalciferol (vitamin D_2) or cholecalciferol (vitamin D_3) equally well.

When the dietary calcium:phosphorus ratio is 1.2:1, daily vitamin D requirements should be met by 8 IU (0.20 µg) per kilogram of body weight for adult maintenance and 22 IU per kilogram of body weight for growing puppies. These amounts will be more than provided by a concentration (dry basis) of 363 IU per kilogram of a food supplying 3,300 kcal ME. Since 40 IU equals 1 µg of vitamin D, this would be equivalent to 9 µg of vitamin D per kilogram of diet (dry weight), or 110 IU/1,000 kcal ME.

Signs Of Deficiency

Vitamin D deficiency signs are frequently confounded by a simultaneous deficiency or imbalance of calcium and phosphorus. Campbell and Douglas (1965) fed a 0.5 percent calcium and 0.3 percent phosphorus diet, with no supplemental vitamin D, to puppies for 15 weeks without signs of rickets or osteoporosis. Likewise, plasma calcium and inorganic phosphorus concentrations, plasma alkaline phosphatase activity, and calcium and phosphorus retention were normal. When the diet contained 0.08 to 0.10 percent calcium and 0.13 to 0.15 percent phosphorus and no supplemental vitamin D, rickets complicated by osteoporosis was observed. When this diet plus a daily supplement of 100 IU vitamin D per kilogram of body weight was supplied, osteoporosis was evident, but rachitic changes were only very slight. A frequently used model of vitamin D deficiency in dogs utilizes the crude cereal-based formula mentioned earlier, which provides only 0.05 percent calcium and 0.42 percent phosphorus. This diet required at least 3 months to depress the serum calcium approximately 2 mg/dl while lowering the serum $25(OH)D_3$ level to <0.4 ng/ml (30 to 60 ng/ml is the normal human value) (Oldham et al., 1980).

Hypervitaminosis D

Morgan and Shimotori (1943) administered a single oral dose of 20,000 IU vitamin D (from tuna liver oil, irradiated ergosterol, or activated animal sterol) per kilogram of body weight to three Cocker Spaniel puppies that had been depleted of vitamin D for 2 months after weaning. They were observed until they were 12 to 14 months old. No deleterious effects on growth, appetite, or general behavior were noted. A transient hypercalcemia was apparent in the first postdosing blood sample taken at 4 hours. Vitamin D was measurable in the blood for 3 days to 5 months postdosing. At 12 to 14 months of age, these dogs were given a second oral dose of 200,000 IU vitamin D (irradiated ergosterol or animal sterol) per kilogram of body weight. Vomiting and diarrhea were observed within 3 days, along with lassitude, weakness, rapid respiration, excessive lacrimation, and anorexia. Serum calcium concentrations first declined and then rose, together with inorganic phosphorus levels, within the first 12 hours. After 3 days the dogs were killed, and tissue vitamin D concentrations were 1.6 to 5.0 IU per gram of fresh tissue in the liver, 3.0 to 8.0 IU per gram in the kidney, and 3.0 to 5.0 IU per gram in the heart.

Morgan et al. (1947) administered a single oral dose of 314,000 to 530,000 IU vitamin D as irradiated ergosterol per kilogram of body weight to 4- to 5-week-old puppies. All exhibited anorexia, polyuria, bloody diarrhea, polydipsia, and prostration. Three were dead within 2 weeks and a fourth was moribund in 5 weeks. Extensive calcification was found in the lungs of these dogs, and moderate calcification in the hearts and kidneys. In the dogs that survived, malocclusion, pitting, irregular placement, and poor development of the teeth were seen.

Hendricks et al. (1947) fed 10,000 IU vitamin D daily per kilogram of body weight to weaned Cocker Spaniel puppies. Irradiated ergosterol, irradiated animal sterol, or tuna liver oil served as the source. Treatment was continued for 8 to 10 months. Anorexia developed, growth was retarded, serum calcium was variably increased, jaws and teeth were deformed, and soft tissues were calcified—particularly the lungs, kidneys, and stomach.

Vitamin D toxicity in the dog is generally associated with a hypercalcemia in excess of 12.5 mg/dl calcium. Caywood et al. (1979) demonstrated that this level could be achieved with a chronic oral dose of 60 to 120 ng/kg of $1,25(OH)_2D_3$ administered over a 4-week period. A level of 180 ng/kg daily induced hypercalcemia in as little as 2 weeks. However, it was recommended that a dose of 100 ng/kg be administered for a month for treatment of bone-loss hypocalcemia.

Spangler et al. (1979) investigated the pathogenesis of vitamin D nephropathy in mixed-breed dogs given 20,000 to 40,000 IU per kilogram vitamin D_2 orally each day for 1 to 3 weeks. Serum calcium rose from approximately 10 to 15 mg/dl, and serum urea nitrogen rose significantly. Serum phosphorus was unchanged, while renin activity increased. Changes in blood pressure were minor, even though vitamin D intoxication usually produces hypertension in the dog. Kidney pathology suggested that renal vascular damage caused ischemia and concomitant hyperplasia, hypertrophy, and hypersecretion of the juxtaglomerular cells. Clinically, the dogs decreased food consumption after 8 days, and by 2 weeks they had lost condition and were dehydrated, with dry, brittle hair and muscle atrophy. The clinical signs were similar to those reported following oral consumption of a smaller dose (200 to 375 µg/kg per day) to dogs for a longer period of 3 to 11 weeks (Mulligan and Strickler, 1948). In the latter experiment metastatic calcification was observed in the lung, Henle's loops in the kidney, and the gastric mucosa.

Vitamin E

While the need for vitamin E in dog diets was demonstrated by Anderson et al. (1939), the interrelationship with dietary selenium concentrations has only recently been studied (Van Vleet, 1975). Since selenium was identified as an essential nutrient in 1957 (Schwarz and Foltz, 1957), few of the vitamin E studies with dogs have taken this factor into account. Both nutrients are important in protecting cell membranes against peroxidation and the destructive effects of free radicals.

Vitamin E serves to quench free radicals in the polyunsaturated fatty acids (PUFA) of membrane phospholipids; and selenium-containing glutathione peroxidase reduces peroxides, particularly those that form in the cytosol and in the mitochondrial matrix, thereby protecting the membrane PUFA from additional insult (Tappel, 1980).

Requirements

Since the PUFA content of membranes can be altered by dietary fats, it is not surprising that the dietary requirement for vitamin E is closely related to the dietary concentration of PUFA. When a large amount of polyunsaturated fat is fed after it has been stripped of tocopherols, as much as 100 mg α-tocopherol per kilogram of diet may be insufficient to protect against lipofuscin formation (Hayes et al., 1969).

Harris and Embree (1963) have proposed a dietary α-tocopherol: PUFA ratio (mg/g) of 0.6:1 as a minimum to protect against PUFA peroxidation. It is noteworthy

that human diets in the United States have an average α-tocopherol:PUFA ratio of 0.43:1, without evidence of vitamin E deficiency (Bieri and Evarts, 1973).

Elvehjem et al. (1944) reported that 0.62 mg (0.68 IU) α-tocopherol per kilogram of body weight per day would not sustain normal reproduction in Fox Terriers fed unsweetened, irradiated evaporated milk, while 1 mg (1.1 IU) would. However, one pup out of four from a bitch receiving the higher level of vitamin E exhibited slight muscular dystrophy.

Van Vleet (1975) fed Beagle puppies a diet containing 5 percent stripped lard and Torula yeast and varying levels of vitamin E or selenium. Signs of deficiency were observed when a basal diet containing 0.01 ppm selenium and 1 mg/kg α-tocopherol was fed. Feeding α-tocopherol at 30 IU per kilogram of diet alone or 1 ppm selenium alone as selenite prevented deficiency both clinically and histopathologically, whereas 0.5 ppm selenium only prevented clinical signs of deficiency.

From these data, and assuming that one feeds a dry diet containing not more than 1 percent linoleic acid and at least 0.1 ppm selenium, the recommended allowance (including that for reproduction and growth) should be satisfied by 20 IU vitamin E per kilogram of a diet supplying 3,300 kcal ME. This is equivalent to 6.1 IU/1,000 kcal ME. The data suggest that approximately 1.1 IU per kilogram of body weight should be allowed daily for pregnancy and 1.2 IU per kilogram of body weight for growth. If dietary PUFA levels are increased, it is suggested that an α-tocopherol:PUFA ratio (mg/g) of at least 0.5 be maintained. Rancid fats should be avoided because of their particular destructiveness to tocopherols.

Signs Of Deficiency

A number of authors (Anderson et al., 1939, 1940; Brinkhous and Warner, 1941; Elvehjem et al., 1944; Cordes and Mosher, 1966; Van Kruiningen, 1967; Hayes et al., 1969, 1970; Hayes and Rousseau, 1970; Riis et al., 1981) have published signs of presumed vitamin E deficiency. Particularly prominent were degeneration of skeletal muscle associated with muscle weakness, degeneration of testicular germinal epithelium and failure of spermatogenesis, failure of gestation, weak and dead pups, brown pigmentation (lipofuscinosis) of intestinal smooth muscle, decreased plasma tocopherol concentrations, increased dialuric acid hemolysis of erythrocytes, and elevated plasma creatine phosphokinase values. A remarkable retinal degeneration was described by Hayes et al. (1970) and reproduced experimentally (Riis et al., 1981) in puppies fed a diet containing stripped corn oil. In as few as three months ophthalmascopic lesions were visible, which represented lipid peroxidation and disruption of photoreceptors with accumulation of lipofuscin pigment.

Vitamin E deficiency in dogs also appears to impair their immune response as measured by the mitogenic response of lymphocytes (Langweiler et al., 1981). The impairment is attributable to a suppressor factor in the serum, since washed cells become responsive and serum alone from deficient dogs is able to depress the response in control lymphocytes.

A combined vitamin E-selenium deficiency described by Van Vleet (1975) included muscle weakness, subcutaneous edema, anorexia, depression, dyspnea, and coma. Pathologic examination revealed extensive skeletal muscle degeneration and regeneration, focal subendocardial necrosis, lipofuscinosis, and renal mineralization.

Vitamin E is not generally thought to be toxic; however, it has been observed that although vitamin E did not interfere with coagulation in normal dogs, it was able to block oxidation of vitamin K to the epoxide form in Warfarin-treated dogs and thereby exacerbate the coagulopathy (Corrigan, 1979).

Vitamin K

Requirements

The metabolic need for vitamin K has been well established in the dog. Anderson and Barnhart (1964) have shown that vitamin K_1 (2-methyl-3-phytyl-1,4-naphtho-quinone) stimulates prothrombin synthesis by the liver parenchymal cells in dogs made hypoprothrombinemic by coumarin compounds. Duello and Matschiner (1971a,b) isolated 19 vitamin K analogs in dog liver and suggested that most were absorbed from the intestine and were not tissue metabolites. A bacterial origin for many of these vitamins was considered likely.

The need for supplemental vitamin K has been demonstrated in adult dogs following diversion of bile from the intestine by means of a cholecystonephrostomy (Quick et al., 1954). Vitamin K absorption from both diet and intestinal bacterial synthesis was apparently reduced, and 0.5 µg vitamin K_1 per kg of body weight injected intravenously each day sustained normal plasma prothrombin levels. Using the same surgical technique with puppies, Quick et al. (1962) concluded that daily intravenous injections of 10 to 15 µg vitamin K_1 per kilogram of body weight were necessary to sustain normal plasma prothrombin levels during active growth, with a decline in requirement to 5 µg or less per kilogram of body weight as the dogs approached mature weight. Robinson et al. (1964) studied whether or not cholestyramine, a bile acid-binding resin, would interfere with vitamin K_1 absorption.

In the dose range used in humans for control of hypercholesterolemia (200 mg per kilogram of body weight), there was no measurable effect on vitamin K_1 absorption. At larger doses (1 to 3 g per kilogram of body weight), vitamin K_1 absorption was decreased and delayed somewhat.

Clark and Halliwell (1963) administered 2.2 mg Warfarin, 3-(α-acetonylbenzyl)-4-hydroxycoumarin, per kilogram of body weight to adult Greyhounds daily for 3 days. This decreased the prothrombin time to 10 percent of normal. Subsequent daily intravenous administration of vitamin K_1 at levels of 0.28 to 4.4 mg per kilogram of body weight returned prothrombin time to 70 percent of normal in 2 to 4 days, although the higher dosages produced a more rapid response. One oral dose of 2.2 mg vitamin K_1 per kilogram of body weight returned prothrombin time to 70 percent of normal in 8 hours, but this value declined to 40 percent at 24 hours. Intramuscular dosage of vitamin K_1 produced a slower, but more sustained, response. Menadione (menaquinone, or 2-methyl-1,4-naphthoquinone) and 2-methyl-1,4-naphthohydroquinone diphosphate administration did not produce a prothrombin response.

Whether dietary vitamin K is likely to be limiting in the absence of compounds that interfere with bacterial vitamin K synthesis, vitamin K absorption, or function is not clearly established. Bratt et al. (1965) reported a suspected vitamin K deficiency in newborn pups that occasionally responded to vitamin K therapy. There were no controls. Reber and Malhotra (1961) fed a diet calculated to contain 60 µg vitamin K per kilogram of solids to adult male Beagles for 40 weeks. No evidence of vitamin K deficiency was seen in the dogs or in adult cats fed the same diet, but 75 percent of weanling Sprague-Dawley rats fed this diet died from hemorrhage.

Although it is doubtful that supplemental vitamin K is necessary for the normal dog, it may be prudent to provide 22 µg menadione (or vitamin K equivalent) per kilogram of body weight daily for adult maintenance and 44 µg per kilogram of body weight during growth. This would be more than supplied by a dry diet concentration of 1.0 mg menadione per kilogram.

Signs Of Deficiency

A simple vitamin K deficiency has not been described in the dog. When vitamin K absorption is reduced by cholecystonephrostomy (Quick et al., 1962), dogs become hypoprothrombinemic and exhibit massive hemorrhage. Similar signs appear subsequent to coumarin administration (Clark and Halliwell, 1963). Coumarins also induce liver parenchymal cell ultrastructural changes (Barnhart et al., 1964), such as collapse of membranous elements of the endoplasmic reticulum around the mitochondria and reduced cytoplasmic ribosome concentration.

Hypervitaminosis K

Vitamin K_1 is apparently safer in large quantities than the water-soluble analogs and derivatives of menadione (vitamin K_3). The latter are widely employed, but they may produce toxic side effects in the newborn when administered parenterally. Doses up to 10 to 25 mg of vitamin K have been administered to pregnant women prior to and during delivery, or to the newborn infant, to prevent hypoprothrombinemia and hemorrhagic disease in the child. When vitamin K_1 was used, this practice was apparently not harmful; however, 5 to 10 mg of menadiol sodium diphosphate administered daily to infants produced hemolytic anemia, and 10 mg given 3 times a day for 3 days to premature infants resulted in kernicterus and death. The mechanism of toxicity involves erythrocyte hemolysis and subsequent overloading of an immature liver with bilirubin, which cannot be sufficiently conjugated and which in turn proves toxic to the neonatal brain (kernicterus) (Miller and Hayes, 1982).

The only reported case of toxicity in the dog occurred in a 1-year-old female Great Dane that ingested a packet of Warfarin and was treated intravenously with 30 mg of vitamin K_1 in 5 percent dextrose and lactated Ringer's solution. An acute urticaria was observed with wheals first appearing on the face before progressing caudally over the entire trunk. Flatulence, lacrimation, and salivation were also noted (Jordan, 1979).

Thiamin

Requirements

Arnold and Elvehjem (1939) demonstrated that a diet containing 2 percent fat and 500 µg thiamin chloride per kilogram was inadequate to maintain food intake and growth of puppies. They estimated the thiamin requirement for growth was at least 750 µg thiamin chloride per kilogram of diet and demonstrated a lower requirement for two dogs given either a 22.5 or a 56.5 percent fat diet. On a body weight basis, their estimated requirement for growth was about 40 µg thiamin per kilogram of body weight per day.

Noel et al. (1971) fed 24 growing Beagle dogs a semipurified diet fortified with B vitamins other than thiamin. The dogs were divided into four groups and given the following quantities of thiamin per kilogram of body weight daily: group 1: 110 µg; group 2: 33 µg; and group

3: 22 µg; the fourth group was given 115 µg twice a week. All dogs except for those in groups 1 and 4 were maintained on this protocol for 29 weeks. For the latter two groups, the supplemental thiamin was reduced for the last 8 weeks to 11 µg per kilogram of body weight daily and 77 µg per kilogram of body weight twice weekly, respectively. No clinical abnormalities were detected in the dogs except for one bitch in group 3, which began to lose weight after 9 weeks of receiving 22 µg thiamin per kilogram of body weight per day, and died at 22 weeks. This dog exhibited transitory diarrhea at week 19, and the terminal signs were a sudden loss of appetite and body weight, accompanied by weakness. The transketolase activity of the erythrocytes of this dog were stimulated markedly by thiamin pyrophosphate, indicating gross thiamin deficiency (Brin and Vincent, 1965). No other dogs showed changes in transketolase activity. These authors concluded that 22 µg thiamin per kilogram of body weight per day was too low to support Beagles in a satisfactory state of health during the first year of life.

Cowgill (1934) reported that a daily intake of 6 µg thiamin per kilogram of body weight was sufficient for maintenance of mature dogs. Slightly higher values were suggested by Street et al. (1941), who reported that adult dogs could be maintained in apparent good health for 129 to 386 days on a 25 percent fat diet when supplied daily with 6.7 to 9.4 µg thiamin per kilogram of body weight. They suggested an allowance of approximately 8 µg thiamin per kilogram of body weight for maintenance of dogs of 6 to 10 kg. Maass et al. (1944) found that less than 10 µg per kilogram of body weight per day was inadequate for weight maintenance of adult dogs and that the thiamin requirement of adult and growing dogs did not appear to be increased by phlebotomy. These workers were the first to use a semipurified diet fortified with crystalline riboflavin, pyridoxine, calcium pantothenate, niacin, and choline chloride to investigate thiamin requirements. Previous workers such as Arnold and Elvehjem (1939) and Street et al. (1941) had relied on autoclaved yeast to supply B vitamins other than thiamin.

Thiamin requirements are influenced by both physiological and dietary factors. Experimental hyperthyroidism has been shown (Drill 1941; Drill and Hays, 1942; Drill and Shaffer, 1942) to increase thiamin requirements of the dog on a body weight basis. There is limited evidence from the dog and other mammals that thiamin requirements per kilogram of diet are greater for pregnancy and lactation than for growth (e.g., Voegtlin and Lake, 1919).

The level of fat and protein in the diet and the inclusion of penicillin, lactose, sorbitol, and ascorbic acid are reported to be inversely related to thiamin requirement in other species (Evans and Lepkovsky, 1929 a, b, 1935; Scott and Griffith, 1957; Haenel et al., 1959). However, a subsequent study with rats did not support the effect of fat (Murdock et al., 1974). Thiamin requirements of the cat were reported by Deady et al. (1981) to be increased by feeding a diet high in glutamic acid (an amino acid present in high concentrations in some vegetable proteins). Factors favoring intestinal microbial synthesis of thiamin, e.g., by diets containing starch rather than sucrose, and practice of coprophagy can markedly influence dietary requirements for this vitamin.

Natural feeds may contain compounds with antithiamin activity that increase dietary needs. Thiaminases are present in animal tissues, notably in some freshwater and saltwater fish, shellfish, and crustaceans; some plants, e.g., ferns; and some bacteria and fungi. In general, these thiaminases are heat-labile, so are inactivated by cooking. Some plants contain small thermostable molecules (e.g., *o*-dihydroxyphenols such as caffeic acid and catechol) which react with thiamin and prevent its giving the thiochrome reaction (Davis and Somogyi, 1969). Evans (1975) suggests that the main product formed from the reaction of these thermostable "antithiamin" factors is thiamin disulfide, which is biologically inactive. Further, he suggests that thermostable antithiamin factors appear to have little nutritional significance to animals.

Thiamin is readily destroyed by heat, especially under basic conditions. Losses of 74 percent of thiamin have been reported for some canned dog foods due to retorting and storage for 14 days (Roche, 1981). Naturally occurring clinical cases of thiamin deficiency in dogs attributed to thermal destruction of thiamin in meat have been reported (Read et al., 1977). Therefore, intake of thiamin should be calculated from analyses of diets taken at the time of consumption.

The thiamin requirements of the normal adult dog for maintenance can be met by 20 µg per kilogram of body weight daily, and the growing dog by 40 µg per kilogram of body weight. There do not appear to be any published data to permit definition of a requirement for pregnancy or lactation. All evidence suggests that 270 µg thiamin/1,000 kcal ME is adequate for maintenance and growth.

