

Keynote Lecture 2

Differences between cats and dogs: a nutritional view

BY VERONIQUE LEGRAND-DEFRETIN

Waltham Centre for Pet Nutrition, Waltham-on-the-Wolds, Melton Mowbray LE14 4RT

Cats and dogs have been associated with man for many centuries, and in modern society they probably represent the two most popular companion animals. In 1992 there were approximately forty million cats and thirty-eight million dogs in Europe, representing 18 and 21% of households respectively. Despite their popularity, the nutritional requirements of both species have only been studied in detail during the last 20 years.

Regarding zoological classification, both cats and dogs fall into the order Carnivora. However, a comparison of the nutritional requirements of the cat and the dog supports the hypothesis that specialization consistent with the evolutionary influence of a strict carnivorous diet has occurred in the cat. In addition, there appears to be more variety in the diets of Canids than in those of Felids. The ancestors of dogs are known to eat mammals, fish, birds and amphibians as well as vegetable matter (berries, apples, pears) whereas wild cats only eat animals (small antelopes, rodents, birds, fish, etc; Röhrs, 1987). Examples of the cat specializations can be summarized as follows:

1. the cat has limited ability to regulate the catabolic enzymes of amino acid metabolism, which causes the cat to require a higher level of dietary protein for maintenance than the dog;
2. the cat has a lower capacity to synthesize the sulphonic acid taurine than the dog and is unable to conjugate bile acids to glycine. Thus the cat, unlike the dog, cannot meet its taurine requirement from dietary S-containing amino acids;
3. the cat cannot synthesize sufficient nicotinic acid from tryptophan because of an increased activity of α -picolinic acid decarboxylase (*EC* 4.1.1.45) leading to the endproduct glutamate rather than nicotinic acid;
4. the cat is unable to convert carotene to retinol and, therefore, cannot satisfy its vitamin A requirements with a herbivorous diet alone;
5. the cat cannot convert sufficient linoleic acid to meet its requirement for arachidonic acid;
6. the cat seems to be unable to cope with high levels of carbohydrate in its diet and appears to be in a constant state of gluconeogenesis.

These feline specific peculiarities (which will be presented in the present paper) appear to confirm that, unlike the dog, the cat is an obligate carnivore and is dependent on a supply of at least some animal-derived materials in its diet.

PROTEIN REQUIREMENT

Total protein

Protein is required in greater amounts by the cat than most other mammals, including dogs (Tables 1 and 2; National Research Council, 1985, 1986). Work conducted at the

Table 1. *Protein requirement (g/MJ diet) of cats and dogs*

	Cat*	Dog†	Cat:dog
Growth	293	230	1.3
Adult maintenance	261	167	1.6

* National Research Council (1986).

† National Research Council (1985).

Table 2. *Minimal essential amino acid requirements of kittens and puppies for growth*

Amino acid (mg/MJ diet)	Kitten*	Puppy†	Kitten:puppy
Arginine	478	327	1.5
Histidine	144	117	1.2
Isoleucine	239	234	1.0
Leucine	574	380	1.5
Lysine	383	335	1.1
Methionine+cysteine	359	253	1.4
Phenylalanine+tyrosine	407	466	0.9
Threonine	335	304	1.1
Tryptophan	72	98	0.7
Valine	287	251	1.1

* National Research Council (1986).

† National Research Council (1985).

Waltham Centre for Pet Nutrition has shown that when all essential amino acids are present at more than adequate concentrations, about 10% protein energy is required to maintain adult cats in protein (N) balance (Burger *et al.* 1984; Burger & Smith, 1987). No such studies exist in dogs. However, the National Research Council (1985) recommends that 6% of energy comes from dispensable amino acids. These studies showed that the higher protein requirement of the cat is not due to an increased requirement for essential amino acids but a need for more protein in total, irrespective of its essential amino acid content. The metabolic explanation seems to lie with the increased activity of the N-catabolizing enzymes. Alanine aminotransferase (*EC* 2.6.1.2) and glutamate dehydrogenase (*EC* 1.4.1.3) activities are greater than those of either the dog or the rat (Table 3; Schaeffer *et al.* 1989). In contrast, the level of enzyme activity for the breakdown of the essential amino acids (e.g. threonine dehydratase (*EC* 4.2.1.16), serine dehydratase (*EC* 4.2.1.13); Rogers *et al.* 1977) is lower in the cat than in rats fed on a high-protein diet. Finally, unlike other mammals that can adjust their rate of protein breakdown, cats seem unable to 'switch off' these mechanisms when presented with a low-protein diet (Table 4; Rogers *et al.* 1977).