Signs Of Deficiency And Pathology

Because of the body's limited capacity to store thiamin, clinical signs may be observed after a relatively short period of ingestion of a thiamin-deficient diet. Anorexia has been consistently observed as an early clinical sign of thiamin deficiency. Read and Harrington (1981)

fed 2- to 5-month-old Beagle dogs a thiamin-deficient diet (20 to 30 µg thiamin per kilogram of diet) and described three clinical stages of the disease: an initial short (18 ± 8 days) stage of induction, during which the dogs grew suboptimally, but were otherwise healthy; an intermediate stage characterized by a variable period (59 ± 37 days) of progressive inappetence, failure to grow, loss of body weight, and coprophagy; and a terminal period that in most dogs was abrupt and short (8 ± 6 days) and consisted of either a neurological syndrome or sudden unexpected death. The neurological syndrome was characterized by anorexia, emesis, central nervous system depression, paraparesis, sensory ataxia, torticollis, circling, tonic-clonic convulsions, profound muscular weakness, and recumbency.

Erythrocyte transketolase activity was depressed in deficient dogs. In vitro addition of thiamin pyrophosphate to red cells from deficient dogs gave a stimulation of transketolase activity above the normal of 11 ± 4 percent (Brin and Vincent, 1965; Noel et al., 1971; Read, 1979). Pathological changes due to thiamin deficiency predominately involve the nervous system and heart. The pattern of changes depends on the period of induction; acute deficiencies tend to involve the brain and produce severe neurological signs, whereas chronic deficiencies produce pathological changes of the myocardium and peripheral nerves (Read, 1979). Brain lesions include symmetrical necrosis of the gray matter of the inferior colliculi, medial vestibular nuclei, cerebellar nodulus, claustra and cerebral cortex (Read et al., 1977; Read, 1979).

Histologically, the peripheral neuropathy reported by Cowgill (1921) and subsequent workers is characterized by diffuse bilateral myelin degeneration and axonal disintegration (Voegtlin and Lake, 1919; Street et al., 1941; Read, 1979).

In contrast to thiamin deficiency in humans, cardiac hypertrophy is not a constant lesion in dogs. Andrews (1912) described hypertrophy of the right heart in one of seven puppies suckled by mothers whose babies had died from beriberi. Voegtlin and Lake (1919) and Street et al. (1941) also reported several cases of enlargement of the heart. Read (1979) described the cardiac lesion as nonspecific multifocal myocardial necrosis, and suggested primary vascular damage may be involved.

The in vitro measurement of erythrocyte transketolase stimulation by thiamin pyrophosphate (Brin and Vincent, 1956; Noel et al., 1971; Read, 1979) has been used to diagnose thiamin deficiency in the dog. However, a decrease in the concentration of thiamin pyrophosphate in the blood of rats has been shown to precede changes in transketolase activity (Warnock et al., 1978) and may be a superior test for the dog.

Hypervitaminosis Thiamin

Rapid intravenous injection of 5 to 50 mg thiamin per kilogram of body weight causes a transient fall in blood pressure, with more severe effects from higher dosages. The lethal dose is approximately 350 mg per kilogram of body weight (Neal and Sauberlich, 1980), and death is due to depression of the respiratory center. Under ether anesthesia, blood thiamin concentrations of 7 to 10 mg/ml were fatal. The ratios of lethal intravenous doses to those administered subcutaneously or orally were estimated to be 1:6:40 (Unna, 1954).

Riboflavin

Microbial biosynthesis of riboflavin and other alloxazines has been shown to occur in the gastrointestinal tract of a number of animal species. However, utilization of this endogenously synthesized riboflavin varies from species to species. Within a single species, utilization depends on the composition of the diet (Christensen, 1973) and incidence of coprophagy. Young rats fed a riboflavin-free, purified diet with sucrose as the only carbohydrate will cease to grow. However, when sucrose is replaced by starch, sorbitol, or lactose, growth is comparable to that of rats supplied with riboflavin (Fridericia et al., 1927; Haenel et al., 1959). Excretion of riboflavin in urine and feces is also dependent on the carbohydrate in the diet and is suppressed by inclusion of sulfa drugs in the diet (De and Roy, 1951). The contribution of symbiotically synthesized riboflavin to the dog's requirement is not known.

Street and Cowgill (1939) fed adult dogs a basal diet containing 30 percent casein, 36 percent sucrose, and 27 percent fat and an extract of rice polishings to supply B vitamins other than riboflavin. Another group of dogs was pair-fed the same diet, plus rice polishing extract and 25 µg riboflavin per kilogram of body weight. Those dogs pair-fed the supplemented diet lost body weight, because of food restriction, but remained healthy for 130 to 196 days. Dogs fed the unsupplemented diet collapsed after 120 ± 18 days of consuming the basal diet and generally responded to treatment with 0.75 mg riboflavin per kilogram of body weight. Street et al. (1941) confirmed, using the same casein-sucrose-fat diet as that used in their 1939 study, that 4 to 8 µg riboflavin per kilogram of body weight daily was inadequate for adult dogs, but 25 µg per kilogram of body weight maintained dogs without clinical signs of deficiency.

Estimates of the minimal riboflavin requirement of the growing dog were made by Axelrod et al. (1940). When riboflavin was supplemented once weekly, 2 mg per kilogram of diet was inadequate, but 4 mg per kilogram

of diet was sufficient. In a later study (Axelrod et al., 1941) they reported a minimal requirement of 2 mg per kilogram of diet, but tissue storage was low, which suggested that 4 mg per kilogram of diet was a satisfactory level. Potter et al. (1942) reported that 60 to 100 µg per kilogram of body weight appeared to give comparable growth rates but marked differences in tissue storage. These authors also calculated that the dietary riboflavin requirement of the growing dog was more than 2 mg per kilogram of diet. They further showed that isocaloric substitution of lard for sucrose in the diet did not increase riboflavin requirements of the growing dog.

Spector et al. (1943) subjected both young and adult dogs given variable riboflavin intakes to repeated phlebotomy. They suggested that 30 µg riboflavin per kilogram of body weight per day is necessary for growing dogs for good hemoglobin production and rapid recovery from anemia and that 15 µg riboflavin per kilogram of body weight per day is required by adult dogs. Heywood and Partington (1971), however, reported corneal lesions in dogs 4.5 to 5 months of age given diets containing various levels of riboflavin for 17 weeks. Without riboflavin supplementation corneal edema was seen in the fifth week, which developed to superficial vascularization at the eleventh week. Bilateral corneal opacities were also observed in one of four dogs receiving 30 µg riboflavin/kg/d. Two out of three male dogs receiving 55 µg/kg/d developed bilateral corneal lesions. Noel et al. (1972) reported corneal opacities without vascularization in two growing male Beagles receiving a diet providing 39 to 60 µg riboflavin per kilogram of body weight.

It is concluded that a daily intake of 50 µg riboflavin per kilogram of body weight for adult dogs and 100 µg riboflavin per kilogram of body weight for growing puppies will provide adequate levels of the vitamin for reasonable tissue storage. No data derived from dogs are available to give a dietary requirement for gestation and lactation. This translates to 0.68 mg riboflavin per 1,000 kcal of dietary ME.

Signs Of Deficiency

Acute riboflavin deficiency may result in anorexia, hypothermia, a decreased respiratory rate, apathy, progressive weakness, ataxia, sudden collapse to a semicomatose condition, and death (Street and Cowgill, 1939). Chronic hyporiboflavinosis has been associated with anorexia; loss of body weight; muscular weakness, particularly of the hind quarters; dry, flaky dermatitis, accompanied by erythema of the hind legs, chest, and abdomen; and ocular lesions. The ocular lesions are generally bilateral, and progress from a watery or purulent discharge accompanied by conjunctivitis to opacity and vascularization of the cornea (Street et al., 1941; Potter et al., 1942; Heywood and Partington, 1971; Noel et al., 1972).

Hyporiboflavinosis is accompanied by a reduction in erythrocyte riboflavin concentration, a reduced urinary excretion of riboflavin, and a low urinary recovery of a riboflavin test load (Axelrod et al., 1941; Potter et al., 1942; Noel et al., 1972). The anemias and fatty livers that early workers associated with hyporiboflavinosis were probably induced by the diet's lacking other essential factors, e.g., choline, folic acid, or vitamin B_{12}, required for normal hematopoiesis and lipid transport (Noel et al., 1972).

Erythrocyte glutathione reductase assay is currently the preferred test for diagnosis of riboflavin deficiency in humans. Factors affecting the assay are described by Thurnham and Rathkette (1982).

Hypervitaminosis Riboflavin

Riboflavin has a low toxicity, which may be a result of its low solubility. Dogs given a single oral dose of 2 g riboflavin per kilogram of body weight showed no ill effects (Unna and Greslin, 1942). Similarly, four 10-week-old puppies were fed 25 mg riboflavin per kilogram of body weight for 5 months, and neither toxic signs were observed, nor pathological changes in the organs at necropsy.

Pantothenic Acid

McKibben et al. (1939, 1940), Morgan and Simms (1940), and Fouts et al. (1940) demonstrated the necessity of pantothenic acid in the diet of the dog. Schaefer et al. (1942) fed weanling puppies and adult dogs a diet of 66 percent sucrose, 19 percent casein, 11 percent fats and oils, minerals and equal amounts of thiamin, riboflavin, niacin, pyridoxine, and choline, and varying levels of calcium pantothenate. These authors concluded that 100 µg calcium pantothenate per kilogram of body weight per day was adequate to prevent deficiency signs in growing puppies, but 60 µg per kilogram of body weight was insufficient. Adult dogs required less calcium pantothenate per unit of body weight than growing dogs.

Sheffy (1964) depleted Beagle puppies 4 to 5 weeks of age of pantothenate by feeding a purified diet not supplemented with pantothenate for variable periods of time. Supplements of calcium pantothenate approximating 0, 50, 100, 200, 500, and 1,000 µg per kilogram of body weight per day were given. Coincidental with the initiation of supplementation, a single inoculation of

a virus was given, and change in body weight and antibody response were measured. Puppies receiving the 0-or 50-µg levels of calcium pantothenate died. No significant difference in growth rate occurred between those receiving 200, 500, and 1,000 µg/kg body weight. Dogs receiving either 500 or 1,000 µg/kg had higher antibody responses at 7 days, but not at 21 days following vaccination, than those receiving 200 µg/kg. As the antigen was given on the first day of supplementation, it is not clear whether a longer period of repletion would have given different results. Sheffy concluded that the daily requirement for growth was between 100 and 200 µg per kilogram of body weight.

Free pantothenate appears to be efficiently absorbed by the dog, as Taylor et al. (1974) found that between 81 and 94 percent of an oral dose of sodium (^{14}C) pantothenate was absorbed. Urinary secretion represents the major route of loss from the body, principally as the β-glucuronide (Taylor et al. 1972). The kidney of the dog is distinct from that of other animals in that it excretes little of the free vitamin, but the excretion of the β-glucuronide approaches the glomerular filtration rate.

From the limited data available on dogs and the requirements of other species, it would appear prudent to provide 200 µg pantothenic acid per kilogram of body weight for adult maintenance and 400 µg per kilogram of body weight for growth of dogs as suggested by NRC (1974). No data are available to give estimates for reproduction and lactation. A dietary concentration of 2.6 mg/1,000 kcal ME is considered adequate.

Signs Of Deficiency

Pantothenic acid-deficient dogs exhibit erratic appetites, depressed rates of growth, reduced urinary excretion of the vitamin (Silber, 1944), lowered antibody response (Sheffy, 1964), and reduced blood concentrations of cholesterol, cholesterol esters, and total lipids (Scudi and Hamlin, 1942). Deficient dogs have reduced concentrations of pantothenate in blood, liver, muscle, and brain (Silber, 1944). In the terminal stages of pantothenic acid deficiency, dogs exhibit spasticity of the hind quarters, sudden prostration or coma, usually accompanied by rapid respiratory and cardial rates and possibly conclusions.

Hypervitaminosis Pantothenic Acid

Large amounts (10 to 20 g) of calcium pantothenate have been administered to humans without evidence of toxicity other than occasional diarrhea (Gershberg et al., 1949).

Niacin (Nicotinic Acid, Nicotinamide)

Historically the dog played an important role as a model for the study of pellagra in humans and in testing antipellagra vitamin preparations (Harvey et al., 1938). In a text on nutrition of humans, Chittenden (1907) described clinical signs of disturbances of the gastrointestinal tract with bloody discharge and inflammation of the mucous membrances of the mouth in a dog given a diet of bread and lard. Goldberger and Wheeler (1928) recognized the similarity between these signs of the disease known as "black tongue" in dogs and those of pellagra in humans (Goldberger and Wheeler, 1920). Elvehjem et al. (1937, 1938) demonstrated that nicotinic acid and nicotinamide were equally effective in curing black tongue and in preventing it in dogs given a black tongue-producing diet. Street and Cowgill (1937) also confirmed that nicotinic acid cured black tongue in dogs.

Sebrell et al. (1938) gave variable amounts of nicotinic acid by intramuscular injection semiweekly to dogs of 6 to 8 kg body weight fed a corn-based diet (modified Goldberger diet). On a body weight basis, 340 µg nicotinic acid per kilogram per day prevented appearance of black tongue, while 126 µg per kilogram per day produced incipient signs of the disease. Margolis et al. (1938), also using a modified Goldberger diet, reported that black tongue induced in adult dogs was cured by a daily dose of 500 µg or greater of nicotinic acid per kilogram of body weight. When doses were reduced to 200 µg per kilogram of body weight daily the response was delayed, and 100 µg per kilogram of body weight was ineffective. Birch (1939) also used a corn-based diet and found that 130 µg nicotinic acid per kilogram of body weight gave protection against signs of black tongue and some increase in body weight of depleted adult dogs, whereas 250 µg per kilogram allowed for a rapid weight gain. Both 84 and 27 µg per kilogram of body weight gave no protection.

Prior to the report of Schaefer et al. (1942), all attempts to define the nicotinic acid requirement of the dog had used a Goldberger-type diet, based largely on corn. These authors were the first to use a semipurified diet, which in this study contained sucrose, 66 percent; acid-washed casein, 19 percent; cottonseed oil, 8 percent; cod liver oil, 3 percent; and salt mixture, 4 percent. Consequently, this diet contained a lower content of niacin than diets based on natural food ingredients. The diet was fortified with thiamin, riboflavin, pyridoxine, pantothenic acid, and choline. The requirement of nicotinic acid (calculated from single-dose feedings) for adult dogs was 200 to 225 µg per kilogram of body weight per day and for growing dogs 250 to 365 µg per kilogram of body weight per day.

In most animals nicotinic acid is a minor end product

of tryptophan degradation. Hence, dietary niacin requirements are dependent on the level of tryptophan in the diet. Singal et al. (1948) fed growing dogs a semipurified niacin-deficient diet based on sucrose, 66 percent, and casein, 19 percent. When they replaced 21 percent of the sucrose in the basal diet with an equal weight of either zein or gelatin (proteins low in tryptophan) the time that elapsed before the dogs were depleted was not prolonged. In contrast, when 21 percent of the sucrose was replaced by casein (i.e., 40 percent casein in the diet) about twice as much weight gain occurred before the dogs' weight plateaued. Also, the response to an injection of 10 mg nicotinic acid per kilogram of body weight was greater for dogs fed the 40 percent casein diet than the basal diet alone or with 21 percent gelatin or zein. While none of the above diets prevented eventual onset of clinical signs of nicotinic acid deficiency, complete protection was obtained by substituting 42 percent of the sucrose with casein. Complete protection was also obtained by adding 0.5 percent L-tryptophan (calculated tryptophan in 42 percent casein) to the basal diet.

Further work suggested that the D-isomer of tryptophan was poorly utilized by the dog for niacin synthesis, but increments of 0.3, 0.2, or 0.1 percent DL-tryptophan added to the basal diet gave protection. The efficiency of utilization of D-tryptophan for growth by the dog is about one-third that of L-tryptophan (Czarnecki and Baker, 1982). If one assumes comparable relative efficiencies between the isomers for niacin synthesis, then the lowest total dietary level of tryptophan giving complete protection was equivalent to 0.37 percent of L-isomer. This conclusion is difficult to reconcile with the finding that a diet of 40 percent casein containing 0.48 percent L-tryptophan resulted in the disease.

From a bioassay procedure, these workers concluded that for the dog 132 mg L-tryptophan is equivalent to approximately 1 mg nicotinic acid. This ratio is considerably wider than that proposed for the rat or human. Hankes et al. (1948) calculated that 33 to 40 mg tryptophan yield 1 mg niacin for the rat, and Horwitt et al. (1956) proposed that 60 mg tryptophan are equivalent to 1 mg nicotinic acid for humans. However, this ratio is probably not constant but varies with the tryptophan and niacin concentration of the diet (Anonymous, 1974).

In naturally occurring foods, particularly cereal grains, a considerable proportion of the niacin may be in the bound form, which is unavailable or only partly available unless hydrolyzed. Ghosh et al. (1963), using a microbiological assay, reported that 85 to 90 percent of the total nicotinic acid in cereals was in a bound form. Mason et al. (1973) showed that extraction of wheat bran under neutral conditions yielded 62 percent of the bound niacin in solution; of this, 90 percent was nondiffusible. Bound nicotinic acid was found to be linked to macromolecules of which 60 percent were polysac-charides and 40 percent peptides or glycopeptides. Oil seeds contain about 40 percent of their total niacin in bound form, while only a small proportion of the niacin in pulses, yeast, crustacea, fish, animal tissue, or milk is bound. By use of a rat assay procedure, Carter and Carpenter (1982) showed that for eight samples of mature cooked cereals (corn, wheat, rice, and milo), only about 35 percent of the total niacin was available. In the calculation of the niacin content of formulated diets, probably all niacin from cereal grain sources should be ignored or at least given a value no greater than one-third of the total niacin.

The association of pellagra in humans and of black tongue in dogs with consumption of diets based on corn appears at least in part due to the combination of two factors—the low availability of niacin in corn and the deficiency in tryptophan of the main protein in corn (zein). Researchers from India (Belavady and Gopalan, 1965, 1966; Belavady et al., 1967; Gopalan et al., 1969) have suggested that high levels of leucine in jowar (*Sorghum vulgare*) may induce black tongue in dogs and pellagra in humans. However, researchers from other laboratories (Truswell et al., 1963; Nakagawa and Sasaki, 1977; Manson and Carpenter, 1978) have been unable to reproduce the reported induction of niacin deficiency by high levels of dietary leucine. Whether the condition existing in India associated with consumption of jowar involves a concurrent pyridoxine deficiency or feedback control of tryptophan pyrrolase activity as suggested by Hankes et al. (1971) has not been resolved. There are a number of reports in the literature (e.g., Handler, 1943; Krehl et al., 1945) to suggest that niacin requirements of dogs fed corn-based diets may be higher than those given purified diets.

For usual diets with minimal quantities of tryptophan, the daily requirement of the adult dog will be met by 225 µg niacin per kilogram of body weight and for the growing dog by 450 µg per kilogram of body weight. These amounts will be supplied by diets containing 3 mg of niacin equivalents per 1,000 kcal ME. No experimental data are available to give a requirement for niacin during pregnancy and lactation. However, for humans the rate of conversion of tryptophan to niacin appears to be enhanced in pregnancy due to higher levels of circulating estrogen (Rose and Braidman, 1971; Horwitt et al., 1975).

Battistacci et al. (1979) have shown that urinary excretion of metabolites of the tryptophan-niacin pathway are markedly increased in dogs following surgery. Anesthesia alone had no significant effect on metabolite

levels. The response to surgery is probably mediated by elevated corticosteroid levels and induction of tryptophan pyrrolase activity.

Signs Of Deficiency

Niacin-deficient dogs exhibit anorexia; loss in weight; erythema; severe inflammation and ulceration of the oral and pharyngeal mucosa; profuse salivation with ropy, bloodstained saliva drooling from the mouth; and foul breath. There is bloody diarrhea, inflammation and hemorrhagic necrosis of duodenum and jejunum with shortening and clubbing of villi, and inflammation and degeneration of the mucosa of the large intestine. Intestinal absorption of water, glucose, sodium, and potassium is reduced. Hepatic periportal fatty metamorphosis, neuronal degeneration of the spinal cord, and distortion of conditioned reflexes are evident. Urinary excretion of N-methylnicotinamide is decreased, and there are decreased liver and skeletal muscle concentrations of nicotinamide adenine dinucleotide and nicotinamide adenine dinucleotide phosphate. Uncorrected deficiencies lead to dehydration, emaciation, and death (Dann and Handler, 1941; Sarett, 1942; Schaefer et al., 1942; Handler, 1943; Smith et al., 1943; Layne and Carey, 1944; Efremov et al., 1954; Nelson et al., 1962; Belavady and Gopalan, 1965; Greengard et al., 1966; Madhavan et al., 1968; Manson and Carpenter, 1978).

Hypervitaminosis Niacin

High doses of nicotinic acid (but not nicotinamide) have been shown to cause vasodilatation and increased intracranial blood flow in humans. A cutaneous flush in dogs appeared within 10 minutes of intravenous injection of 1 to 100 mg nicotinic acid per kilogram of body weight (Pereira, 1967). Intravenous nicotinic acid also increases the flow of gastric secretions in dogs (Bailey et al., 1972). In rats, neutralized nicotinic acid injections produced a transient decline in plasma-free fatty acid concentrations possibly due to an inhibitory effect on norepinephrine-induced lipolysis (Pereira and Mears, 1971). Intravenous nicotinic acid administered to dogs prior to thermal trauma reduces plasma volume loss (Hilton and Wells, 1976). Although there are variable reports on the effect of nicotinic acid upon hypercholesterolemia in the dog (Grande and Amatuzio, 1960; Zanetti and Tennent, 1963), Grande (1966) established that nicotinic acid has a plasma cholesterol-depressing effect in normal dogs that depends upon the dose used and the initial cholesterol level.

Vitamin B_6

Vitamin B_6, usually in the form of pyridoxal phosphate and occasionally as the amine, acts as a cofactor for a large number of enzymes involved in various aspects of amino acid metabolism including aminotransferases (transaminases), decarboxylases, racemases, dehydratases, synthetases, and hydroxylases. Pyridoxal phosphate is required for the synthesis of γ-aminolevulinic acid, a precursor of heme. Hematological parameters have been the main criteria used to determine the requirement of the dog for vitamin B_6 rather than the more recent and sensitive indices of adequate status, based on analysis of plasma pyridoxal 5'-phosphate, urinary 4-pyridoxic acid, and urinary tryptophan metabolite excretion following a tryptophan load (Leklem and Reynolds, 1980).

Requirements

Fouts et al. (1938) showed that weaned puppies given a semipurified diet lacking vitamin B_6 developed a severe microcytic hypochromic anemia. This anemia could be overcome in adult dogs by oral administration of 60 µg per kilogram of body weight of crystalline pyridoxine isolated from natural sources (Fouts et al., 1939); or in puppies (McKibben et al., 1939–1940) and in adults (Borson and Mettier, 1940) by administration of the synthetic vitamin.

Michaud and Elvehjem (1944) quoted unpublished experiments in which growing dogs given 5 µg pyridoxine per kilogram of body weight died before evidence of anemia appeared, whereas 10 µg per kilogram gave fairly good growth, but not equal to that with 60 µg per kilogram. They suggested that the level of 10 µg per kilogram of body weight per day may be sufficient for maintenance.

Vitamin B_6 deficiency was induced in adult dogs given a semipurified diet by Street et al. (1941). Control dogs pair-fed to those given the deficient diet and supplemented with a pyridoxine concentrate lost less body weight and had normal hematology. The potency of the pyridoxine concentrate given was not determined, but a similarly prepared concentrate to that used would have supplied the equivalent of 5 µg pyridoxine per kilogram per day. This intake of vitamin B_6 was inadequate for a single ad libitum control dog, which required 10 µg pyridoxine per kilogram of body weight per day.

Besides blood dyscrasia, anorexia, and body weight loss, Street et al. (1941) described a range of pathological changes including ataxia, cardiac dilatation and hypertrophy, congestion of various tissues, and demyelination of peripheral nerves.

Axelrod et al. (1945) showed that following a tryptophan load, young vitamin B_6-deficient dogs excreted xanthurenic acid and kynurenine in their urine. Dogs supplemented with pyridoxine excreted kynurenine and kynurenic acid but no xanthurenic acid. They raised young dogs on vitamin B_6-deficient diets containing either high or moderate levels of protein (casein). Dogs given the high level exhibited a greater decline in hemoglobin concentration and more rapid appearance of clinical signs following tryptophan loading than dogs raised on moderate levels of protein.

The foregoing experimental data do not permit the derivation of a definitive requirement for either maintenance or growth of the dog. The requirement for maintenance appears to be at least 10 µg per kilogram of body weight per day, and for growth, greater than 10 µg per kilogram of body weight per day but probably less than 60 µg per kilogram of body weight per day. Values of 60 µg per kilogram per day for the growing dog and 22 µg per kilogram of body weight per day for the adult are suggested as the requirement. On a dietary basis these requirements are satisfied by 300 µg pyridoxine per 1,000 kcal ME. By comparison, recommended allowances for growth of the rat are 6.0 mg per kilogram of diet (NRC, 1978) and the pig 1.5 mg per kilogram of diet (NRC, 1979).

Pyridoxal in serum from dogs appears to be more heat-stable than in the serum from horse, rabbit, and man (Davis and Smith, 1975). A naturally occurring heat-stable vitamin B_6 antagonist (linatine) has been isolated from flaxseed (Klosterman et al., 1967). The presence of B_6 antagonists in ingredients used in dog foods does not appear to have been examined.

Signs Of Deficiency

Acute deficiency of vitamin B_6 in growing puppies may lead to sudden death without untoward clinical signs other than anorexia, slow growth, or body weight loss. Vitamin B_6 deficiency has been associated with microcytic hypochromic anemia, generalized convulsions, and elevated plasma iron concentration (Fouts et al., 1938, 1939; McKibbin et al., 1939–1940; Borson and Mettier, 1940; Street et al., 1941; McKibbin et al., 1942). Because of the involvement of pyridoxal phosphate in amino acid metabolism, altered metabolites from tryptophan have been observed in urine (Axelrod et al., 1945). Dietary deficiencies of vitamin B_6 or metabolic deficiencies induced by deoxypyridoxine have been shown to result in prolonged tolerances of skin grafts and renal transplants (Humphries et al., 1961; Fisher et al., 1963).

The toxicity of the tuberculostatic drug isoniazid to dogs has been alleviated by injections of pyridoxine (Chin et al., 1978).

Hypervitaminosis Vitamin B_6

The vitamins B_6 are not considered highly toxic and have been used in a relatively large dose (20 mg pyridoxine per kilogram of body weight intravenously) as an antidote to a rodenticide "Castrix" (Ullrich, 1967), and to protect against the toxic pressor effects of strophanthin K (Eremeev, 1968).

Folacin

Although dogs have been extensively used as a model for humans in the study of absorption of folic acid from the gut (Baugh et al., 1971, 1975; Bernstein et al., 1972, 1975), critical studies from which a requirement for folic acid can be derived are not available. Most of the early experiments on folic acid supplementation were undertaken before the isolation of vitamin B_{12} and before the interrelationships between folic acid and vitamin B_{12} were recognized. Krehl and Elvehjem (1945) proposed that folic acid may be synthesized in significant amounts by the intestinal bacteria and may contribute to the dog's requirement. Krehl and Elvehjem (1945) and Krehl et al. (1946) also demonstrated that dogs given niacin-deficient diets showed an enhanced response to nicotinic acid when the diet contained folic acid. Folic acid seemed to play a role in maintaining a more adequate blood picture.

While intestinal synthesis in dogs has been elegantly demonstrated by Bernstein et al. (1972, 1975), the extent to which it contributes to meeting body needs has not been quantified. Furthermore, data from other species indicate the contribution is likely to be dependent on the type of diet (Teply et al., 1947; Miller and Luckey, 1963; Klipstein and Samloff, 1966).

The jejunum is the preferential site of folic acid absorption (Hepner et al., 1968; Bernstein et al., 1972). Naturally occurring dietary folates are mostly in the form of polyglutamates, which are not absorbed intact into the circulation. Cleavage of the polyglutamate side chain to monoglutamate or diglutamate seems to occur in the interior of intestinal epithelial cells (Baugh et al., 1975). There is no evidence of significant intraluminal conjugase activity in mammals, nor is the enzyme found in isolated brush border fractions (Hoffbrand and Peters, 1969 a, b; Halsted et al., 1975). Polyglutamates are readily absorbed by the intestinal mucosal cells and are apparently cleaved by conjugases that occur in the lysosomal particles.

Afonsky (1954) reported weight loss and a progressive

decline in hemoglobin concentration in a dog given a semipurified diet. Subcutaneous injections of 15 µg of folic acid per kilogram of body weight restored hemoglobin concentration.

Sheffy (1964) depleted 4- to 5-week-old Beagle puppies of folic acid by feeding a casein-based diet containing sulfasuxidine and 0.11 mg vitamin B_{12} per kilogram of diet. After 9 weeks of depletion, all dogs developed erratic appetites and weight gains decreased, but there were no changes in concentration of hemoglobin. All dogs were inoculated with distemper and hepatitis antigens, and half the dogs were also given 27.5 µg folic acid per kilogram of body weight. Depleted dogs had delayed antibody production responses against both distemper and infectious hepatitis antigens. Antibodies were detected in depleted dogs supplemented with folic acid 8 days after challenge with antigen, whereas depleted dogs without folic acid did not show antibodies until 17 days after being challenged. Folic acid supplementation allowed resumption of normal growth. Sheffy suggested a requirement for folic acid of less than 1.2 µg per kilogram of body weight.

The folic acid requirement of dogs fed an adequate diet of nonpurified ingredients that does not contain bacteriostatic agents is probably met by microbial synthesis in the intestine. Diets inadequate in choline, methionine, and vitamin B_{12} may induce deficiencies because of their interaction with folic acid.

It is suggested that the requirement given by the National Research Council (1974) for dogs of 4 µg folic acid per kilogram of body weight for adults and 8 µg folic acid per kilogram of body weight for growing dogs be maintained. On a dietary basis, these quantities are supplied in diets containing 54 µg folic acid per 1,000 kcal ME.

Signs Of Deficiency

Folacin deficiency results in erratic appetite, decreased weight gain, watery exudate from the eyes, glossitis leukopenia, hypochromic anemia with a tendency to microcytosis, and decreased antibody response to infectious canine hepatitis and canine distemper virus (Krehl and Elvehjem, 1945; Afonsky, 1954; Sheffy, 1964).

In the metabolism of histidine in the folate-replete animal, the formimino group from formiminoglutamate is transferred to tetrahydrofolate. When there is a metabolic deficiency of folate the urinary excretion of formiminoglutamate is elevated. A clinical test for folate deficiency is the administration of a load of histidine and the measurement of formiminoglutamate in urine (Tabor and Wyngarden, 1958).

Hypervitaminosis Folacin

Although oral toxicity of folacin has not been described in the dog, Vogel et al. (1964) demonstrated inhibition of hepatic alcohol dehydrogenase in the dog by intravenous administration of 80 mg folic acid per kilogram of body weight 4 hours after intravenous ethanol infusion.

Biotin

Requirements

Spontaneous biotin deficiency occurs rarely in animals because biotin is well distributed among foodstuffs, and a good part, if not all, of the requirement for the vitamin is met by microbial synthesis in the gut (Murthy and Mistry, 1977). The deficiency can, however, be induced by the inclusion of unheated (raw) egg white in the diet. Raw egg white contains the protein avidin, which forms a stable and biologically inactive complex with biotin. One molecule of avidin binds four molecules of biotin (Green, 1963) so firmly that 15 min of steaming at 100°C released only 0 to 10 percent of the bound biotin (Wei and Wright, 1964). Steaming for 2 h at 100°C released 55 to 65 percent of the biotin, while autoclaving for 15 min at 120°C produced complete dissociation. Uncombined avidin was found to be relatively heat-labile.

Shen et al. (1977) took 18-day-old Beagle puppies and divided them into two groups. One group was force-fed raw egg white along with a basal diet without biotin. The other group received an equal amount of heated egg white with the basal diet containing biotin. Within 10 days the group fed the raw egg white showed a significant reduction in activities of pyruvate and propionyl CoA carboxylases in liver and kidney homogenates. However, activities of these enzymes in heart and brain were less affected, which is consistent with data from rats and chicks.

Intestinally active antibiotics or sulfa drugs that inhibit microbial synthesis of biotin may also be expected to increase the need for biotin in the diet. Greve (1963) fed diets containing spray-dried egg white and sulfaguanidine to dogs and produced evidence of biotin deficiency. Assay of the urine of these dogs revealed less than 0.05 pg biotin per milliliter, as compared to "normal" dog urine that contained 7 to 13 pg biotin per milliliter. Unfortunately, the biotin concentration of the diet was not reported.

Incomplete availability of biotin to chicks has been reported by Wagstaff et al. (1961) and Anderson and Warnicke (1970). There have been reports of apparent

biotin deficiencies in poultry (Johnson, 1967; Marusick et al., 1970) and swine (Adams et al., 1967) fed practical diets. While a definitive requirement for biotin for the dog cannot be stated, the inclusion in a diet of 30 µg biotin per 1,000 kcal ME may be prudent as a safeguard against a possible deficiency. This level is similar to that suggested for the growing pig (National Research Council, 1979).

Signs Of Deficiency

No adequate descriptions of biotin deficiency in the dog are available. Greve (1963) reported scurfy skin (due to hyperkeratosis of the superficial and follicular epithelia) and a marked decline in urinary biotin concentration. No alopecia or achromotrichia was noted.

Vitamin B_{12}

Requirements

In mammals vitamin B_{12} is required as a factor for the transmethylation of 5-methyl tetrahydrofolate to homocysteine and the formation of tetrahydrofolate and methionine. Vitamin B_{12} also participates as a coenzyme for the conversion of methylmalonyl-CoA to succinyl-CoA (Weissbach and Taylor, 1970). It has been hypothesized that a metabolic deficiency of vitamin B_{12} results in folic acid being "trapped" as methyl tetrahydrofolate and that it depletes 5- to 10-methylene tetrahydrofolate required for thymidylate synthesis and therefore DNA biosynthesis (Herbert and Zalusky, 1962; Mertz et al., 1968; Butterworth and Krumdieck, 1975). This hypothesis explains much, but not all, of the interaction of vitamin B_{12} in hematopoiesis (Chanarin et al., 1981).

The dog has been used intensively as a model in the study of the mechanism of vitamin B_{12} absorption (Reizenstein et al., 1960; Fleming et al., 1962; Hermann et al., 1964; Bryant and Stafford, 1965; Gazet and McColl, 1967; Weisberg et al., 1968; Lavrova, 1969; Taylor et al., 1969; Yamaguchi et al., 1969a,b; Weisberg and Rhodin, 1970), plasma transport of vitamin B_{12} (Markelova, 1960; Rappazzo and Hall, 1972; Sonneborn et al., 1972; Hall and Rappazzo, 1974), and tissue vitamin B_{12} distribution (Cooperman et al., 1960; Woods et al., 1960; Skeggs et al., 1963; Rosenblum et al., 1963); however, no definitive data on dietary vitamin B_{12} requirements for the dog are available. Arnrich et al. (1952) fed a semipurified diet containing 20 percent vitamin-free casein without supplemental vitamin B_{12} to weanling Cocker Spaniel puppies for 20 weeks. No anemia developed and gains were satisfactory, although a supplement of 50 µg vitamin B_{12} per kilogram of diet appeared to increase gains (primarily fat). Likewise, urinary vitamin B_{12} excretion has been studied in the dog (Nelp et al., 1964; Coppi et al., 1970; and Silverman, 1979), but no data were presented that would provide a guide to vitamin B_{12} status in relation to vitamin B_{12} intake.

In rats the toxicity of methionine can be reduced by the inclusion of vitamin B_{12} in the diet at about 3 times the requirement (Areshkina et al., 1973). The requirement for dietary vitamin B_{12} varies with the dietary content of choline, methionine, and folic acid.

In the absence of information on the requirement of dogs for vitamin B_{12}, it is suggested that the recommended requirement for the baby pig (NRC, 1979) of 0.5 µg vitamin B_{12} per kilogram of body weight be adopted for maintenance of the adult dog and twice this level (1.0 µg per kilogram of body weight) be used for growth of puppies. These amounts would be supplied by 26 µg vitamin B_{12} per kilogram of dry diet containing 3.67 kcal ME per gram, or 7 µg per 1,000 kcal dietary ME. No data are available to make a recommendation of the requirement during pregnancy and lactation. The requirement for bitches fed diets based on soy protein may be greater during pregnancy than the above recommendation, as Woodward and Newberne (1966) reported hydrocephaly in rat pups from female rats fed a soy-based diet. This condition was prevented by supplementation of the diet by 50 µg per kilogram of vitamin B_{12} recommended by NRC (1978) for the rat.

Signs Of Deficiency

Uncomplicated vitamin B_{12} deficiency has not been described in the dog. Lavrova (1969) reported an anemia in dogs with an internal biliary fistula, which may have been associated with a failure in vitamin B_{12} absorption. The anemia was generally macrocytic hypochromic, macrocytic normochromic, normocytic hypochromic, or normocytic normochromic in type. The bone marrow erythropoietic centers appeared hypoplastic. Serum and liver vitamin B_{12} concentrations were decreased.

In vitamin B_{12} deficiency there is an enhanced urinary excretion of methylmalonic acid. Vitamin B_{12} is required as a coenzyme for the isomerization of methylmalonyl coenzyme A to succinyl coenzyme A. A clinical test for vitamin B_{12} deficiency is to load the animal with a precursor of methylmalonic acid (e.g., valine) and measure urinary excretion of methylmalonic acid (Williams et al., 1969; Chanarin et al., 1973).

Hypervitaminosis B_{12}

Although frank vitamin B_{12} toxicity has not been described in the dog, Pshonik and Gribanov (1961) noted

disturbances of reflex activity in the form of reduction in size of vascular conditioned reflexes, exaggeration of unconditioned reflexes, and intensification of successive inhibition when vitamin B_{12} was injected subcutaneously in doses of 2 to 33 µg per kilogram of body weight.

Choline

Requirements

The importance of choline in the nutrition of the dog was suggested by its lipotrophic action on the liver of the depancreatized dog, according to Best et al. (1933). The dietary requirement for choline is markedly affected by the concentration of other methyl donors in the diet, of which the most important is methionine. High levels of methionine in the diet will obviate the need for dietary choline. Schaefer et al. (1941) pointed out that a number of workers have found that dietary casein concentrations of 40 percent or more tend to obviate the need for dietary choline. In the studies of Schaefer et al. (1941) themselves, puppies receiving a 19 percent casein diet became choline-deficient. Controls receiving 50 mg choline per kilogram of body weight per day grew satisfactorily over the 37-day experimental period. On a 15 percent casein diet, Fouts (1943) found that 10 or 20 mg choline per kilogram of body weight would not prevent or cure the deficiency state in puppies, while a 100-mg level would. When 41 percent casein was provided, no choline deficiency nor any response to supplemental choline could be shown.

McKibbin et al. (1944) fed a diet containing natural proteins low in sulfur amino acids (10 percent protein from peanut flour plus 10 percent casein) to puppies and concluded that choline requirements were probably not greater than 1,000 mg per kilogram of diet or 50 mg per kilogram of body weight per day.

Complex interactions occur involving single carbon transfer by choline, methionine, folate, and vitamin B_{12}. Choline requirements can only be determined when the diet contains a minimal, but adequate level of methionine and adequate levels of folate and vitamin B_{12}. When the previous experiments were undertaken the minimal requirement of the dog for methionine was not known, and isolated sources of vitamin B_{12} and folic acid were not available. Furthermore, for rats the dietary requirement of choline is influenced by the lipid content of the diet, the chain length and degree of saturation of the fatty acids, and the total caloric content of the diet (Best et al., 1954; Salmon and Newberne, 1962; Zachi et al., 1965; Patek et al., 1966).

It is concluded that the choline requirements for adult maintenance may be met by 25 mg per kilogram of body weight per day and those for growth of puppies by 50 mg per kilogram of body weight per day. Diets containing 340 mg choline per 1,000 kcal ME will supply these quantities of choline when fed to adult or growing dogs.

Signs Of Deficiency

Dutra and McKibbin (1945) described the pathology of "uncomplicated" choline deficiency in young puppies. They reported fatty metamorphosis of the liver and atrophic changes of the thymus. The morphologic changes in the liver, correlated with impairment in liver function as measured by delayed bromsulfalein elimination, were reported by McKibbin et al. (1944, 1945) and Anonymous (1945a,b). Plasma phosphatase activity and blood prothrombin times were also elevated in the choline-deficient puppies.

Choline-deficient dogs with fatty livers show an increased rate of hepatic phospholipid synthesis following choline supplementation (Di Luzio and Zilversmit, 1959).

Excess Dietary Choline

Acara and Rennick (1973) reported renal clearance studies on dogs that indicated that only one-thirtieth of the choline filtered at the glomerulus was excreted in the urine, suggesting active tubular reabsorption. When exogenous choline was infused intravenously, choline renal clearance exceeded glomerular filtration rate, indicating active tubular excretion. Solomon (1966) has reported that infusion of choline results in urinary alkalinization, primarily from an increased urinary bicarbonate output. At the same time, there is a decrease in ammonia output.

Vitamin C

Innes (1931) demonstrated that the dog, unlike the guinea pig, was independent of an exogenous supply of vitamin C. Puppies fed a diet devoid of vitamin C for 147 to 154 days showed neither growth impairment nor lesions of bones or teeth, although the same diet killed guinea pigs within 25 days with severe signs of scurvy. Furthermore, the livers of dogs on the deficient diet contained the vitamin in sufficient amounts to prevent the onset of scurvy in guinea pigs, indicating that the dog can synthesize its own vitamin C. Naismith (1958) showed that this synthetic ability is present in puppies during the first weeks of postnatal life. Litters were divided: some puppies were left with the bitch; others were fed a synthetic diet minus vitamin C, or plus vitamin C. No significant differences in blood ascorbic acid concentration were evident, regardless of treatment. Naismith and Pellet (1960) reported that the concentration

of ascorbic acid in the milk from bitches is approximately 4 times that of the blood. The comparative rates of hepatic synthesis of ascorbic acids in dogs and cats appear to be lower than that in ruminants, rodents, and lagomorphs (Chatterjee et al., 1975).