Arginine

In addition to a high total protein requirement, the cat's requirements for a number of the individual amino acids are of particular interest. Both cats and dogs show signs of

Table 3. *Effect of dietary protein on hepatic alanine aminotransferase activity (EC 2.6.1.2; $\mu\text{mol/min per g}$) (From Schaeffer et al. 1989)*

(Mean values with their standard errors)

Diet	Kittens		Puppies		Rats	
	Mean	SE	Mean	SE	Mean	SE
Standard-protein	51	2	17	1	15	2
High-protein	63	3	31	2	41	1
Food deprived (5 d)	51	4*	8	1	17†	

* Adult cats.

† Calculated from percentage change when 3 d food deprived.

Table 4. *Comparison of alanine aminotransferase activity (EC 2.6.1.2) in the cat and rat fed on low- and high-protein diets* (From Rogers et al. 1977)*

Diet	Cats	Rats
Low protein level	100	100
Normal protein level	126	340
High protein level	80	1177
Starved (5 d for cats, 3 d for rats)	146	392

* Expressed in per cent of activity measured when fed on the low-protein diet.

hyperammonaemia when fed on an arginine-free diet (Morris & Rogers, 1978*a,b*; Czarnecki & Baker, 1984). However, arginine deficiency is more severe in the cat, since a *single* arginine-free meal results in severe adverse effects 2–5 h later. Hyperammonaemia occurs following an inability to metabolize N compounds via the urea cycle, and in serious cases can lead to death (Morris & Rogers, 1978*a,b*). A comparison of the clinical signs in cats, dogs, ferrets and rats following consumption of an arginine-free diet led Morris (1985) to the conclusion that cats and ferrets are more sensitive to a deficiency of dietary arginine, dogs being intermediate, while growing rats exhibit only a depression in food intake.

The addition of ornithine to an arginine-free diet can prevent hyperammonaemia in kittens and puppies, although other clinical signs such as body-weight loss in kittens are not prevented (Morris & Rogers, 1978*b*; Czarnecki & Baker, 1984). Other studies on citrulline supplementation showed that this latter intermediate of the urea cycle was not as efficiently utilized as arginine (Morris *et al.* 1979; Czarnecki & Baker, 1984). The susceptibility of cats to arginine-free diets seems to be related to the low activities of two enzymes involved in intestinal ornithine synthesis (pyrroline-5-carboxylate synthase (EC 1.5.1.2) and ornithine aminotransferase (EC 2.6.1.13); for further details, see Morris, 1985; Rogers & Phang, 1985). After an overnight fast, depletion of the urea-cycle intermediates occurs in the liver and in the circulation. When the cat is given an arginine-free meal, protein catabolism and amino acid deamination overload the urea cycle which is then unable to dispose of the NH_3 produced. Reliance of the cat on its diet

for precursors of the urea-cycle intermediates, rather than *de novo* synthesis, is indicative of the natural animal-derived diet of the cat.

Sulphur amino acids and taurine

No discussion of the amino acid requirements of companion animals would be complete without mention of the importance of the S amino acids and of taurine in cats. The dietary requirement of cats for methionine and cysteine is higher than that for most other mammals, including the dog (Table 2). It was first thought that the urinary excretion in cats of felinine, a unique branched-chain amino acid, was one of the reasons for this high requirement. However, Rogers (1963) showed that ^{35}S from [^{35}S]methionine or [^{35}S]cysteine was not incorporated into felinine. Thus, if the physiological significance of felinine is territorial marking (urinary excretion of felinine is higher in adult male than in adult female cats) or involvement in the regulation of sterol metabolism, the route of its synthesis is not known. Methionine and cysteine may act to a limited extent as precursors of taurine synthesis. However, the quantity of S amino acids needed for this synthesis does not account for their high requirement (National Research Council, 1986). Cysteine is metabolized by at least four pathways, only one of which involves the oxidation of cysteine followed by decarboxylation of cysteinesulphinat to produce taurine. The other pathways involve desulphydration of cysteine to release pyruvate, NH_3 and S. The direct pathway of desulphydration accounted for 81–88% of the enzyme activity in the tissue of cats fed on a high-protein diet (Park *et al.* 1991). The reasons for cats requiring more S amino acids than dogs are still not explained; one suggestion is that it is related to the thick coat of the cat (MacDonald *et al.* 1984).