Despite the above evidence, a number of clinical case history reports (Garlick, 1946; Meier et al., 1957; Ditchfield and Phillipson, 1960; Holmes, 1962; Hunt, 1962; Sadek, 1962; Bendefy, 1965; Belfield, 1967, 1976; Vaananen and Wikman, 1979) have been published purporting to describe scurvy in the dog with or without concomitant hip dysplasia or osteodystrophy. None of these reports included observations on control untreated animals. Also, the effect on the dog of pharmacological doses of ascorbic acid (e.g., 3,000 mg intravenously per day) may be quite distinct from its nutritional contribution. Teare et al. (1980) reported that 600 mg of ascorbic acid twice daily only aggravated the skeletal disease induced by overfeeding protein, energy, and calcium to Labrador Retriever puppies.

In addition, vitamin C has been proposed as a prophylactic agent against canine distemper (Belfield, 1967; Leveque, 1969), and some veterinary practitioners apparently advocate vitamin C for the treatment of kennel cough. Sheffy (1972) conducted some carefully controlled studies concerned with these issues and established that exogenous vitamin C was of no benefit in alleviating clinical signs of illness, mortality, or gross or microscopic pathology associated with experimentally produced canine herpes virus infection, kennel cough, or infectious canine hepatitis. In addition, as determined by measuring blood ascorbic acid levels, the latter disease did not affect vitamin C synthesis. Other data on blood and urine ascorbic acid values in the dog have been published by Majumdar et al. (1964), Kleit et al. (1965), Crilly et al. (1976), and Robinson et al. (1979). Csaba and Toth (1966), in controlled studies, established that ascorbic acid given before antigen challenge in dogs has no protective action against anaphylactic shock and does not influence histamine release. Weintraub and Griner (1974) found that high doses of ascorbic acid had no effect on biological half-life or kinetics of Warfarin-induced hypoprothrombinemia.

It is concluded that there is no adequate evidence to justify recommendation of routine vitamin C additions to the diet of the normal dog. However, dogs with hepatic dysfunction may have lowered plasma concentrations of ascorbic acid (Strombeck et al., 1983). Whether lower plasma concentrations are of clinical significance remains to be demonstrated.

3

Water

Water is undoubtedly the most important nutrient; it is vital to the functioning of all living cells. The body of the adult dog contains about 60 percent water (Gaebler and Choitz, 1964), and this proportion is even higher in the puppy. The body has a limited capacity to store water, and water deprivation causes death much more quickly than does deprivation of food.

Dogs obtain water in liquid form, from food, and as a consequence of oxidation of hydrogen during metabolism, the latter known as metabolic water. Oxidation of 100 g protein yields about 40 g metabolic water; 100 g carbohydrate, about 55 g metabolic water; and 100 g fat, about 107 g metabolic water. In general, about 10 to 16 g metabolic water are produced for each 100 kcal of energy metabolized. Thus, a dog consuming 2,000 kcal ME per day may derive 200 to 320 g water from body metabolism.

Water gain (whether from liquid water, food, or metabolic water) is balanced by water loss, principally through the urine, lungs, skin, and feces. In the lactating bitch, a considerable amount of water is secreted in the milk.

Under normal conditions, the body water content is remarkably constant. Therefore, water intake plus metabolic water must balance water outgo. The dog can cope with a large fluid intake by virtue of a readily adjustable urine volume, but the unsalvageable water losses of the body dictate the minimum intake. In the growing puppy and the idle adult, voluntary water intake will usually range from 2 to 3 times the dry matter intake. During lactation, hot weather, or severe exertion, water intakes may reach 4 or more times dry matter intake.

The individual dog's requirement for drinking water is self-regulated, depending on factors such as type of food, environmental temperature, amount of exercise, physiological state, and temperament. The need can be met by permitting free access to water at all times or by offering water at least 3 times a day. A dog should not be allowed large amounts of cold water immediately following violent exercise, because of the dangers of water intoxication. When the total ration consists of soft moist foods, which contain an intermediate amount of water, or of dry-type dog foods, water is a necessary adjunct to feeding.

4

Composition of Ingredients of Dog Foods

Specific nutrient content of some feed ingredients commonly used in dog foods have been extracted from *United States-Canadian Tables of Feed Composition* (NRC, 1982) and compiled by the International Feedstuffs Institute, Logan, Utah, as presented in Tables 6, 7, and 8. These may be used as an aid in compounding practical foods for dogs.

Alternately, for preparation of "home cooked" formulas or for formulation of therapeutic diets or supplements, the user is directed to food composition tables published in the USDA Agriculture Handbook No. 456, *Nutritive Value of American Foods (In Common Units)*, by Catherine F. Adams (USDA, 1975).

Nutrient concentrations are organized in Tables 6, 7, and 8 as follows: fat and fatty acid composition (Table 6), composition excluding amino acids (Table 7), and amino acid composition (Table 8). All data are expressed on a 100 percent dry matter basis.

INTERNATIONAL NOMENCLATURE

In Tables 6, 7, and 8 and in the *United States-Canadian Tables of Feed Composition*, the Feed Name Descriptions are based on a scheme proposed by Harris et al. (1980, 1981). The names are designed to give a qualitative description of each product, where such information is available and pertinent. A complete name consists of as many as six facets, separated by commas and written in linear form. The facets are these:

- Origin, consisting of scientific name (genus, species, variety); common name (generic name, breed or kind, strain or chemical formula)
- Part fed to animals as affected by process(es)
- Process(es) and treatment(s) to which the part has been subjected
- Stage of maturity or development
- Cutting (applicable to forages)
- Grade (official grades with guarantees)

INTERNATIONAL FEED CLASSES

Feeds are grouped into eight classes on the basis of their composition and their use in formulating diets. (The first digit of each hyphenated set of numbers in the International Feed Number column of Tables 6, 7, and 8 is the feed class.) The numbers and the classes they designate are as follows:

Code	
1.	Dry forages and roughages
2.	Pasture, range plants, and forages fed fresh
3.	Silages
4.	Energy feeds
5.	Protein supplements
6.	Mineral supplements
7.	Vitamin supplements
8.	Additives

Feeds on a dry basis that contain more than 18 percent crude fiber or 35 percent cell wall are classified as forages or roughages; feeds that contain less than 20 percent protein and less than 18 percent crude fiber or less than 35 percent cell wall are classified as energy feeds; and those that contain 20 percent or more protein are classified as protein supplements.

INTERNATIONAL FEED NUMBER (IFN)

Each international feed name is assigned a five-digit international feed number (IFN) for identification and

computer manipulation. The IFN is particularly useful as a tag to recall nutrient data for calculating diets. As indicated above, the feed class number has been entered in front of the international feed number (see Tables 6, 7, and 8).

The following table shows how three feeds are described:

Components of Name	Feed No. 1	Feed No. 2	Feed No. 3
Origin (or parent material)	Soybean	Alfalfa	Wheat
Species variety or kind	—	—	soft white winter
Part eaten	seeds	—	grain
Process(es) and treatment(s) to which product has been subjected	meal solvent extracted	meal dehydrated	—
Stage of maturity	—	—	—
Grade or quality designations	—	17% protein	—
Classification; first digit in International feed number (IFN)	(5) protein supplements	(1) forages and roughages	(4) energy feeds
IFN	5-04-604	1-00-023	4-05-337

Thus, the names of the three feeds are written as follows:

No. 1: Soybean, seeds, meal solvent extracted

No. 2: Alfalfa, meal dehydrated, 17% protein

No. 3: Wheat, soft white winter, grain

CAROTENE CONVERSION

International standards for vitamin A activity as related to vitamin A and β-carotene are as follows:

1 IU vitamin A = 1 USP unit

= vitamin A activity of 0.300 µg crystalline all-*trans* retinol (vitamin A alcohol), which corresponds to 0.344 µg all-*trans* retinyl acetate (vitamin A acetate) or 0.550 µg all-*trans* retinyl palmitate (vitamin A palmitate).

β-carotene is the standard for provitamin A.

1 IU vitamin A = 0.6 µg all-*trans* β-carotene.

1 mg-carotene = 1,667 IU vitamin A.

International standards for vitamin A are based on the utilization of vitamin A and β-carotene by the rat. Since it is not well established that dogs convert carotene to vitamin A in the same ratio as rats, it is suggested that consideration be given to reducing carotene conversion to vitamin A in Table 7 as follows:

1 mg provitamin A (carotene) = 833 IU vitamin A activity for the dog.

DATA

The analytical data are expressed in the metric system and are shown on a dry basis. See Table 9 (p. 62) for weight-unit conversion factors.

Analytical data may differ in the various NRC reports because the data are updated for each report. The feed names may also differ as feeds are more precisely described or as official definitions change. However, if the feed is the same, the international feed number will remain the same.

METABOLIZABLE ENERGY (ME)

Since ME content of food ingredients listed in Table 7 have not been determined by studies in dogs, no values were included; instead approximated values have to be calculated. For this purpose, the Atwater factors of 4-9-4 for crude protein (CP), ether extract (EE), and nitrogen-free extract (NFE), respectively, commonly used, are inappropriate. These were developed for and are more applicable to foods consumed by humans and not to combinations of ingredients used in dog foods. Their use here would overestimate the ME values of dog foods, since their derivation was based on assumed digestibility coefficients of 91, 96, and 96 percent, respectively. The studies of Kendall et al. (1982) strongly support this conclusion. Their study included data from 106 digestibility trials of commercial foods including 42 canned, 24 intermediate moisture, and 40 dry-type dog foods. The overall mean apparent digestibility reported for CP, acid ether extract (AEE), and NFE was 81, 85, and 79 percent, respectively. Energy losses in urine were not measured, nor was there a separation of fiber from the NFE term. However, acid ether extraction was used, assuring maximal measurement of fat content. The above were both positive and negative factors contributing to average coefficients suggested in the NRC (1974) report for calculation of ME values for commercial dog foods and/or their individual constituents.

For this revision the Subcommittee on Dog Nutrition suggests average coefficients of 80, 90, and 85 percent, respectively, for CP, AEE, and NFE, and 0.0 for fiber. Users are cautioned that these average values may result in underestimating ME content of low-fiber, low-connective-tissue-containing meat and animal by-product foods and in overestimating foods primarily from plant and cereal sources that contain elevated fiber contents.

5

Formulated Diets for Dogs

Dogs require specific nutrients, not specific feedstuffs. This fact and the remarkable adaptability of the dog have led to the successful use of commercial diets that differ widely in their ingredient composition. Commercial dog foods are of the three basic types, as described below, although foods with moisture levels ranging from 5 to 78 percent are common in the marketplace.

DRY DOG FOODS

Low in moisture content (usually about 10 to 12 percent), dry dog foods commonly contain whole or dehulled cereal grains (e.g., corn, wheat, oats, barley), cereal by-products (e.g., wheat middlings, wheat germ meal, corn gluten meal), soybean products (e.g., soybean meal, soy grits), animal products (e.g., meat meal, meat and bone meal, meat by-products, poultry by-products), milk products (e.g., dried skimmed milk, dried whey), fats and oils (e.g., animal fat), and mineral and vitamin supplements. Crude fat content usually ranges from 5.0 to 12.5 percent on a dry basis. The higher fat levels (and improved palatability) may be achieved by spraying a liquefied fat on the surface of pelleted or extruded products. Dry-type foods may be marketed as meals, pellets, biscuits, kibbles (broken biscuits), or expanded (extruded) products. Processing methods should include sufficient heat to partially dextrinize starch for improved digestibility.

SEMIMOIST DOG FOODS

Moderate in moisture content (usually 25 to 30 percent), semimoist dog foods are protected against spoilage without refrigeration by their content of sucrose, propylene glycol, and sorbates. They also commonly contain animal products (e.g., meat, meat by-products, meat digests), milk products (e.g., dried whey, cheese rind), fats and oils (e.g., animal fat), soybean products (e.g., soybean meal, soy flour), carboxy-methylcellulose, and mineral and vitamin supplements. They may be shaped into "patties" of a size convenient for feeding or packaged as simulated meat chunks.

Most recently a modification of semimoist foods, namely, "soft-dry" foods, have been introduced to the market. These foods generally may contain lower proportions of fresh meat and meat by-products and depend for preservation on a low pH (4.2 ±) with the use of phosphoric acid or other acids, coupled with mold inhibitors.

CANNED DOG FOODS

High in moisture content (usually 74 to 78 percent), canned dog foods are commonly formulated to be nutritionally complete. The composition of these foods varies from premium foods containing high proportions of meat and/or meat by-products to formulations with low meat and meat by-product content. The latter foods are similar in composition to dry food to which water has been added prior to canning. A meat-based formulation may contain from 25 to 75 percent of meat and meat by-products. The latter products are usually designated as "dinners." Most such canned dinners also contain textured soy protein simulating the appearance of meat. These products have almost totally replaced earlier fortified "all-meat" foods. Typically "dinners" or "all-meat" foods on a dry matter basis are high-nutrient-density foods. Higher energy density dictates higher concentration of protein, vitamins, and minerals. Although these foods are designed to be fed alone as complete

and balanced diets, they are commonly used as supplements to improve acceptability of dry foods for feeding during more stressful situations. Although additions of canned food, milk, meat, eggs, or broths invariably improve palatability of dry foods, the nutritional value of properly balanced dry foods may not always be enhanced.

Formulas for examples of products representing typical dry, semimoist, and canned foods are presented in Table 11 (see p. 62). These examples are intended only as illustrations of formulations from commonly available feed sources.

Formulation for semipurified foods as commonly used in nutrition research may be found in any number of publications relating to vitamin and/or amino acid requirements of dogs (see References). Such diets frequently contain crystalline amino acids, casein, or isolated soy protein as the sole protein source, depending upon the nature of research being conducted.

Tables

TABLE 1 Minimum Nutrient Requirements of Dogs for Growth and Maintenance (amounts per kg of body weight per day)[a]

Nutrient	Unit	Growth[b]	Adult Maintenance[c]
Fat	g	2.7	1.0
Linoleic acid	mg	540	200
Protein[d]			
Arginine	mg	274	21
Histidine	mg	98	22
Isoleucine	mg	196	48
Leucine	mg	318	84
Lysine	mg	280	50
Methionine-cystine	mg	212	30
Phenylalanine-tyrosine	mg	390	86
Threonine	mg	254	44
Tryptophan	mg	82	13
Valine	mg	210	60
Dispensable amino acids	mg	3,414	1,266
Minerals			
Calcium	mg	320	119
Phosphorus	mg	240	89
Potassium	mg	240	89
Sodium	mg	30	11
Chloride	mg	46	17
Magnesium	mg	22	8.2
Iron	mg	1.74	0.65
Copper	mg	0.16	0.06
Manganese	mg	0.28	0.10
Zinc	mg	1.94	0.72
Iodine	mg	0.032	0.012
Selenium	μg	6.0	2.2
Vitamins			
A	IU	202	75
D	IU	22	8
E[e]	IU	1.2	0.5
K[f]			
Thiamin	μg	54	20
Riboflavin	μg	100	50
Pantothenic acid	μg	400	200
Niacin	μg	450	225
Pyridoxine	μg	60	22
Folic acid	μg	8	4
Biotin[f]			
B_{12}	μg	1.0	0.5
Choline	mg	50	25

[a] Needs for other physiological states have not been determined.
[b] Average 3-kg-BW growing Beagle puppy consuming 600 kcal ME/day.
[c] Average 10-kg-BW adult dog consuming 742 kcal ME/day.
[d] Quantity sufficient to supply minimum amounts of available indispensable and dispensable amino acids specified below.
[e] Requirement depends on intake of PUFA and other antioxidants. A fivefold increase may be required under conditions of high PUFA intake.
[f] Dogs have a metabolic requirement, but a dietary requirement was not demonstrated when natural ingredients were fed.

TABLE 2 Required Minimum Concentrations of Available Nutrients in Dog Food Formulated for Growth

Nutrient	Per 1,000 kcal ME	Dry Basis (3.67 kcal ME/g)
Protein[a]		
Indispensable amino acids		
Arginine	1.37 g	0.50%
Histidine	0.49 g	0.18%
Isoleucine	0.98 g	0.36%
Leucine	1.59 g	0.58%
Lysine	1.40 g	0.51%
Methionine-cystine	1.06 g	0.39%
Phenylalanine-tyrosine	1.95 g	0.72%
Threonine	1.27 g	0.47%
Tryptophan	0.41 g	0.15%
Valine	1.05 g	0.39%
Dispensable amino acids	17.07 g	6.26%
Fat	13.6 g	5.0%
Linoleic acid	2.7 g	1.0%
Minerals		
Calcium	1.6 g	0.59%
Phosphorus	1.2 g	0.44%
Potassium	1.2 g	0.44%
Sodium	0.15 g	0.06%
Chloride	0.23 g	0.09%
Magnesium	0.11 g	0.04%
Iron	8.7 mg	31.9 mg/kg
Copper	0.8 mg	2.9 mg/kg
Manganese	1.4 mg	5.1 mg/kg
Zinc[b]	9.7 mg	35.6 mg/kg
Iodine	0.16 mg	0.59 mg/kg
Selenium	0.03 mg	0.11 mg/kg
Vitamins		
A	1,011 IU	3,710 IU/kg
D	110 IU	404 IU/kg
E[c]	6.1 IU	22 IU/kg
K[d]	—	—
Thiamin[e]	0.27 mg	1.0 mg/kg
Riboflavin	0.68 mg	2.5 mg/kg
Pantothenic acid	2.7 mg	9.9 mg/kg
Niacin	3 mg	11.0 mg/kg
Pyridoxine	0.3 mg	1.1 mg/kg
Folic acid	0.054 mg	0.2 mg/kg
Biotin[d]	—	—
Vitamin B_{12}	7 μg	26 μg/kg
Choline	340 mg	1.25 g/kg

[a] Quantities sufficient to supply the minimum amounts of available indispensable and dispensable amino acids as specified below. Compounding practical foods from natural ingredients (protein digestibility ± 70%) may require quantities representing an increase of 40% or greater than the sum of the amino acids listed below, depending upon ingredients used and processing procedures.
[b] In commercial foods with natural ingredients resulting in elevated calcium and phytate content, borderline deficiencies were reported from feeding foods with less than 90 mg zinc per kg (Sanecki et al., 1982).
[c] A fivefold increase may be required for foods of high PUFA content.
[d] Dogs have a metabolic requirement, but a dietary requirement was not demonstrated when foods from natural ingredients were fed.
[e] Overages must be considered to cover losses in processing and storage.

TABLE 3 Factors for Consideration in Formulation of Dog Foods From Natural Ingredientsa

Nutrient	Factors for Consideration
Fat	Degree of unsaturation, antioxidants, vitamin E
Carbohydrate	Fiber, lactose, reducing sugars, processing, stage-of-life cycle
Protein	Energy content, digestibility, amino acid balance, processing, antinutrients, antitryptic factors
Amino acids	Availability; heat treatment in presence of reducing sugars reduces availability, especially of lysine; requirement for individual amino acids increases with increased dietary nitrogen.
Minerals	Ratios, source, availability
Calcium	Phytates, ligands, vitamin D
Phosphorus	Phytates, calcium, plant-animal
Sodium, potassium, chloride	High availability
Zinc	Phytates, calcium, plant-animal, fiber
Copper	Phytates, zinc
Iron	Source, availability, plant-animal
Vitamins	Processing, lipid content, source
A	Oxidation, toxicity
D	Toxicity, calcium level
E	PUFA, selenium
B_1	Losses in processing and storage, product pH, storage time and temperature, thiaminases
B_2	UV light
B_6 (Pyridoxine)	Protein level in diet
Niacin	Tryptophan, low availability of plant sources
Folate	Processing losses
B_{12}	Plant versus animal proteins
Choline	Methionine, folate, vitamin B_{12}, availability, fat

[a] See text discussion for details relative to individual nutrients.

TABLE 4 Calculated Metabolizable Protein and Metabolizable Energy Requirements of Dogs in Various Physiological Statesa

Physiological State	Protein Requirement (g metabolizable protein $W_{kg}^{0.67}$ per day)	Metabolizable Energy Requirement (kcal per $W_{kg}^{0.67}$ per day)
Weaning		
Start (3 weeks)	8.1	400
Finish (6 weeks)	6.5	375
Early growth	6.0	353
Half grown	3.8	225
Adult (average)	1.5	132–159
Pregnancy, late	5.7	225
Lactation	12.4	560

[a] Adapted from Payne (1965). Calculated metabolizable protein equals food nitrogen minus fecal and urine N (retained N) × 6.25. Calculated metabolizable energy estimates were based on 4 kcal/g of dietary carbohydrate and protein and 9 kcal/g of dietary fat. These requirements are presumed to apply in a thermoneutral environment at moderate levels of activity.

TABLE 5 Recommended Energy Needs of Adult Dogs at Maintenance (kcal ME/day)a

Body Weight (kg)	NRC (1974) (132 $W_{kg}^{0.75}$)	Thonney (1983) (100 $W_{kg}^{0.88}$)[b]	Thonney (1983) (144 + 62.2 W_{kg})[b]
1	132	100	207
3	301	262	331
5	441	412	455
10	742	758	766
20	1,248	1,396	1,388
30	1,692	1,995	2,010
40[c]	2,099	2,569	2,632
50[c]	2,482	3,127	3,254
60[c]	2,846	3,671	3,876

[a] Intended to apply in a thermoneutral environment at moderate activity.