The particular importance of taurine in cat nutrition was discovered less than 20 years ago when a taurine deficiency in cats was associated with central retinal degradation (Hayes *et al.* 1975). More recent research suggests that taurine deficiency is also associated with poor reproductive performance in breeding females, poor growth in kittens and dilated cardiomyopathy in adult cats (Sturman *et al.* 1986; Pion *et al.* 1987). Taurine is a β -amino sulphonic acid (2-amino ethane-sulphonic acid) and as such is not present in protein, but its concentration in animal-derived materials is high (National Research Council, 1986). Taurine is an endproduct of S amino acid metabolism and is normally synthesized from cysteine in the liver (Fig. 1). Its main physiological significance is its conjugation with bile acids, its presence in some peptides and its role in the osmoregulation of cells. It is not metabolized as such by cat tissues since endogenous or dietary taurine is excreted in urine without modification. However, taurine can undergo microbial degradation in the gastrointestinal tract. Unlike most other animals, cats are not able to synthesize sufficient taurine to meet their needs due to a low activity of the enzymes cysteinesulphinat decarboxylase (*EC* 4.1.1.29) and cysteine dioxygenase (*EC* 1.13.11.20) (Hardison *et al.* 1977; Knopf *et al.* 1978; for review, see Morris & Rogers, 1992). However, according to current thinking, the high requirement of cats for taurine is more probably linked to bile acid enterohepatic circulation. In mammals, bile acids neosynthesized in the liver or recycled from the intestine are conjugated to glycine or taurine before being secreted into bile. Cats, unlike humans but like rats and dogs, conjugate most of their bile acids to taurine. In addition, cats and dogs, unlike rats, are unable to switch to glycoconjugation when taurine is limiting, and in taurine depletion some free bile acids appear in bile in cats (Hickman *et al.* 1992). Bile salts secreted into

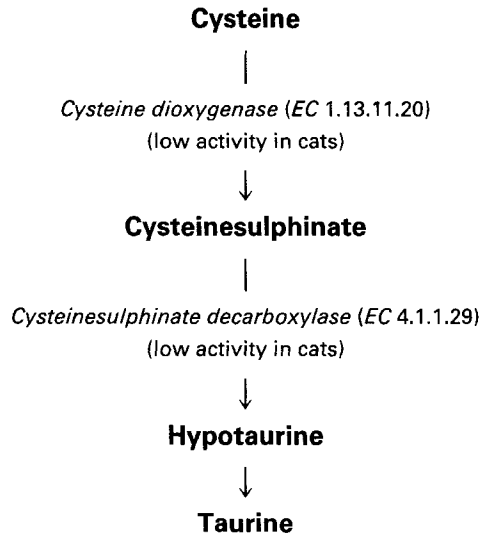


Fig. 1. Synthesis of taurine from cysteine in animals (simplified pathway).

the small intestine will be mainly reabsorbed by passive and active transport in the jejunum and ileum respectively, but they might also be deconjugated and/or metabolized into secondary bile salts by intestinal bacteria. Taurine released by deconjugation may be either reabsorbed from the intestine or lost from the taurine pool. Thus, bile salt production represents an obligatory loss of taurine for the cat (Sturman *et al.* 1978). The cat's requirement for taurine is higher when fed on a canned food than when fed on a dry food (canned food 2200–2500 mg taurine/kg dry matter; Pion *et al.* 1989; Earle & Smith, 1991; dry food 1000 mg taurine/kg dry matter; Douglass *et al.* 1991). The reasons for this discrepancy have yet to be explained but one hypothesis is that the enterohepatic circulation of bile salts is increased in cats fed on canned products since, on a dry matter basis, these generally contain more fat than dry products. If this is the case, deconjugation of bile salts by intestinal bacteria will be increased and, although some taurine is usually reabsorbed from the intestine, its excretion from the digestive tract may be augmented.

VITAMIN REQUIREMENTS

Nicotinic acid

In most animals, nicotinic acid is an endproduct of tryptophan degradation (Fig. 2). Thus, dietary requirements are dependent on the level of tryptophan in the diet. However, the efficiency of this pathway is species-dependent according to the presence and/or activities of the enzymes involved in tryptophan catabolism. In cats, rate of removal of the intermediate α -amino- β -carboxymuconic- ϵ -semialdehyde is so rapid that no nicotinic acid is produced (Da Silva *et al.* 1952; De Castro *et al.* 1957; Ikeda *et al.* 1965). This is due to the high activity of α -picolinic carboxylase which actively transforms tryptophan into glutamic acid even after tryptophan loading (Da Silva *et al.* 1952). Accordingly, although dogs have historically played an important role in the understand-