[b] The contributions by Professor M. L. Thonney, Cornell University, to the development of these data are gratefully acknowledged, as is the assistance of Dr. C. A. Banta, Allen Products; Dr. Hanson Lee, Quaker Oats; and Dr. Lloyd Miller, Carnation, for supplying data on individual dogs.

[c] Data based on feeding records are needed for dogs in these weight categories.

TABLE 6 Fat and Fatty Acid Composition of Feed Ingredients; Data Expressed on a Dry Basis (100% Dry Matter)

Entry Number	Feed Name Description	International Feed Number	Dry Matter (%)	Ether Extract (%)	Saturated Fat[a] (%)	Unsaturated Fat[a] (%)	Linoleic Acid (%)	Arachidonic Acid (%)
	ALFALFA *Medicago sativa*							
01	meal dehydrated, 17% protein	1-00-023	92.0	2.5	0.3	0.7	0.43	—
02	leaves, meal dehydrated	1-00-137	93.0	3.1	0.3	0.9	0.56	—
	ANIMAL							
	tallow—see FATS AND OILS							
	BARLEY *Hordeum vulgare*							
03	grain	5-00-549	89.0	2.1	0.6	1.4	0.27	—
	COCONUT *Cocos nucifera*							
	oil—see FATS AND OILS							
	CORN, DENT YELLOW *Zea mays indentata*							
04	grain	4-02-935	89.0	4.5	0.9	3.7	2.05	—
05	distillers solubles, dehydrated	5-28-237	93.0	9.5	2.0	7.5	4.80	—
06	gluten, meal	5-28-241	91.0	8.4	1.5	6.8	4.21	—
07	grits by-product (hominy feed)	4-03-011	90.0	7.2	1.2	6.1	3.71	—
	CRAB *Callinectes sapidus*							
08	process residue, meal (crab meal)	5-01-663	92.0	1.9	0.5	1.3	0.35	—
	FATS AND OILS							
09	bran oil, rice	4-14-504	100.0	100.0	18.5	81.1	36.50	—
10	fat, swine (lard)	4-04-790	100.0	100.0	35.9	64.1	18.30	0.3–1.0
11	offal fat, poultry	4-09-319	100.0	100.0	39.1	60.9	22.30	0.5–1.0
12	oil, coconut	4-09-320	100.0	100.0	90.3	9.7	1.10	—
13	oil, corn	4-07-882	100.0	100.0	12.3	87.7	55.40	—
14	oil, fish, menhaden	7-08-049	100.0	100.0	40.0	60.0	2.70	20.0–25.0
15	oil, flax, common (linseed oil)	4-14-502	100.0	100.0	8.2	91.8	13.90	—
16	oil, pecan	4-20-525	100.0	100.0	6.9	93.1	30.60	—
17	oil, safflower	4-20-526	100.0	100.0	10.5	89.5	72.70	—
18	tallow, animal	4-08-127	100.0	100.0	47.6	52.4	4.30	0.0–0.2
	FISH							
19	solubles, condensed	5-01-969	51.0	12.8	5.7	7.1	0.39	—
	FISH, MENHADEN *Brevoortia tyrannus*							
20	meal mechanically extracted	5-02-009	92.0	8.4	4.8	3.6	0.12	—
	oil—see FATS AND OILS							
	FLAX, COMMON *Linum usitatissimum*							
21	meal solvent extracted (linseed meal)	5-02-048	91.0	1.9	0.4	1.5	0.41	—
	oil (linseed oil)—see FATS AND OILS							
	MEAT							
22	meal rendered	5-00-385	94.0	10.6	5.00	5.70	0.36	—
23	with blood, meal rendered (tankage)	5-00-386	92.0	8.8	4.40	4.50	0.30	—
	MILK *Bos taurus*							
24	skimmed dehydrated (cattle)	5-01-175	94.0	1.0	0.40	0.60	0.01	—
	OATS *Avena sativa*							
25	grain	4-03-309	89.0	5.1	1.20	3.90	1.67	—
	PEANUT *Arachis hypogaea*							
26	kernels, meal mechanically extracted (peanut meal)	5-03-649	92.0	7.3	1.70	5.50	1.36	—
	PECAN *Caya illinoensis*							
	oil—see FATS AND OILS							
	POULTRY							
27	by-product, meal rendered (viscera with feet with heads)	5-03-798	93.0	12.5	4.50	8.00	1.98	—
	offal fat—see FATS AND OILS							
	RICE *Oryza sativa*							
	bran oil—see FATS AND OILS							
	SAFFLOWER *Carthamus tinctorius*							
	oil—see FATS AND OILS							
	SKIM MILK—SEE MILK							
	SORGHUM *Sorghum bicolor*							
28	grain	4-04-383	90.0	3.2	0.70	2.50	1.20	—

Entry Number	Feed Name Description	International Feed Number	Dry Matter (%)	Ether Extract (%)	Saturated Fat[a] (%)	Unsaturated Fat[a] (%)	Linoleic Acid (%)	Arachidonic Acid (%)
	SOYBEAN *Glycine max*							
29	flour by-product (soybean mill feed)	4-04-594	90.0	6.8	1.30	5.40	3.29	–
30	seeds	5-04-610	92.0	20.0	3.30	16.70	8.66	–
31	seeds, meal solvent extracted	5-04-604	90.0	1.1	0.03	0.08	0.61	–
32	seeds without hulls, meal solvent extracted	5-04-612	90.0	0.9	0.30	0.60	0.39	–
	SWINE *Sus scrofa* fat (lard)–see FATS AND OILS							
	WHEAT *Triticum* spp							
33	bran	4-05-190	89.0	4.6	0.90	3.70	2.53	–
34	flour by-product, less than 9.5% fiber (wheat middlings)	4-05-205	89.0	5.2	1.00	4.10	2.79	–
35	grain	4-05-211	89.0	1.9	0.40	1.50	0.65	–
	WHEY *Bos taurus*							
36	dehydrated (cattle)	4-01-182	93.0	0.9	0.60	0.30	0.01	–
	YEAST, BREWERS *Saccharomyces cerevisiae*							
37	dehydrated	7-05-527	93.0	1.1	0.20	0.80	0.05	–

[a] Calculated by assuming that ether extract was all triglyceride (except for alfalfa products). Thus, values were calculated by multiplying percent ether extract by fraction that was saturated or unsaturated. Alfalfa ether extract was presumed to be 40 percent triglyceride equivalent, and the percentage of ether extract was multiplied by 0.04 and then by the fraction that was saturated or unsaturated.

TABLE 7 Composition of Some Common Feed Ingredients of Dog Food, Excluding Amino Acids; Data Expressed on a Dry Basis (100% Dry Matter)

Entry Number	Feed Name Description	International Feed Number[a]	Dry Matter (%)	Crude Protein (%)	Ether Extract (%)	Crude Fiber (%)	Nitrogen-free Extract (%)	Ash (%)	Calcium (%)	Copper (mg/kg)	Iodine (mg/kg)
	ALFALFA *Medicago sativa*										
001	meal dehydrated, 15% protein	1-00-022	90	17.3	2.5	29.4	40.9	10.0	1.37	10	0.13
002	meal dehydrated, 17% protein	1-00-023	92	18.9	3.0	26.2	41.3	10.6	1.52	11	0.16
003	meal dehydrated, 20% protein	1-00-024	92	22.0	3.7	22.5	40.6	11.3	1.74	12	0.15
	BARLEY *Hordeum vulgare*										
004	grain	4-00-549	88	13.5	2.1	5.7	76.0	2.6	0.05	9	0.65
005	grain, Pacific Coast	4-07-939	89	10.8	2.0	7.1	77.1	3.1	0.06	9	—
006	malt sprouts, dehydrated	5-00-545	94	28.1	1.4	16.0	47.5	7.0	0.23	—	—
	BEET MOLASSES—SEE MOLASSES										
	BEET, SUGAR *Beta vulgaris altissima*										
007	pulp, dehydrated	4-00-669	91	9.7	0.6	19.8	64.5	5.4	0.69	14	—
	BLOOD										
008	meal	5-00-380	92	87.2	1.4	1.1	4.5	5.8	0.32	11	—
009	spray dehydrated (blood flour)	5-00-381	93	93.0	1.4	1.1	1.8	7.1	0.52	9	—
	BONE										
010	meal steamed	6-00-400	97	8.4	3.4	2.1	10.7	72.6	31.53	14	30.77
011	phosphate	6-00-406	99	0.4	0.3	—	—	—	28.03	—	—
	BREAD—SEE WHEAT										
	BREWERS										
012	grains, dehydrated	5-02-141	92	29.4	7.2	14.4	45.1	3.9	0.33	33	0.07
	BUTTERMILK *Bos taurus*										
013	condensed (cattle)	5-01-159	29	36.9	8.3	0.3	42.3	12.3	1.44	1	—
	CALCIUM, CARBONATE										
014	$CaCO_3$	6-01-069	100	—	—	—	—	—	39.39	—	—
	CALCIUM, PHOSPHATE										
015	dibasic, from defluorinated phosphoric acid	6-01-080	97	—	—	—	—	—	22.00	10	—
	CASEIN										
016	dehydrated	5-01-162	91	92.7	0.7	0.2	3.9	2.4	0.67	4	—
	CATTLE *Bos taurus*										
017	kidneys, fresh	5-01-165	27	59.5	34.0	—	3.7	4.0	0.06	12	—
018	lips, fresh	5-07-940	30	60.0	33.3	—	—	—	—	—	—
019	livers, fresh	5-01-166	28	69.6	18.3	0.6	6.7	4.9	0.04	22	—
020	lungs, fresh	5-07-941	21	63.8	32.8	—	—	3.6	0.06	5	0.31
021	spleens, fresh	5-07-942	24	68.7	16.1	4.0	5.3	6.0	0.02	1	0.76
022	tripe, fresh	5-09-806	33	46.1	43.6	0.5	6.4	3.4	0.44	3	—
023	udders, fresh	5-07-943	20	58.6	30.0	1.2	2.7	7.4	2.62	3	—
	CEREALS										
024	distillers grains, dehydrated	5-02-144	93	29.5	8.0	13.8	47.0	1.7	0.15	52	—
	CHICKEN *Gallus domesticus*										
025	whole, fresh, day-old	5-07-946	13	57.9	26.3	3.6	1.6	6.1	—	—	—
026	broilers, flesh, fresh	5-28-310	24	76.5	20.2	—	—	3.3	0.04	—	—
027	hens, whole, fresh	5-07-950	46	46.2	42.2	—	—	7.1	—	—	—
028	eggs with shells, fresh	5-01-213	43	32.4	14.4	13.5	46.3	3.4	22.20	—	—
029	feet, fresh	5-07-947	33	54.5	23.1	—	—	16.6	6.45	2	0.37
030	gizzards, fresh	5-07-948	25	80.4	10.8	—	2.8	6.0	0.04	—	—
031	heads, fresh	5-07-949	33	57.6	18.2	—	—	—	—	—	—
032	by-product, fresh	5-07-951	44	48.3	36.1	—	7.7	12.4	2.64	—	—
033	viscera with heads, fresh	5-07-952	34	43.7	42.2	0.7	8.7	3.1	1.00	—	—
	CITRUS *Citrus* spp										
034	pomace without fines, dehydrated (pulp)	4-01-237	91	6.7	3.7	12.7	70.2	6.6	1.84	6	—
	COCONUT *Cocos nucifera*										
035	kernels with coats, meal mechanically extracted (copra meal)	5-01-572	92	22.4	6.9	12.8	50.6	7.3	0.22	15	—
036	kernels with coats, meal solvent extracted (copra meal)	5-01-573	91	23.4	3.9	15.4	50.7	6.6	0.19	10	—
	CORN, DENT YELLOW *Zea mays indentata*										
037	grain	4-02-935	89	10.9	4.3	2.9	80.4	1.5	0.03	4	—
038	grain, boiled dehydrated	4-02-853	88	10.5	5.2	1.8	80.4	2.1	—	—	—
039	grain, flaked	4-28-244	89	11.2	2.2	0.7	84.9	1.0	0.02	—	—
040	grits by-product (hominy feed)	4-03-011	90	11.5	7.7	6.7	72.8	3.1	0.05	15	—
041	distillers grains with solubles, dehydrated	5-28-236	92	25.0	10.3	9.9	45.5	4.8	0.15	58	—
042	distillers solubles, dehydrated	5-28-237	93	29.7	9.2	5.0	48.3	7.8	0.35	89	0.12
043	germs, meal wet milled solvent extracted	5-28-240	91	22.3	4.1	13.1	56.3	4.2	0.04	5	—
044	gluten meal, 41% protein	5-12-354	91	46.3	2.5	4.2	43.4	3.6	0.14	—	—

Entry Number	Iron (mg/kg)	Magnesium (%)	Manganese (mg/kg)	Phosphorus (%)	Potassium (%)	Sodium (%)	Zinc (mg/kg)	Biotin (mg/kg)	Choline (mg/kg)	Folic Acid (mg/kg)	Niacin (mg/kg)	Pantothenic Acid (mg/kg)	Carotene (Provitamin A) (mg/kg)	Vitamin B_6 (mg/kg)	Riboflavin (mg/kg)	Thiamin (mg/kg)	Vitamin B_{12} (µg/kg)	Vitamin E (mg/kg)	Vitamin A (IU/g)
001	309	0.31	31	0.24	2.48	0.08	21	0.88	1739	1.7	46	22.9	82	6.9	11.7	3.3	—	91	—
002	441	0.32	34	0.25	2.60	0.11	21	0.36	1494	4.8	40	32.4	131	7.7	14.1	3.7	—	121	—
003	415	0.36	39	0.30	2.73	0.14	22	0.39	1547	3.2	52	38.8	174	9.6	16.6	5.9	—	165	—
004	85	0.15	18	0.38	0.47	0.03	19	0.17	1177	0.6	94	9.1	2	7.3	1.8	5.0	—	25	—
005	97	0.14	18	0.39	0.58	0.02	17	0.17	1102	0.6	53	8.0	—	3.3	1.7	4.7	—	30	—
006	193	0.20	34	0.75	0.23	1.26	82	4.40	1713	0.2	54	9.5	—	10.2	3.2	8.9	—	16	—
007	329	0.27	38	0.10	0.20	0.21	1	—	902	—	18	1.5	0	—	0.8	0.4	—	—	—
008	4064	0.24	6	0.26	0.10	0.35	5	0.09	854	0.1	34	2.6	—	4.8	2.2	0.4	49	—	—
009	2993	0.24	7	0.26	0.10	0.42	—	0.30	645	0.4	24	3.5	—	4.8	3.1	0.3	13	—	—
010	780	0.64	34	14.22	0.19	0.40	342	—	—	—	4	2.5	—	—	0.9	0.4	—	—	—
011	—	—	—	11.31	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
012	266	0.16	40	0.55	0.09	0.23	30	0.68	1757	7.7	47	8.9	1	0.8	1.6	0.7	—	29	—
013	9	0.52	4	1.01	0.90	0.90	44	—	—	—	—	—	52	—	42.8	—	—	—	—
014	300	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
015	8667	0.55	242	19.02	0.06	1.96	69	—	—	—	—	—	—	—	—	—	—	—	—
016	15	0.01	5	0.90	0.01	0.01	30	0.05	229	0.5	1	2.9	—	0.5	1.7	0.5	—	—	—
017	229	0.04	3	0.82	0.66	0.73	58	3.02	1943	6.9	168	94.6	—	3.6	83.4	8.8	960	26	19.3
018	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
019	165	0.04	10	0.82	0.72	0.35	95	3.51	5093	8.4	269	164.9	—	18.0	92.2	6.3	1523	25	439.1
020	322	0.03	1	0.69	0.53	0.69	55	0.12	7933	0.9	49	2.6	—	1.8	8.4	2.8	423	13	3.3
021	1691	0.05	—	1.13	0.91	0.58	81	0.16	2036	4.8	25	8.2	—	1.3	15.3	3.1	247	56	3.0
022	318	0.02	15	0.40	0.11	0.15	34	0.09	510	0.3	33	3.9	—	0.6	4.2	0.7	229	1	1.6
023	102	0.08	3	1.37	0.79	0.58	104	0.30	4320	0.3	102	46.7	—	6.8	14.6	32.7	562	49	9.0
024	284	0.10	38	0.58	0.21	0.05	—	—	2584	0.2	49	12.2	8	6.0	7.1	2.6	—	31	—
025	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
026	81	—	—	0.62	—	—	—	—	—	—	230	—	—	—	15.6	2.9	—	—	—
027	—	0.22	—	0.59	0.31	0.83	—	0.46	6288	0.5	225	20.4	—	4.6	6.4	2.4	278	310	—
028	—	—	—	0.33	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
029	96	0.10	2	2.33	0.26	0.38	49	0.08	533	2.4	117	12.6	—	1.9	2.8	0.3	55	13	1.5
030	116	—	—	0.42	0.96	0.26	—	—	—	—	180	—	—	—	8.0	1.2	—	—	—
031	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
032	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
033	—	—	—	0.70	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
034	378	0.17	7	0.12	0.79	0.09	15	—	867	—	24	15.4	0	—	2.5	1.6	—	—	—
035	730	0.33	71	0.66	1.62	0.04	53	—	1036	1.5	26	6.8	—	—	3.4	0.8	—	—	—
036	750	0.36	72	0.66	1.63	0.04	—	—	1189	0.3	28	6.9	—	4.8	3.7	0.7	—	—	—
037	39	0.14	5	0.29	0.37	0.03	14	0.08	567	0.3	28	6.6	3	5.3	1.4	3.8	—	25	—
038	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
039	39	0.04	4	—	—	—	13	—	—	—	—	—	—	—	—	—	—	1	—
040	75	0.26	16	0.57	0.65	0.09	3	0.15	1280	0.3	52	9.1	10	12.1	2.3	8.9	—	—	—
041	250	0.18	25	0.71	0.44	0.57	—	0.85	2803	1.0	79	15.3	3	5.4	10.0	3.1	—	43	—
042	610	0.65	80	1.37	1.80	0.25	92	1.79	5151	1.4	134	25.2	1	9.5	22.7	7.3	3	49	—
043	370	0.34	4	0.47	0.31	0.08	114	0.24	1785	0.2	33	4.6	2	6.8	4.2	4.9	—	94	—
044	—	0.05	8	0.44	—	0.11	—	0.16	363	0.2	58	12.5	18	8.8	2.2	0.2	—	46	—

Entry Number	Feed Name Description	International Feed Number[a]	Dry Matter (%)	Crude Protein (%)	Ether Extract (%)	Crude Fiber (%)	Nitrogen-free Extract (%)	Ash (%)	Calcium (%)	Copper (mg/kg)	Iodine (mg/kg)	
045	gluten meal, 60% protein	5-28-242	90	67.5	2.3	2.0	26.3	1.9	0.08	—	—	
	CORN, DENT WHITE *Zea mays indentata*											
046	grits by-product (hominy feed)	4-02-990	90	11.8	8.8	5.7	70.5	3.3	0.04	15	—	
	CORN, FLINT *Zea mays indurata*											
047	grain	4-02-948	89	11.1	4.9	2.1	80.2	1.7	—	13	—	
	COTTON *Gossypium* spp											
048	seeds, meal mechanically extracted, 41% protein	5-01-617	93	44.3	5.0	12.8	31.3	6.6	0.21	20	—	
049	seeds, meal prepressed solvent extracted, 41% protein	5-07-872	91	45.6	1.3	14.1	32.0	7.0	0.22	20	—	
050	seeds, meal solvent extracted, 41% protein	5-01-621	91	45.2	1.6	13.3	32.8	7.1	0.18	22	—	
051	seeds without hulls, meal prepressed solvent extracted, 50% protein	5-07-874	93	54.0	1.4	8.8	28.8	7.1	0.19	16	—	
	FATS AND OILS											
052	fat, animal-poultry	4-00-409	99	—	99.0	—	—	—	—	—	—	
053	fat (lard), swine	4-04-790	99	—	99.0	—	—	—	—	—	—	
054	oil, soybean	4-07-983	99	1.4	96.0	0.0	2.3	0.3	—	—	—	
	FISH											
055	livers, meal mechanically extracted	5-01-968	93	67.7	18.6	1.3	5.8	6.6	—	—	—	
056	solubles, condensed	5-01-969	50	65.3	11.2	0.9	3.5	19.2	0.43	92		2.21
057	solubles, dehydrated	5-01-971	93	69.2	8.9	1.5	7.0	13.5	1.39	—	—	
	FISH, ALEWIFE *Pomolobus pseudoharengus*											
058	meal mechanically extracted	5-09-830	90	65.7	12.9	—	—	14.6	6.63	23	—	
059	whole, fresh	5-07-964	26	59.3	28.9	—	—	9.5	—	—	—	
	FISH, ANCHOVY *Engraulis ringens*											
060	meal mechanically extracted	5-01-985	92	71.2	4.5	1.1	7.1	16.1	4.08	10		3.41
	FISH, CARP *Cyprinus carpio*											
061	meal boiled	5-09-831	90	58.6	—	0.8	—	—	—	—	—	
062	whole, fresh	5-01-966	31	61.9	29.4	—	—	9.4	0.23	—	—	
	FISH, CATFISH *Ictalurus* spp											
063	boiled	5-09-833	40	69.7	—	—	—	—	—	8	—	
064	cuttings, fresh	5-09-832	34	64.5	—	—	—	—	5.57	7	—	
065	meal mechanically extracted	5-09-835	92	55.3	—	—	—	—	7.77	28	—	
066	whole, fresh	5-07-965	22	55.1	25.3	—	—	14.1	—	—	—	
	FISH, FLOUNDER Bothidae (family)-Pleuronectidae (family)											
067	whole, fresh	5-01-996	17	68.2	2.9	—	—	—	—	—	—	
	FISH, HADDOCK *Melanogrammus aeglefinus*											
068	flesh, fresh	5-09-281	20	93.8	0.5	—	—	7.2	0.12	—	—	
	FISH, HAKE *Merluccius* spp-*Urophycis* spp											
069	whole, boiled	5-07-967	26	57.9	20.4	0.2	10.4	11.2	—	—	—	
070	whole, boiled acidified	5-07-968	25	—	21.2	1.1	—	—	—	—	—	
071	whole, fresh	5-07-969	20	67.1	14.6	0.2	6.1	12.0	3.06	—	—	
	FISH, HERRING *Clupea harengus*											
072	meal mechanically extracted	5-02-000	92	78.3	9.2	0.7	0.4	11.4	2.40	6		0.57
073	flesh, fresh	5-19-693	26	70.4	24.5	—	—	6.3	—	—	—	
	FISH, MACKEREL, ATLANTIC *Scomber scombrus*											
074	whole, fresh	5-07-971	30	53.3	35.0	—	—	11.3	3.64	3		0.76
	FISH, MACKEREL, PACIFIC *Scomber japonicus*											
075	flesh, fresh	5-07-309	30	72.5	24.2	—	—	4.6	0.03	—	—	
	FISH, MENHADEN *Brevoortia tyrannus*											
076	meal mechanically extracted	5-02-009	92	66.7	10.5	1.0	1.1	20.8	5.65	12		1.19
	FISH, REDFISH *Sciaenops ocellata*											
077	meal mechanically extracted	5-07-973	93	61.0	9.8	1.0	1.1	27.1	6.96	—	—	
078	whole, fresh	5-08-191	24	68.1	22.5	—	—	8.3	—	—	—	
	FISH, ROCKFISH *Sebastodes* spp											
079	whole, fresh	5-07-974	21	89.6	8.5	—	—	—	—	—	—	
	FISH, SALMON *Oncorhynchus* spp											
080	flesh, fresh	5-07-311	36	70.6	27.0	—	—	4.1	0.22	—	—	

Entry Number	Iron (mg/kg)	Magnesium (%)	Manganese (mg/kg)	Phosphorus (%)	Potassium (%)	Sodium (%)	Zinc (mg/kg)	Biotin (mg/kg)	Choline (mg/kg)	Folic Acid (mg/kg)	Niacin (mg/kg)	Pantothenic Acid (mg/kg)	Carotene (Provitamin A) (mg/kg)	Vitamin B_6 (mg/kg)	Riboflavin (mg/kg)	Thiamin (mg/kg)	Vitamin B_{12} (µg/kg)	Vitamin E (mg/kg)	Vitamin A (IU/g)
045	—	0.09	7	0.50	0.20	0.05	34	0.21	390	0.28	66	3.9	34	7.6	2.2	0.3	—	26	—
046	79	0.26	15	0.77	0.71	0.08	—	0.15	1066	—	52	8.1	—	14.7	2.5	10.8	—	—	—
047	30	—	8	0.31	0.36	—	—	—	—	—	18	—	—	—	—	—	—	—	—
048	197	0.58	24	1.16	1.45	0.05	69	1.19	2965	2.3	38	11.2	0	5.4	5.7	7.0	—	35	—
049	223	0.55	23	1.21	1.39	0.04	69	0.61	3141	2.8	44	8.2	—	4.6	4.9	3.7	—	—	—
050	228	0.59	23	1.21	1.52	0.05	68	1.06	3056	1.5	45	15.0	—	6.2	5.2	7.3	—	17	—
051	120	0.50	25	1.24	1.56	0.06	79	0.48	3154	1.0	48	15.4	—	8.8	5.3	8.8	—	12	—
052	—	—	—	—	0.23	—	—	—	—	—	—	—	—	—	—	—	—	8	—
053	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	23	—
054	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
055	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
056	445	0.06	27	1.18	3.22	4.67	87	0.58	6759	0.4	350	70.8	3	24.2	25.2	10.0	1007	—	—
057	326	—	54	1.60	—	—	83	0.43	5954	0.6	276	54.3	—	25.9	14.6	8.0	524	7	—
058	756	0.18	24	3.54	0.73	0.29	122	—	5160	—	33	9.1	—	—	3.2	0.1	346	—	4.3
059	—	—	—	0.85	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
060	237	0.27	12	2.70	0.78	0.95	114	0.21	4036	0.2	89	10.9	—	5.0	8.2	0.5	233	5	—
061	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
062	40	—	—	1.14	1.29	0.23	—	—	—	—	68	—	—	—	1.8	0.5	—	—	—
063	500	—	15	2.43	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
064	90	—	11	2.55	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
065	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
066	20	—	—	—	1.50	0.27	—	—	—	—	77	—	—	—	1.4	1.8	—	—	—
067	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
068	36	—	—	1.01	1.56	0.31	—	—	—	—	154	—	—	—	3.6	2.1	—	—	—
069	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
070	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
071	—	—	—	1.93	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
072	136	0.16	6	1.82	1.17	0.66	143	0.52	5752	0.4	93	18.2	—	5.2	11.0	0.4	467	24	—
073	50	—	—	0.96	2.04	0.36	—	—	—	—	145	—	—	—	6.1	0.8	—	—	—
074	90	0.10	—	1.28	0.55	0.56	78	0.12	3422	8.5	24	17.9	—	1.2	9.6	2.9	753	34	85.5
075	70	—	—	0.91	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
076	524	0.16	37	3.16	0.76	0.43	162	0.20	3398	0.2	60	9.4	—	5.1	5.2	0.8	133	13	—
077	—	—	8	3.64	—	—	—	0.18	3681	—	44	9.0	—	—	7.5	0.2	152	6	—
078	—	—	—	—	1.26	0.30	—	—	—	—	—	—	—	—	—	—	—	—	—
079	—	—	—	—	1.85	0.28	—	—	—	—	—	—	—	—	5.7	2.8	—	—	—
080	27	—	—	0.68	1.12	0.11	—	—	—	—	192	—	—	—	4.3	2.8	—	—	—

Entry Number	Feed Name Description	International Feed Number[a]	Dry Matter (%)	Crude Protein (%)	Ether Extract (%)	Crude Fiber (%)	Nitrogen-free Extract (%)	Ash (%)	Calcium (%)	Copper (mg/kg)	Iodine (mg/kg)	
	FISH, SALMON *Oncorhynchus* spp-*Salmo* spp											
081	meal mechanically extracted	5-02-012	93	65.6	12.2	0.3	2.7	19.1	5.88	13	—	
	FISH, SARDINE *Clupea* spp-*Sardinops* spp											
082	meal mechanically extracted	5-02-015	93	70.0	5.4	1.1	6.5	17.0	4.95	22	—	
	FISH, SMELT *Osmerus* spp											
083	whole, fresh	5-07-975	21	88.6	10.0	—	—	—	—	—	—	
	FISH, SOLE Soleidae (family)											
084	whole, fresh	5-07-976	20	65.6	21.0	0.2	1.0	12.3	3.19	—	—	
	FISH, TUNA *Thunnus thynnus-Thunnus albacares*											
085	meal mechanically extracted	5-02-023	93	63.6	7.4	0.9	4.5	23.6	8.48	11	—	
086	process residue, ground	5-07-977	94	56.8	21.8	—	—	—	—	—	—	
	FISH, TURBOT *Psetta maxima*											
087	whole, fresh	5-07-978	25	57.3	24.3	0.2	8.1	10.3	1.25	—	—	
	FISH, WHITE Gadidae (family)-Lophiidae (family)-Rajidae (family)											
088	meal mechanically extracted	5-02-025	91	68.2	5.1	0.8	0.5	25.4	8.02	6	—	
	FISH, WHITING *Gadus merlangus*											
089	whole, fresh	5-07-979	23	69.9	8.7	—	—	—	—	—	—	
	GROUNDNUT—SEE PEANUT											
	HORSE *Equus caballus*											
090	meat, fresh	5-07-980	29	63.6	32.5	0.9	—	—	0.07	—		0.29
091	meat with bone, fresh	5-07-981	34	51.4	19.4	—	—	—	—	—	—	
	LIMESTONE											
092	ground	6-02-632	100	—	—	—	—	—	37.30	—	—	
	LIVERS											
093	meal	5-00-389	92	71.4	17.0	1.5	3.4	6.8	0.61	97	—	
	MEAT											
094	meal rendered	5-00-385	94	54.8	9.7	2.8	3.9	28.8	9.44	10	—	
095	with blood, meal rendered (tankage)	5-00-386	92	64.5	9.7	2.2	0.2	23.4	6.37	42	—	
096	with bone, meal rendered	5-00-388	93	54.1	10.4	2.4	1.7	31.5	11.06	2		1.41
	MILK *Bos taurus*											
097	dehydrated (cattle)	5-01-167	96	26.5	27.8	0.2	39.8	5.7	0.95	1	—	
098	skimmed dehydrated (cattle)	5-01-175	94	35.8	0.9	0.2	54.6	8.4	1.36	1	—	
	MOLASSES *Beta vulgaris altissima*											
099	beet, sugar, molasses, more than 48% invert sugar more than 79.5 degrees brix	4-00-668	78	8.5	0.2	—	79.9	11.3	0.17	22	—	
	MOLASSES *Saccharum officinarum*											
100	sugarcane, molasses, dehydrated	4-04-695	94	10.3	0.9	6.7	68.8	13.3	1.10	79		2.10
101	sugarcane, molasses, more than 48% invert sugars more than 79.5 degrees brix (black strap)	4-04-696	75	5.8	0.2	0.5	80.2	13.3	1.00	79		2.10
	MILLET *Setaria* spp											
102	grain	4-03-098	90	13.5	4.5	6.4	72.6	3.1	0.05	24	—	
	OATS *Avena sativa*											
103	cereal by-product, less than 4% fiber (feeding oat meal) (oat middlings)	4-03-300	91	16.4	7.0	3.9	70.2	2.5	0.08	5	—	
104	grain	4-03-309	89	13.3	5.4	12.1	65.8	3.4	0.07	7		0.11
105	grain, grade 1 heavy 46.3 kg/hl (36 lb/bu)	4-03-312	89	14.2	5.8	10.0	67.0	3.0	0.11	7	—	
106	grain, grade 1 43.8 kg/hl (34 lb/bu)	4-03-313	90	13.3	5.0	12.2	—	3.7	0.09	—	—	
107	grain, grade 2 41.2 kg/hl (32 lb/bu)	4-03-316	89	12.8	4.7	12.2	66.6	3.7	0.07	—	—	
108	groats	4-03-331	90	17.7	6.9	2.8	70.3	2.4	0.08	7		0.12
109	groats, boiled ground	4-07-982	91	18.4	6.4	3.3	—	—	0.08	—	—	
110	hulls	1-03-281	92	3.9	1.8	33.4	54.4	6.6	0.15	4	—	
	OYSTER *Crassostrea* spp-*Ostrea* spp											
111	shells, fine ground (oyster shell flour)	6-03-481	99	—	—	—	—	—	38.00	—	—	
	PEA *Pisum* spp											
112	seeds, ground	5-06-623	89	24.9	1.5	7.5	62.7	3.4	0.17	—	—	
	PEANUT *Arachis hypogaea*											
113	kernels, meal mechanically extracted (peanut meal)	5-03-649	93	52.0	6.3	7.5	28.8	5.5	0.20	16		0.07

Entry Number	Iron (mg/kg)	Magnesium (%)	Manganese (mg/kg)	Phosphorus (%)	Potassium (%)	Sodium (%)	Zinc (mg/kg)	Biotin (mg/kg)	Choline (mg/kg)	Folic Acid (mg/kg)	Niacin (mg/kg)	Pantothenic Acid (mg/kg)	Carotene (Provitamin A) (mg/kg)	Vitamin B_6 (mg/kg)	Riboflavin (mg/kg)	Thiamin (mg/kg)	Vitamin B_{12} (μg/kg)	Vitamin E (mg/kg)	Vitamin A (IU/g)
081	193	—	9	3.72	—	—	—	—	2990	—	27	7.4	—	—	6.2	0.9	—	—	—
082	321	0.11	25	2.88	0.35	0.19	—	0.11	3518	—	81	11.8	—	—	5.8	0.3	256	—	—
083	—	—	—	1.20	—	—	—	—	—	—	67	—	—	—	5.7	0.5	—	—	—
084	—	—	—	2.00	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
085	383	0.25	9	4.54	0.77	0.80	227	0.22	3227	—	155	8.4	—	—	7.3	1.6	324	6	—
086	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
087	—	—	—	0.88	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
088	199	0.20	14	4.17	0.91	0.85	98	0.09	3397	0.4	65	10.9	—	6.5	10.0	1.8	98	10	—
089	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
090	167	0.04	1	1.06	0.38	0.18	60	0.08	1043	0.8	16	4.8	—	0.7	—	1.4	142	25	—
091	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
092	770	—	276	0.02	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
093	681	0.11	10	1.36	—	—	67	0.02	2281	6.0	221	31.5	—	—	39.1	0.2	542	—	—
094	470	0.29	10	4.74	0.61	1.37	85	0.13	2177	0.4	60	6.5	—	2.9	5.6	0.2	72	1	—
095	2283	0.39	21	3.33	0.60	1.81	—	—	2391	1.7	40	2.8	—	—	2.4	0.4	147	—	—
096	735	1.09	14	5.48	1.43	0.77	96	0.11	2196	0.4	53	4.4	—	9.4	4.9	0.2	116	1	—
097	10	0.10	0	0.74	1.08	0.38	23	0.40	—	—	9	23.8	—	4.9	20.6	3.9	—	—	11.8
098	10	0.13	2	1.00	1.70	0.49	41	0.35	1450	0.7	12	38.6	—	4.5	20.5	3.9	54	10	—
099	87	0.29	6	0.03	6.07	1.48	18	—	1063	—	53	5.8	—	—	2.9	—	—	5	—
100	250	0.47	57	0.15	3.60	0.20	33	—	—	—	—	—	—	—	—	—	—	6	—
101	250	0.43	56	0.11	3.84	0.22	30	0.92	1012	0.1	49	50.3	—	5.7	3.8	1.2	—	7	—
102	70	0.18	33	0.32	0.48	0.04	15	—	852	0.2	54	10.1	—	—	1.6	7.3	—	—	—
103	421	0.16	48	0.49	0.55	0.10	154	0.24	1267	0.5	25	18.6	—	—	1.9	7.7	—	26	—
104	85	0.14	42	0.38	0.44	0.08	41	0.31	1116	0.4	16	8.8	0	2.8	1.7	7.1	—	15	—
105	80	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
106	—	—	42	0.33	0.41	0.07	—	0.12	1222	0.3	20	14.4	—	—	1.2	—	—	22	—
107	—	—	—	0.30	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
108	82	0.13	31	0.48	0.39	0.06	0	—	1264	0.6	11	15.4	—	1.2	1.3	7.2	—	16	—
109	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
110	111	0.09	20	0.15	0.62	0.04	—	—	281	1.0	10	3.4	—	2.4	1.9	0.7	—	—	—
111	2870	0.30	100	0.07	0.10	0.21	—	—	—	—	—	—	—	—	—	—	—	—	—
112	—	—	—	0.33	0.44	0.04	33	—	712	0.4	41	11.0	—	1.1	2.0	—	—	—	—
113	169	0.31	28	0.61	1.35	0.23	22	0.35	2052	0.7	186	49.7	0	8.0	8.8	6.6	—	3	—

Entry Number	Feed Name Description	International Feed Number[a]	Dry Matter (%)	Crude Protein (%)	Ether Extract (%)	Crude Fiber (%)	Nitrogen-free Extract (%)	Ash (%)	Calcium (%)	Copper (mg/kg)	Iodine (mg/kg)
114	kernels, meal solvent extracted (peanut meal)	5-03-650	92	52.3	1.4	10.8	29.2	6.3	0.29	17	0.07
	PHOSPHATE										
115	defluorinated	6-01-780	100	—	—	—	—	—	32.00	20	—
	POTATO *Solanum tuberosum*										
116	tubers, dehydrated	4-07-850	91	8.9	0.5	2.3	79.6	8.7	0.08	—	—
	POULTRY										
117	feathers, hydrolyzed	5-03-795	93	91.3	3.2	1.5	0.3	3.8	0.28	7	0.05
	RICE *Oryza sativa*										
118	bran with germs (rice, bran)	4-03-928	91	14.1	15.1	12.8	45.2	12.8	0.08	15	—
119	grain, ground (ground rough rice) (ground paddy rice)	4-03-938	89	8.9	1.9	10.0	73.8	5.3	0.07	3	0.05
120	groats, ground	4-03-935	88	9.6	1.3	1.0	86.9	1.4	0.04	5	—
121	groats, polished (rice, polished)	4-03-942	89	8.2	0.5	0.4	90.4	0.6	0.03	3	—
122	polishings	4-03-943	90	13.4	13.9	3.6	60.9	8.3	0.05	4	—
	RYE *Secale cereale*										
123	grain	4-04-047	88	13.8	1.7	2.5	80.0	1.9	0.07	8	—
	SEAWEED, KELP Laminariales (order)-Fucales (order)										
124	whole, sun-cured	1-04-190	89	9.6	2.5	7.7	57.8	22.4	1.83	—	1,500.00
	SHRIMP *Pandalus* spp-*Penaeus* spp										
125	process residue, meal (shrimp meal)	5-04-226	90	44.2	4.3	15.6	6.1	29.7	10.80	—	—
	SODIUM, PHOSPHATE										
126	monobasic, $NaH_2PO_4 \cdot H_2O$	6-04-288	97	—	—	—	—	—	—	—	—
	SODIUM, TRIPOLYPHOSPHATE										
127	$Na_5P_3O_{10}$	6-08-076	96	—	—	—	—	—	—	—	—
	SORGHUM *Sorghum bicolor*										
128	grain	4-04-383	90	12.4	3.1	2.6	79.9	2.0	0.04	11	0.04
	SORGHUM, MILO *Sorghum bicolor subglabrescens*										
129	grain	4-04-444	89	11.3	3.1	2.5	81.3	1.8	0.05	5	0.07
	SOYBEAN *Glycine max*										
130	seeds, meal mechanically extracted	5-04-600	90	47.7	5.3	6.6	33.7	6.7	0.29	24	—
131	seeds, meal solvent extracted	5-04-604	90	49.9	1.4	6.5	35.2	7.0	0.34	25	0.15
132	seeds without hulls, meal solvent extracted	5-04-612	90	55.1	1.0	3.7	33.7	6.5	0.29	22	0.12
	SUGARCANE, MOLASSES—SEE MOLASSES										
	SUNFLOWER, COMMON *Helianthus annuus*										
133	seeds without hulls, meal mechanically extracted	5-04-738	93	44.6	8.7	13.1	26.4	7.1	0.42	4	—
134	seeds without hulls, meal solvent extracted	5-04-739	93	49.8	3.1	12.2	26.7	8.1	0.44	4	—
	SWINE *Sus scrofa*										
135	kidneys, fresh	5-09-813	22	73.8	16.3	0.2	—	5.4	0.05	—	—
136	livers, fresh	5-04-792	30	68.8	16.5	0.3	9.1	5.3	0.04	187	1.12
137	lungs, fresh	5-26-140	20	70.2	12.3	0.3	13.4	3.8	0.05	1	0.82
	TOMATO *Lycopersicon esculentum*										
138	pomace, dehydrated	5-05-041	92	23.5	10.3	26.4	32.2	7.5	0.43	33	—
	TORULA DRIED YEAST—SEE YEAST, TORULA										
	TURKEY *Meleagris gallopavo*										
139	flesh, fresh	5-24-927	32	75.7	20.8	0.0	0.0	3.5	0.02	—	—
140	viscera, fresh	5-08-616	28	44.0	40.5	—	—	6.0	—	—	—
141	viscera, fresh, chick	5-07-985	28	54.7	37.2	0.9	0.3	6.9	—	—	—
	WHALE *Balaena glacialis-Balaenoptera spp-Physeter catodon*										
142	meat, fresh	5-07-986	29	70.8	25.8	—	—	3.4	0.03	—	—
	WHEAT *Triticum aestivum*										
143	bran	4-05-190	89	17.1	4.4	11.3	60.3	6.9	0.13	14	0.07
144	bread, dehydrated	4-07-944	95	13.0	2.4	0.3	81.9	2.4	0.07	—	—
145	flour, less than 1.5% fiber (wheat feed flour)	4-05-190	88	13.4	1.4	1.5	83.2	0.5	0.03	1	0.10
146	flour by-product, less than 4% fiber (wheat red dog)	4-05-203	88	17.4	3.8	2.9	73.4	2.5	0.05	7	—
147	flour by-product, less than 7% fiber (wheat shorts)	4-05-201	88	18.6	5.2	7.7	63.6	4.9	0.10	13	—
148	germs, ground	5-05-218	88	28.1	9.5	3.5	54.2	4.7	0.06	11	—