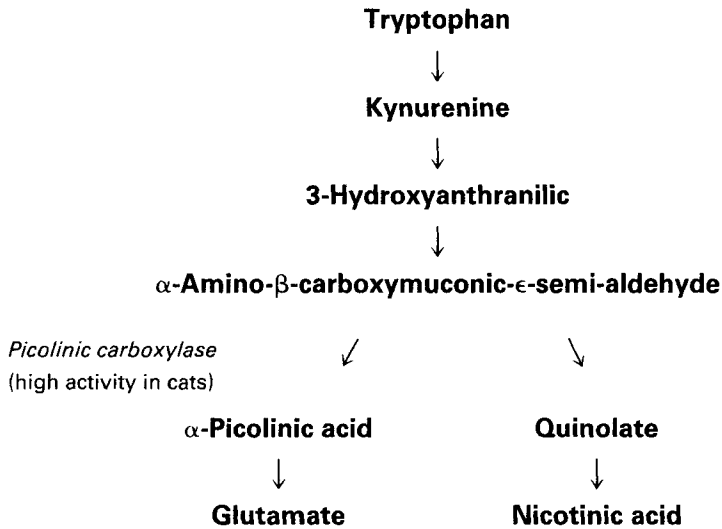


Fig. 2. Synthesis of nicotinic acid from tryptophan in animals (simplified pathway).

ing of the development of pellagra in humans, their requirements for nicotinic acid are smaller than those of cats (growing dog 717 $\mu\text{g}/\text{MJ}$ metabolizable energy (ME), growing cat 1912 $\mu\text{g}/\text{MJ}$ ME; Morris & Rogers, 1989).

Vitamin A

The term vitamin A correctly applies only to retinol, retinaldehyde and retinoic acid. Preformed retinol is found in foods of animal origin and some bacteria, while only small amounts of retinaldehyde and retinoic acid may occur in foods. Dehydroretinol can be reduced to retinol *in vivo*, has half the biological activity of retinol, and can be found as a dietary source in freshwater fish and amphibians. A number of carotenoids, called provitamin A carotenoids, are present in plants, fruits and milk. Among these compounds, β -carotene is the most important because in most animals it has the highest vitamin A activity when transformed by intestinal carotene dioxygenase (*EC* 1.14.99.5) to retinaldehyde (Turner, 1934). However, the activity of this enzyme is low, so that in many species such as dogs and humans a relatively large proportion of ingested β -carotene may appear in the circulation unchanged. In cats, this situation is pushed to an extreme since this enzyme is undetectable in their intestinal mucosa (Gershoff *et al.* 1957). Thus, the requirement of cats for vitamin A can be fulfilled only from food of animal origin, which provides preformed vitamin A.

ESSENTIAL FATTY ACID REQUIREMENTS

Most animals have a requirement for polyunsaturated fatty acids ($n-6$ series) which can be satisfied by the provision of linoleic acid (18:2 $n-6$) in the diet. Alternating steps of desaturation and chain elongation allow production of γ -linolenic (18:3 $n-6$), dihomo- γ -linolenic (20:3 $n-6$) and arachidonic acids (20:4 $n-6$) successively (Fig. 3). Early studies on these metabolic pathways were carried out in rats and it was assumed that they were

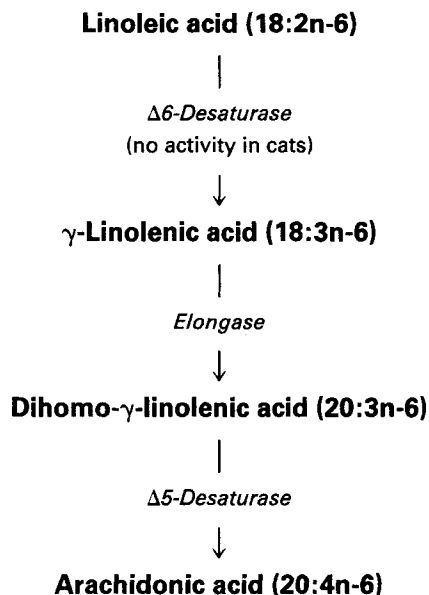


Fig. 3. Synthesis of arachidonic acid from linoleic acid in animals (simplified pathway).