Entry Number	Iron (mg/kg)	Magnesium (%)	Manganese (mg/kg)	Phosphorus (%)	Potassium (%)	Sodium (%)	Zinc (mg/kg)	Biotin (mg/kg)	Choline (mg/kg)	Folic Acid (mg/kg)	Niacin (mg/kg)	Pantothenic Acid (mg/kg)	Carotene (Provitamin A) (mg/kg)	Vitamin B_6 (mg/kg)	Riboflavin (mg/kg)	Thiamin (mg/kg)	Vitamin B_{12} (µg/kg)	Vitamin E (mg/kg)	Vitamin A (IU/g)
114	154	0.17	29	0.68	1.23	0.08	22	0.36	2120	0.7	188	50.7	—	6.9	9.6	6.2	—	3	—
115	6700	0.42	200	18.00	0.08	4.90	69	—	—	—	—	—	—	—	—	—	—	—	—
116	—	0.12	2	0.22	2.15	0.01	2	0.11	2879	0.7	37	22.0	—	15.5	1.1	—	—	—	—
117	81	0.22	14	0.72	0.31	0.76	74	0.05	962	0.2	23	9.7	—	3.2	2.1	0.1	90	—	—
118	210	1.04	415	1.70	1.92	0.04	32	0.47	1357	2.4	330	25.2	—	—	2.8	24.7	—	66	—
119	57	0.15	20	0.32	0.36	0.06	17	0.09	1076	0.4	39	9.1	—	5.0	1.2	3.2	—	11	—
120	18	0.06	5	0.25	0.24	0.01	—	—	—	—	53	—	—	—	0.6	3.9	—	—	—
121	16	0.02	12	0.13	0.12	0.02	2	—	1018	0.2	17	3.9	—	0.4	0.6	0.7	—	4	—
122	178	0.87	14	1.48	1.27	0.12	29	0.68	1383	—	560	51.4	—	—	2.0	22.1	—	100	—
123	69	0.14	66	0.37	0.52	0.03	36	0.06	479	0.7	21	9.1	0	2.9	1.9	4.2	—	17	—
124	—	6.37	—	0.18	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
125	116	0.60	33	2.65	0.92	1.74	32	—	6102	—	—	—	—	—	4.4	—	—	—	—
126	—	—	—	22.50	—	16.68	—	—	—	—	—	—	—	—	—	—	—	—	—
127	40	—	—	25.00	—	31.00	—	—	—	—	—	—	—	—	—	—	—	—	—
128	51	0.18	18	0.33	0.39	0.03	19	0.42	737	0.2	43	12.5	1	5.0	1.4	4.7	—	12	—
129	54	0.14	18	0.34	0.35	0.04	19	0.81	720	0.2	42	12.4	0	4.0	1.3	4.7	—	13	—
130	175	0.28	35	0.68	1.08	0.03	66	0.36	2916	7.1	34	15.8	0	7.2	3.8	4.3	—	7	—
131	133	0.30	32	0.70	2.20	0.04	48	0.38	2915	0.7	31	18.2	0	6.7	3.2	6.2	—	3	—
132	148	0.32	41	0.70	2.30	0.03	61	0.36	3054	0.8	24	16.4	—	5.5	3.2	3.4	—	3	—
133	33	0.78	22	1.14	1.14	0.24	—	—	—	—	—	—	—	—	—	—	—	—	—
134	33	0.77	20	0.98	1.14	0.24	—	—	4430	—	288	43.9	—	14.8	4.2	3.4	—	12	—
135	303	—	—	0.99	0.81	0.52	—	6.79	—	—	443	138.6	—	22.6	78.3	26.2	317	—	—
136	480	0.04	6	1.22	0.85	0.24	146	2.49	—	6.9	544	77.9	—	10.0	90.3	7.7	935	—	—
137	475	0.04	—	1.05	0.30	0.96	68	0.32	4373	0.9	80	4.1	—	2.2	13.4	2.2	152	27	—
138	4600	0.20	51	0.60	3.63	—	—	—	—	—	—	—	—	—	6.7	12.3	—	—	—
139	47	—	—	0.69	0.90	0.21	—	—	—	—	252	—	—	—	4.4	2.5	—	—	—
140	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
141	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
142	—	—	—	0.48	0.07	0.28	—	—	—	—	—	—	—	—	2.7	3.1	—	—	63.9
143	128	0.60	125	1.38	1.56	0.04	128	0.32	1797	1.6	268	33.5	3	9.6	4.6	7.9	—	21	—
144	—	—	—	0.11	—	—	—	—	—	—	30	—	—	—	2.1	2.9	—	—	—
145	6	0.06	4	0.20	0.16	0.01	7	—	947	0.1	14	7.0	—	1.0	0.6	2.1	—	3	—
146	52	0.18	62	0.56	0.58	0.05	74	0.12	1742	0.9	48	15.1	—	5.2	2.5	25.9	—	37	—
147	82	0.28	132	0.91	1.06	0.03	124	—	2090	1.9	121	25.3	—	8.2	4.7	21.7	—	61	—
148	58	0.28	151	1.05	1.09	0.03	135	0.24	3468	2.4	81	22.8	—	12.9	6.8	25.8	—	160	—

Entry Number	Feed Name Description	International Feed Number[a]	Dry Matter (%)	Crude Protein (%)	Ether Extract (%)	Crude Fiber (%)	Nitrogen-free Extract (%)	Ash (%)	Calcium (%)	Copper (mg/kg)	Iodine (mg/kg)
149	grain	4-05-211	89	16.0	2.0	2.9	77.2	1.9	0.04	7	0.10
150	grain, hard red spring	4-05-258	88	17.2	2.0	2.9	76.1	1.8	0.04	7	—
151	grain, hard red winter	4-05-268	88	14.4	1.8	2.8	79.1	1.9	0.05	5	—
152	grain, soft red winter	4-05-294	86	13.0	1.8	2.4	80.6	2.1	0.05	7	—
153	grain, soft white winter	4-05-337	89	11.3	1.9	2.6	82.4	1.8	0.07	8	—
154	grain, soft white winter, Pacific Coast	4-08-555	89	11.2	2.2	2.8	81.7	2.1	0.10	—	—
155	grain screenings	4-05-216	89	15.8	3.9	7.7	66.5	6.1	0.15	3	—
156	grits, cracked screened	4-07-852	90	12.7	1.0	0.4	85.4	0.4	0.03	—	—
157	middlings, less than 9.5% fiber	4-05-205	89	18.2	4.8	8.3	63.7	5.2	0.14	18	—
158	mill run, less than 9.5% fiber	4-05-206	90	17.2	4.6	9.2	63.2	5.9	0.11	21	—
	WHEAT, DURUM *Triticum durum*										
159	grain	4-05-224	88	15.9	2.0	2.5	77.7	1.8	0.10	8	—
	WHEY *Bos taurus*										
160	dehydrated (cattle)	4-01-182	93	14.2	0.7	0.2	75.0	9.8	0.92	50	—
161	low lactose, dehydrated (dried whey product) (cattle)	4-01-188	93	17.9	1.1	0.2	64.3	16.5	1.71	8	10.55
	YEAST, BREWERS *Saccharomyces cerevisiae*										
162	dehydrated	7-05-527	93	46.9	0.9	3.1	42.1	7.1	0.13	35	0.38
	YEAST, PETROLEUM *Candida utilis*										
163	oil residue, solvent extracted dehydrated	7-09-836	92	51.1	—	—	—	—	0.02	—	—
	YEAST, PRIMARY *Saccharomyces cerevisiae*										
164	dehydrated	7-05-533	93	51.8	1.1	3.3	35.1	8.6	0.39	—	—
	YEAST, TORULA *Torulopsis utilis*										
165	dehydrated	7-05-534	93	52.7	1.7	2.4	35.4	8.3	0.54	14	2.69

[a] First digit is class of feed: 1, dry forages and roughages; 2, pasture, range plants, and forages fed green; 3, silages; 4, energy feeds; 5, protein supplements; 6, minerals; 7, vitamins; 8, additives.

Entry Number	Iron (mg/kg)	Magnesium (%)	Manganese (mg/kg)	Phosphorus (%)	Potassium (%)	Sodium (%)	Zinc (mg/kg)	Biotin (mg/kg)	Choline (mg/kg)	Folic Acid (mg/kg)	Niacin (mg/kg)	Pantothenic Acid (mg/kg)	Carotene (Provitamin A) (mg/kg)	Vitamin B_6 (mg/kg)	Riboflavin (mg/kg)	Thiamin (mg/kg)	Vitamin B_{12} (μg/kg)	Vitamin E (mg/kg)	Vitamin A (IU/g)
149	61	0.16	42	0.42	0.42	0.05	50	0.11	1085	0.5	64	11.4	0	5.6	1.6	4.8	1	17	—
150	64	0.17	42	0.43	0.41	0.03	52	0.13	1200	0.5	65	11.2	0	5.8	1.6	4.8	—	14	—
151	35	0.13	33	0.43	0.49	0.02	43	0.12	1179	0.4	61	11.1	—	3.4	1.6	4.8	—	12	—
152	30	0.11	36	0.43	0.46	0.01	48	—	1053	0.5	59	10.9	—	3.6	1.7	5.1	—	18	—
153	41	0.13	43	0.36	0.46	0.04	29	0.12	1097	0.4	59	12.6	—	4.6	1.3	5.3	—	20	—
154	60	0.15	—	0.34	0.51	0.10	—	—	1090	—	52	12.4	—	—	1.2	—	—	—	—
155	60	0.18	33	0.39	0.58	0.10	44	—	980	0.5	65	12.7	—	—	1.0	7.2	—	—	—
156	17	—	—	0.12	0.09	0.00	—	—	—	—	8	—	—	—	1.1	0.7	—	—	—
157	104	0.40	127	0.99	1.12	0.02	95	0.27	1387	1.1	103	19.4	—	10.3	2.3	17.1	—	26	—
158	105	0.52	116	1.13	1.33	0.02	—	0.34	1118	1.2	129	15.2	—	12.2	2.4	17.0	—	32	—
159	48	0.17	32	0.41	0.51	—	37	—	—	0.4	60	10.1	—	3.4	1.2	7.3	—	—	—
160	181	0.14	6	0.82	1.23	0.70	3	0.38	1921	0.9	11	49.6	—	3.6	29.4	4.3	20	0	0.5
161	202	0.23	9	1.12	3.16	1.54	5	0.54	4133	0.8	19	80.3	—	5.3	52.1	5.4	38	—	—
162	117	0.27	6	1.49	1.79	0.05	41	1.08	4227	10.3	482	118.4	—	39.8	38.1	99.2	1	2	—
163	—	—	—	5.87	4.02	—	—	—	—	—	—	—	—	—	—	—	—	—	—
164	324	0.39	4	1.86	—	—	—	1.74	—	33.6	325	336.9	—	—	41.9	6.9	7	—	—
165	126	0.18	9	1.71	2.04	0.04	100	1.47	3823	26.0	525	100.6	—	38.9	47.6	6.6	4	—	—

TABLE 8 Amino Acid Composition of Some Common Feed Ingredients of Dog Food; Data Expressed on a Dry Basis (100% Dry Matter)

Entry Number	Feed Name Description	International Feed Number	Dry Matter (%)	Crude Protein (%)	Arginine (%)	Cystine (%)	Histidine (%)	Isoleucine (%)	Leucine (%)	Lysine (%)	Methionine (%)	Phenylalanine (%)	Threonine (%)	Tryptophan (%)	Tyrosine (%)	Valine (%)
	ALFALFA *Medicago sativa*															
001	meal dehydrated, 15% protein	1-00-022	90	17.3	0.65	0.23	0.30	0.71	1.13	0.66	0.24	0.69	0.62	0.42	0.45	0.83
002	meal dehydrated, 17% protein	1-00-023	92	18.9	0.84	0.31	0.36	0.88	1.39	0.93	0.29	0.87	0.77	0.37	0.59	0.96
003	meal dehydrated, 20% protein	1-00-024	92	22.0	1.05	0.35	0.41	0.97	1.54	0.98	0.34	1.03	0.88	0.45	0.67	1.13
	BARLEY *Hordeum vulgare*															
004	grain	4-00-549	88	13.5	0.58	0.24	0.28	0.51	0.85	0.44	0.17	0.66	0.42	0.17	0.38	0.64
005	grain, Pacific Coast	4-07-939	89	10.8	0.50	0.22	0.23	0.45	0.67	0.30	0.16	0.53	0.35	0.14	0.34	0.52
006	malt sprouts, dehydrated	5-00-545	94	28.1	1.19	0.25	0.56	1.19	1.76	1.29	0.35	0.98	1.07	0.44	0.65	1.55
	BEET MOLASSES—SEE MOLASSES															
	BEET, SUGAR *Beta vulgaris altissima*															
007	pulp, dehydrated	4-00-669	91	9.7	0.33	0.01	0.22	0.33	0.66	0.66	0.01	0.33	0.44	0.11	0.44	0.44
	BLOOD															
008	meal	5-00-380	92	87.2	3.55	1.35	4.34	0.95	10.86	6.92	0.97	6.00	3.89	1.07	2.09	7.12
009	spray dehydrated (blood flour)	5-00-381	93	93.0	3.88	0.78	5.59	0.98	11.86	8.04	0.95	6.36	3.93	1.13	2.44	8.13
	BREAD—SEE WHEAT															
	BREWERS															
010	grains, dehydrated	5-02-141	92	29.4	1.38	0.38	0.56	1.68	2.70	0.95	0.59	1.56	1.01	0.40	1.30	1.75
	BUTTERMILK *Bos taurus*															
011	condensed (cattle)	5-01-159	29	36.9	—	—	—	—	—	—	—	—	—	—	—	—
	CASEIN															
012	dehydrated	5-01-162	91	92.7	3.85	0.34	2.86	6.32	9.71	7.88	3.10	5.31	4.32	1.19	5.41	7.40
	CATTLE *Bos taurus*															
013	kidneys, fresh	5-01-165	27	59.5	—	—	—	—	—	—	—	—	—	—	—	—
014	lips, fresh	5-07-940	30	60.0	—	—	—	—	—	—	—	—	—	—	—	—
015	livers, fresh	5-01-166	28	69.6	—	—	—	—	—	—	—	—	—	—	—	—
016	lungs, fresh	5-07-941	21	65.0	3.11	0.66	1.13	1.37	2.74	2.59	0.61	1.46	1.42	0.28	1.04	1.89
017	spleens, fresh	5-07-942	24	68.7	—	—	—	—	—	—	—	—	—	—	—	—
018	tripe, fresh	5-09-806	33	46.1	—	—	—	—	—	—	—	—	—	—	—	—
019	udders, fresh	5-07-943	20	58.6	—	—	—	—	—	—	—	—	—	—	—	—
	CEREALS															
020	distillers grains, dehydrated	5-02-144	93	29.5	1.18	0.41	0.57	1.25	2.88	0.88	0.50	1.12	0.88	0.22	0.79	1.32
	CHICKEN *Gallus domesticus*															
021	whole, fresh, day-old	5-07-946	13	57.9	—	—	—	—	—	—	—	—	—	—	—	—
022	broilers, whole, fresh	5-07-945	24	76.5	—	—	—	—	—	—	—	—	—	—	—	—
023	hens, whole, fresh	5-07-950	33	60.3	2.59	0.62	0.77	2.00	2.46	1.66	0.74	1.26	1.35	0.31	0.74	1.82
024	eggs with shells, fresh	5-01-213	43	22.4	—	—	—	—	—	—	—	—	—	—	—	—
025	feet, fresh	5-07-947	33	54.5	—	—	—	—	—	—	—	—	—	—	—	—
026	gizzards, fresh	5-07-948	25	80.4	—	—	—	—	—	—	—	—	—	—	—	—
027	heads, fresh	5-07-949	33	57.6	—	—	—	—	—	—	—	—	—	—	—	—
028	by-product, fresh	5-07-951	44	48.3	—	—	—	—	—	—	—	—	—	—	—	—
029	viscera with heads, fresh	5-07-952	34	43.7	—	—	—	—	—	—	—	—	—	—	—	—
	CITRUS *Citrus* spp															
030	pomace without fines, dehydrated (pulp)	4-01-237	91	6.7	0.27	0.12	0.10	0.20	0.34	0.22	0.10	0.20	0.20	0.07	—	0.28
	COCONUT *Cocos nucifera*															
031	kernels with coats, meal mechanically extracted (copra meal)	5-01-572	92	22.4	2.60	0.23	0.46	0.68	1.36	0.64	0.34	0.91	0.66	0.22	0.57	1.02
032	kernels with coats, meal solvent extracted (copra meal)	5-01-573	91	23.4	2.65	0.27	0.41	0.91	1.59	0.66	0.35	0.95	0.73	0.22	0.63	1.14
	CORN, DENT YELLOW *Zea mays indentata*															
033	grain	4-02-935	89	10.9	0.48	0.25	0.29	0.39	1.37	0.28	0.19	0.54	0.40	0.09	0.43	0.50
034	grain, boiled dehydrated	4-02-853	88	10.5	—	—	—	—	—	—	—	—	—	—	—	—
035	grain, flaked	4-28-244	89	11.2	0.49	0.28	0.31	0.38	1.40	0.28	0.17	0.50	0.39	—	0.44	0.53
036	grits by-product (hominy feed)	4-03-011	90	11.5	0.52	0.16	0.22	0.43	0.94	0.42	0.18	0.36	0.44	0.12	0.55	0.55
037	distillers grains with solubles, dehydrated	5-28-236	92	25.0	1.05	0.32	0.70	1.52	2.43	0.77	0.54	1.64	1.01	0.19	0.76	1.63
038	distillers solubles, dehydrated	5-28-237	93	29.7	1.05	0.48	0.73	1.43	2.54	0.99	0.60	1.60	1.10	0.26	0.94	1.67
039	germs, meal wet milled solvent extracted	5-28-240	91	22.3	1.43	0.44	0.76	0.76	1.97	0.98	0.64	0.98	1.19	0.21	0.76	1.31
040	gluten, meal	5-28-241	91	46.8	1.53	0.73	1.06	2.46	7.92	0.87	1.14	3.05	1.56	0.23	1.11	2.40
	CORN, DENT WHITE *Zea mays indentata*															
041	grits by-product (hominy feed)	4-02-990	90	11.8	0.48	0.12	0.21	0.37	0.91	0.40	0.13	0.37	0.37	0.12	0.44	0.48
	CORN, FLINT *Zea mays indurata*															
042	grain	4-02-948	89	11.1	—	—	—	—	—	0.30	0.20	—	—	0.10	—	—
	COTTON *Gossypium* spp															
043	seeds, meal mechanically extracted, 41% protein	5-01-617	93	44.3	4.51	0.78	1.15	1.56	2.50	1.73	0.62	2.35	1.44	0.57	1.01	2.05
044	seeds, meal prepressed solvent extracted, 41% protein	5-07-872	91	45.6	4.71	0.90	1.27	1.59	2.67	2.01	0.62	2.21	1.48	0.56	1.27	2.20

Entry Number	Feed Name Description	International Feed Number	Dry Matter (%)	Crude Protein (%)	Arginine (%)	Cystine (%)	Histidine (%)	Isoleucine (%)	Leucine (%)	Lysine (%)	Methionine (%)	Phenylalanine (%)	Threonine (%)	Tryptophan (%)	Tyrosine (%)	Valine (%)
045	seeds, meal solvent extracted, 41% protein	5-01-621	91	45.2	4.62	0.85	1.21	1.67	2.56	1.86	0.64	2.46	1.52	0.61	1.13	2.06
046	seeds without hulls, meal prepressed solvent extracted, 50% protein	5-07-874	93	54.0	5.20	1.13	1.30	1.59	2.45	1.82	0.81	2.81	1.78	0.67	0.87	2.32
	FATS AND OILS															
047	fat, animal-poultry	4-00-409	99	—	—	—	—	—	—	—	—	—	—	—	—	—
048	fat (lard), swine	4-04-790	99	—	—	—	—	—	—	—	—	—	—	—	—	—
049	oil, soybean	4-07-983	99	1.4	—	—	—	—	—	—	—	—	—	—	—	—
	FISH															
050	livers, meal mechanically extracted	5-01-968	93	67.7	—	—	—	—	—	—	—	—	—	—	—	—
051	solubles, condensed	5-01-969	50	65.3	3.25	0.54	2.85	2.06	3.72	3.71	1.42	2.04	1.73	0.68	0.87	2.43
052	solubles, dehydrated	5-01-971	93	69.2	3.29	0.66	2.28	2.21	3.21	3.79	1.27	1.65	1.46	0.64	0.92	2.26
	FISH, ALEWIFE *Pomolobus pseudoharengus*															
053	meal mechanically extracted	5-09-830	90	62.6	5.98	0.62	2.44	4.34	6.90	7.00	2.44	3.70	4.20	0.78	3.45	4.58
054	whole, fresh	5-07-964	26	75.8	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, ANCHOVY *Engraulis ringen*															
055	meal mechanically extracted	5-01-985	92	71.2	4.11	0.66	1.76	3.38	5.43	5.49	2.16	3.03	3.00	0.82	2.44	3.81
	FISH, CARP *Cyprinus carpio*															
056	meal boiled	5-09-831	90	58.6	—	—	—	—	—	—	1.56	—	—	—	—	—
057	whole, fresh	5-01-986	31	61.9	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, CATFISH *Ictalurus* spp															
058	boiled	5-09-833	40	69.7	—	—	—	—	—	—	—	—	—	—	—	—
059	cuttings, fresh	5-09-832	34	64.5	—	—	—	—	—	—	—	—	—	—	—	—
060	meal mechanically extracted	5-09-835	92	55.3	—	—	—	—	—	—	—	—	—	—	—	—
061	whole, fresh	5-07-965	22	94.3	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, FLOUNDER Bothidae (family)-Pleuronectidae (family)															
062	whole, fresh	5-01-996	17	88.2	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, HADDOCK *Melanogrammus aeglefinus*															
063	whole, fresh	5-07-966	20	94.4	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, HAKE *Merluccius* spp-*Urophycis* spp															
064	whole, boiled	5-07-967	26	57.9	—	—	—	—	—	—	—	—	—	—	—	—
065	whole, boiled acidified	5-07-968	25	—	—	—	—	—	—	—	—	—	—	—	—	—
066	whole, fresh	5-07-969	20	67.1	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, HERRING *Clupea harengus*															
067	meal mechanically extracted	5-02-000	92	78.3	5.02	0.81	1.80	3.41	5.64	5.83	2.27	2.94	3.16	0.83	2.39	4.68
068	whole, fresh	5-01-999	26	70.4	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, MACKEREL, ATLANTIC *Scomber scombrus*															
069	whole, fresh	5-07-971	30	53.3	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, MACKEREL, PACIFIC *Scomber japonicus*															
070	whole, fresh	5-07-972	30	72.5	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, MENHADEN *Brevoortia tyrannus*															
071	meal mechanically extracted	5-02-009	92	66.7	4.09	0.61	1.58	3.15	4.89	5.15	1.91	2.69	2.73	0.71	2.12	3.52
	FISH, REDFISH *Sciaenops ocellata*															
072	meal mechanically extracted	5-07-973	93	61.0	4.36	0.44	1.39	3.72	5.22	7.04	1.94	2.68	2.79	0.65	1.81	3.55
073	whole, fresh	5-08-191	24	68.1	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, ROCKFISH *Sebastodes* spp															
074	whole, fresh	5-07-974	21	89.6	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, SALMON *Oncorhynchus* spp															
075	whole, fresh	5-02-011	36	57.6	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, SALMON *Oncorhynchus* spp-*Salmo* spp															
076	meal mechanically extracted	5-02-012	93	66.6	5.59	0.75	—	—	—	8.17	1.72	—	—	0.54	—	—
	FISH, SARDINE *Clupea* spp-*Sardinops* spp															
077	meal mechanically extracted	5-02-015	93	70.0	2.90	0.86	1.93	3.59	—	6.34	2.16	2.15	2.79	0.54	3.00	4.40
	FISH, SMELT *Osmerus* spp															
078	whole, fresh	5-07-975	21	88.6	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, SOLE Soleidae (family)															
079	whole, fresh	5-07-976	20	65.6	—	—	—	—	—	—	—	—	—	—	—	—
	FISH, TUNA *Thunnus thynnus-Thunnus albacares*															
080	meal mechanically extracted	5-02-023	93	63.6	3.60	0.50	1.89	2.64	4.09	4.54	1.58	2.32	2.49	0.62	1.82	2.98
081	process residue, ground	5-07-977	94	56.5	3.65	0.45	1.56	2.53	4.09	4.14	1.56	2.33	2.46	0.60	2.17	3.01

Entry Number	Feed Name Description	International Feed Number	Dry Matter (%)	Crude Protein (%)	Arginine (%)	Cystine (%)	Histidine (%)	Isoleucine (%)	Leucine (%)	Lysine (%)	Methionine (%)	Phenylalanine (%)	Threonine (%)	Tryptophan (%)	Tyrosine (%)	Valine (%)
	FISH, TURBOT *Psetta maxima*															
082	whole, fresh	5-07-978	25	57.3	—	—	—	—	—	—	—	—	0.41	—	0.44	—
	FISH, WHITE Gadidae (family)-Lophiidae (family)-Rajidae (family)															
083	meal mechanically extracted	5-02-025	91	68.2	4.41	0.82	1.47	2.98	4.78	4.96	1.84	2.50	2.82	0.73	2.00	3.31
	FISH, WHITING *Gadus merlangus*															
084	whole, fresh	5-07-979	23	69.9	—	—	—	—	—	—	—	—	—	—	—	—
	GROUNDNUT—SEE PEANUT															
	HORSE *Equus caballus*															
085	meat, fresh	5-07-980	29	63.6	—	—	—	—	—	—	—	—	—	—	—	—
086	meat with bone, fresh	5-07-981	34	51.4	—	—	—	—	—	—	—	—	—	—	—	—
	LIVERS															
087	meal	5-00-389	92	71.4	4.37	1.01	1.60	3.36	5.74	5.63	1.32	3.15	2.70	0.74	1.84	4.49
	MEAT															
088	meal rendered	5-00-385	94	54.8	3.84	0.70	1.02	1.86	3.40	3.45	0.75	1.94	1.75	0.37	1.02	2.68
089	with blood, meal rendered (tankage)	5-00-386	92	64.5	3.90	0.49	1.99	2.09	5.56	4.06	0.79	2.76	2.52	0.70	1.40	4.10
090	with bone, meal rendered	5-00-388	93	54.1	3.75	0.53	1.04	1.76	3.29	3.11	0.70	1.83	1.77	0.32	0.85	2.63
	MILK *Bos taurus*															
091	dehydrated (cattle)	5-01-167	96	26.5	0.96	—	0.75	1.39	2.67	2.35	0.64	1.39	1.07	0.43	1.39	1.81
092	skimmed dehydrated (cattle)	5-01-175	94	35.8	1.23	0.48	0.92	2.32	3.53	2.70	0.96	1.66	1.67	0.46	1.22	2.43
	MOLASSES *Beta vulgaris altissima*															
093	beet, sugar, molasses, more than 48% invert sugar more than 79.5 degrees brix	4-00-668	78	8.5	—	—	—	—	—	—	—	—	—	0.31	—	—
	MOLASSES *Saccharum officinarum*															
094	sugarcane, molasses, dehydrated	4-04-695	94	10.3	—	—	—	—	—	—	—	—	—	—	—	—
095	sugarcane, molasses, more than 46% invert sugars more than 79.5 degrees brix (black strap)	4-04-696	75	5.8	—	—	—	—	—	—	—	—	—	—	—	—
	MILLET *Setaria* spp															
096	grain	4-03-098	90	13.5	0.39	0.13	0.26	0.54	1.37	0.29	0.33	0.66	0.49	0.14	—	0.69
	OATS *Avena sativa*															
097	cereal by-product, less than 4% fiber (feeding oat meal) (oat middlings)	4-03-303	91	16.4	0.92	0.28	0.33	0.60	1.17	0.59	0.23	0.76	0.53	0.22	0.79	0.89
098	grain	4-03-309	89	13.3	0.79	0.22	0.21	0.49	0.91	0.44	0.19	0.58	0.40	0.17	0.52	0.63
099	grain, grade 1 heavy 46.3 kg/hl (36 lb/bushel)	4-03-312	89	14.2	—	—	—	—	—	—	—	—	—	—	—	—
100	grain, grade 1 43.8 kg/hl (34 lb/bushel)	4-03-313	90	13.3	0.89	0.24	0.22	0.59	1.01	0.56	0.20	0.67	0.45	0.18	0.59	0.78
101	grain, grade 2 41.2 kg/hl (32 lb/bushel)	4-03-316	89	12.8	—	—	—	—	—	—	—	—	—	—	—	—
102	groats	4-03-331	90	17.7	0.96	0.23	0.28	0.61	1.16	0.59	0.23	0.75	0.50	0.21	0.64	0.84
103	groats, boiled ground	4-07-982	91	18.4	—	—	—	—	—	—	—	—	—	—	—	—
104	hulls	1-03-281	92	3.9	0.19	0.07	0.09	0.19	0.30	0.19	0.09	0.18	0.18	0.09	0.19	0.22
	PEA *Pisum* spp															
105	seeds, ground	5-06-623	89	24.9	1.54	0.19	0.79	1.21	1.98	1.76	0.34	1.43	1.03	0.26	—	1.43
	PEANUT *Arachis hypogaea*															
106	kernels, meal mechanically extracted (peanut meal)	5-03-649	93	52.0	5.46	0.81	1.17	1.83	3.26	1.62	0.53	2.53	1.34	0.51	1.79	2.24
107	kernels, meal solvent extracted (peanut meal)	5-03-650	92	52.3	4.95	0.79	1.03	1.91	2.94	1.93	0.46	2.22	1.26	0.52	1.65	2.04
	POTATO *Solanum tuberosum*															
108	tubers, dehydrated	4-07-850	91	8.9	0.28	0.08	0.17	0.28	0.66	0.45	0.11	0.44	0.52	0.15	—	0.40
	POULTRY															
109	feathers, hydrolyzed	5-03-795	93	91.3	7.58	3.48	1.06	4.37	7.46	2.49	0.59	3.28	4.27	0.86	2.49	6.97
110	necks with backs with wings with legs, fresh	5-03-797	100	—	—	—	—	—	—	—	—	—	—	—	—	—
111	trimmings, fresh	5-16-424	100	—	—	—	—	—	—	—	—	—	—	—	—	—
	RICE *Oryza sativa*															
112	bran with germs (rice, bran)	4-03-928	91	14.1	0.79	0.11	0.25	0.51	0.77	0.54	0.26	0.49	0.47	0.11	0.76	0.76
113	grain, ground (ground rough rice) (ground paddy rice)	4-03-938	89	8.9	0.64	0.13	0.15	0.34	0.63	0.30	0.18	0.37	0.27	0.12	0.60	0.50
114	groats, ground	4-03-935	88	9.6	—	—	—	—	—	—	—	—	—	—	—	—
115	groats, polished (rice, polished)	4-03-942	89	8.2	0.50	0.11	0.20	0.50	0.80	0.32	0.28	0.60	0.40	0.11	0.70	0.60
116	polishings	4-03-943	90	13.4	0.57	0.14	0.19	0.39	0.78	0.58	0.22	0.42	0.38	0.11	0.46	0.80
	RYE *Secale cereale*															
117	grain	4-04-047	88	13.8	0.61	0.21	0.29	0.53	0.80	0.48	0.19	0.64	0.41	0.13	0.30	0.64
	SEAWEED, KELP Laminariales (order)-Fucales (order)															
118	whole, sun-cured	1-04-190	89	9.6	—	—	—	—	—	—	—	—	—	—	—	—
	SHRIMP *Pandalus* spp-*Penaeus* spp															
119	process residue, meal (shrimp meal)	5-04-226	90	44.2	2.79	0.66	1.07	1.86	2.98	2.41	0.91	1.76	1.58	0.40	1.47	2.03

Entry Number	Feed Name Description	International Feed Number	Dry Matter (%)	Crude Protein (%)	Arginine (%)	Cystine (%)	Histidine (%)	Isoleucine (%)	Leucine (%)	Lysine (%)	Methionine (%)	Phenylalanine (%)	Threonine (%)	Tryptophan (%)	Tyrosine (%)	Valine (%)
	SORGHUM *Sorghum bicolor*															
120	grain	4-04-383	90	12.4	0.43	0.22	0.26	0.50	1.80	0.28	0.15	0.62	0.40	0.12	0.46	0.58
	SORGHUM, MILO *Sorghum bicolor subglabrescens*															
121	grain	4-04-444	89	11.3	0.42	0.15	0.26	0.49	1.46	0.26	0.16	0.55	0.39	0.11	0.40	0.59
	SOYBEAN *Glycine max*															
122	seeds, meal mechanically extracted	5-04-600	90	47.7	3.41	0.63	1.26	2.92	4.02	3.10	0.72	2.45	1.92	0.68	1.72	2.53
123	seeds, meal solvent extracted	5-04-604	90	49.9	3.38	0.83	1.19	2.27	3.65	2.99	0.58	2.36	1.85	0.71	1.45	2.25
124	seeds without hulls, meal solvent extracted	5-04-612	90	55.1	4.07	0.83	1.35	2.73	4.14	3.52	0.79	2.71	2.15	0.77	1.66	2.82
	SUGARCANE, MOLASSES—SEE MOLASSES															
	SUNFLOWER, COMMON *Helianthus annuus*															
125	seeds without hulls, meal mechanically extracted	5-04-738	93	44.6	3.72	0.74	0.97	1.90	2.66	1.73	1.01	1.94	1.47	0.54	1.08	2.17
126	seeds without hulls, meal solvent extracted	5-04-739	93	49.8	4.75	0.79	1.32	2.42	4.12	2.06	1.25	2.54	2.07	0.65	1.49	2.60
	SWINE *Sus scrofa*															
127	kidneys, fresh	5-09-813	22	73.8	—	—	—	—	—	—	—	—	—	—	—	—
128	livers, fresh	5-04-792	30	68.8	—	—	—	—	—	—	—	—	—	—	—	—
129	lungs, fresh	5-26-140	16	88.6	—	—	—	—	—	—	—	—	—	—	—	—
	TOMATO *Lycopersicon esculentum*															
130	pomace, dehydrated	5-05-041	92	23.5	1.30	—	0.43	0.76	1.85	1.74	0.11	0.98	0.76	0.22	0.98	1.09
	TORULA DRIED YEAST—SEE YEAST, TORULA															
	TURKEY *Meleagris gallopavo*															
131	flesh, fresh	5-24-927	32	75.7	—	—	—	—	—	—	—	—	—	—	—	—
132	viscera, fresh	5-08-816	28	44.0	—	—	—	—	—	—	—	—	—	—	—	—
133	viscera, fresh, chick	5-07-985	28	54.7	—	—	—	—	—	—	—	—	—	—	—	—
	WHALE *Balaena glacialis-Balaenoptera* spp-*Physeter catodon*															
134	meat, fresh	5-07-986	29	70.8	—	—	—	—	—	—	—	—	—	—	—	—
	WHEAT *Triticum aesticum*															
135	bran	4-05-190	89	17.1	1.09	0.36	0.44	0.57	1.03	0.65	0.22	0.62	0.51	0.28	0.48	0.78
136	bread, dehydrated	4-07-944	95	13.0	—	0.19	—	—	—	0.22	0.19	—	—	—	—	—
137	flour, less than 1.5% fiber (wheat feed flour)	4-05-199	88	13.4	0.49	0.35	0.28	0.53	0.99	0.28	0.21	0.69	0.37	0.14	0.39	0.57
138	flour by-product, less than 4% fiber (wheat red dog)	4-05-203	88	17.4	1.09	0.42	0.46	0.62	1.20	0.67	0.26	0.75	0.57	0.22	0.52	0.82
139	flour by-product, less than 7% fiber (wheat shorts)	4-05-201	88	18.6	1.34	0.41	0.51	0.66	1.23	0.59	0.31	0.76	0.68	0.24	0.53	0.93
140	germs, ground	5-05-218	88	28.1	2.12	0.54	0.74	1.02	1.75	1.74	0.49	1.07	1.09	0.34	0.83	1.32
141	grain	4-05-211	89	16.0	0.67	0.31	0.32	0.53	0.98	0.41	0.20	0.68	0.42	0.17	0.43	0.64
142	grain, hard red spring	4-05-258	88	17.2	0.67	0.30	0.27	0.61	1.00	0.40	0.21	0.75	0.41	0.16	0.58	0.67
143	grain, hard red winter	4-05-268	88	14.4	0.73	0.36	0.34	0.58	1.00	0.41	0.24	0.71	0.42	0.19	0.49	0.67
144	grain, soft red winter	4-05-294	88	13.0	0.73	0.41	0.36	0.51	1.02	0.41	0.24	0.72	0.44	0.30	0.43	0.65
145	grain, soft white winter	4-05-337	89	11.3	0.52	0.29	0.24	0.46	0.80	0.35	0.17	0.53	0.35	0.14	0.41	0.52
146	grain, soft white winter, Pacific Coast	4-08-555	89	11.2	0.50	0.27	0.22	0.45	0.84	0.34	0.16	0.54	0.34	0.13	0.41	0.52
147	grain screenings	4-05-216	89	15.8	0.44	0.14	0.34	0.52	0.83	0.43	0.17	0.55	0.38	0.14	0.26	0.62
148	grits, cracked screened	4-07-852	90	12.7	—	—	—	—	—	—	—	—	—	—	—	—
149	mill run, less than 9.5% fiber	4-05-206	90	17.2	1.04	0.26	0.44	0.78	1.33	0.64	0.37	—	0.56	0.23	0.56	0.89
	WHEAT, DURUM *Triticum durum*															
150	grain	4-05-224	88	15.9	0.68	0.15	0.32	0.57	1.54	1.08	0.17	0.66	0.43	0.30	0.36	0.65
	WHEY *Bos taurus*															
151	dehydrated (cattle)	4-01-182	93	14.2	0.36	0.32	0.18	0.84	1.26	1.00	0.20	0.37	0.96	0.19	0.26	0.73
152	low lactose, dehydrated (dried whey product) (cattle)	4-01-186	93	17.9	0.64	0.46	0.29	1.03	1.65	1.50	0.43	0.59	1.01	0.29	0.49	0.93
	YEAST, BREWERS *Saccharomyces cerevisiae*															
153	dehydrated	7-05-527	93	46.9	2.35	0.53	1.17	2.37	3.45	3.33	0.79	1.96	2.27	0.55	1.80	2.52
	YEAST, PETROLEUM *Candida utilis*															
154	oil residue, solvent extracted dehydrated	7-09-536	92	51.1	—	—	—	—	—	—	—	—	—	—	—	—
	YEAST, PRIMARY *Saccharomyces cerevisiae*															
155	dehydrated	7-05-533	93	51.5	2.51	0.54	6.05	3.89	4.00	4.10	1.08	2.70	2.70	0.43	—	3.46
	YEAST, TORULA *Torulopsis utilis*															
156	dehydrated	7-05-534	93	52.7	2.83	0.65	1.42	3.06	3.78	4.01	0.83	3.06	2.83	0.56	2.14	3.17

TABLE 9 Weight-Unit Conversion Factors

Units Given	Units Wanted	For Conversion Multiply by
lb	g	453.6
lb	kg	0.4536
oz	g	28.35
kg	lb	2.2046
kg	mg	1,000,000.
kg	g	1,000.
g	mg	1,000.
g	µg	1,000,000.
mg	µg	1,000.
mg/g	mg/lb	453.6
mg/kg	mg/lb	0.4536
µg/kg	µg/lb	0.4536
Mcal	kcal	1,000.
kcal/kg	kcal/lb	0.4536
kcal/lb	kcal/kg	2.2046
ppm	µg/g	1.
ppm	mg/kg	1.
ppm	mg/lb	0.4536
mg/kg	%	0.0001
ppm	%	0.0001
mg/g	%	0.1
g/kg	%	0.1

TABLE 10 Weight Equivalents

1 lb = 453.6 g = 0.4536 kg = 16 oz
1 oz = 28.35 g
1 kg = 1,000 g = 2.2046 lb
1 g = 1,000 mg
1 mg = 1,000 µg = 0.001 g
1 µg = 0.001 mg = 0.000001 g
1 µg per g or 1 mg per kg is the same as ppm

TABLE 11 Examples of Three Types of Commercial Foods (percent)a

	Dry[b]	Semimoist[b]	Canned[b]
Corn	49.1	–	–
Corn gluten feed	19.0	–	–
Meat and bone meal	19.0	–	–
Meat and meat by-products	–	32.8	65–80
Poultry and poultry by-products	–	–	10–20
Soybean meal	7.5	–	–
Soybean flakes, bran flakes	–	32.3	–
Textured soy protein, soy flour	–	–	10–20
Soluble carbohydrates	–	21.0	–
Animal fat	4.5	1.0	–
Mineral mix[c]	0.8	3.3	0.5
Vitamin mix[c]	0.1	0.3	0.2
Antimycotic and emulsifier	–	3.8	–
Propylene glycol	–	3.0	–
Dried skimmed milk	–	2.5	–

[a] For examples of foods from semipurified and purified sources, refer to papers listed in References under Protein and Amino Acids, and Vitamins.

[b] Courtesy of M. C. Stillions, Agway, Inc.; Gaines Nutrition Center, Gaines Foods, Inc.; and C. A. Banta, Alpo Petfoods, Inc.

[c] Quantities to meet NRC requirements with sufficient overages to compensate for lack of availability and/or losses due to processing and storage.

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