present in other animals. Rivers *et al.* (1975) reported low levels of plasma arachidonic acid in cats fed on purified diets containing linoleic acid but no arachidonic acid. Subsequently, Rivers *et al.* (1976a,c) and Hassam *et al.* (1977) reported that cats were unable to synthesize γ -linolenic acid from linoleic acid or arachidonic acid from dihomogamma-linolenic acid because of a defect in $\Delta 6$ -desaturase and $\Delta 5$ -desaturase activities. Frankel & Rivers (1978) then fed evening primrose oil (rich in γ -linolenic acid) to cats that had previously been fed on safflower seed oil as the only source of fat. After 5 d there were increased levels of γ -linolenic and dihomogamma-linolenic acids but not of arachidonic acid that led these authors to postulate a lack of $\Delta 5$ -desaturase activity. However, after 10 weeks the level of arachidonic acid was increased (Frankel, 1980). Meanwhile, Sinclair *et al.* (1979) showed the ability of cats to synthesize arachidonic acid from γ -linolenic and dihomogamma-linolenic acids but not from linoleic acid and concluded that $\Delta 5$ -desaturase activity was present in the liver of the cat. In conclusion, cats, unlike dogs, have an essential requirement for arachidonic acid because of limiting $\Delta 6$ -desaturase activity. Since arachidonic acid is present only in fat of animal origin, cats must be seen as obligate carnivores.

CARBOHYDRATE METABOLISM

Providing their diet supplies sufficient gluconeogenic amino acids and fat (thus, glycerol), cats can be maintained on a carbohydrate-free diet. In dogs, although carbohydrate can be of importance in the diets of some racing breeds (Legrand-Defretin & Munday, 1993), there is no known minimum requirement for this nutrient. However, petfoods commonly contain moderate to high levels of carbohydrate (i.e. cat foods 70–500 g/kg dry weight, dog foods 60–700 g/kg dry weight). Starch, sucrose and lactose are the main dietary carbohydrates, although lactose and sucrose, present in milk and

sugar cane respectively, are less common in the diets of cats and dogs. In addition, it has been reported that both adult cats and dogs can exhibit lactose intolerance characterized by severe diarrhoea (Morris *et al.* 1977; Mundt & Meyer, 1989). Cats also differ from dogs in their carbohydrate metabolism since they appear to be in a constant state of gluconeogenesis. The concentration of hexokinase (EC 2.7.1.1) in the feline liver is similar to that of other omnivorous animals but glucokinase (EC 2.7.1.2) activity is lower, suggesting that cats will not be able to handle high-carbohydrate diets (Ballard, 1965). Pancreatic amylase (EC 3.2.1.1) activity is approximately three times higher in the dog than in the cat and high levels of dietary starch stimulate intestinal amylase activity to a greater extent in dogs than in cats (Meyer & Kienzle, 1991). Cats can maintain their plasma glucose levels when starved after having been fed on a high-protein diet, whereas cats previously fed on a high-carbohydrate diet showed decreased glucosaemia (Kettlehut *et al.* 1978). Increasing the protein level of cat rations does not stimulate the activity of phosphoenolpyruvate carboxykinase (EC 4.1.1.32), a key gluconeogenic enzyme (Kettlehut *et al.* 1978). The activity of another gluconeogenic enzyme, hepatic serine-pyruvate aminotransferase (EC 2.6.1.51), has been reported to be very high in cats and other carnivores (Rowell *et al.* 1979). Finally, both glucagon and insulin seem to be more responsive to amino acid stimuli than to glucose (for review, see Morris & Rogers, 1989).

CONCLUSION

Although the present review of the nutritional differences between dogs and cats is not intended to be exhaustive, the examples of feline peculiarities presented here illustrate the obligate carnivorous nature of this animal. The field of carnivorous nutrition is wide and has not been extensively studied. However, it can be suggested that the cat could be used as a model to illustrate and show well-established pathways in a new light. For example, lions, like cats, are unable to desaturate linoleic acid (Rivers *et al.* 1976b). It is believed that the lack of adaptation of cats to changes in dietary composition is the consequence of low evolutionary pressure. Although both cats and dogs have been associated with humans for millennia, the latter have developed a dependence on humans for finding food whereas the former have kept their hunting nature. In terms of practical feeding, it seems evident that cat food should contain animal-derived raw materials to ensure that all their requirements are met.

REFERENCES

- Ballard, F. J. (1965). Glucose utilization in mammalian liver. *Comparative Biochemistry & Physiology* **14**, 437–443.
- Burger, I. H., Blaza, S. E., Kendall, P. T. & Smith, P. M. (1984). The protein requirement of adult cats for maintenance. *Feline Practice* **14**, 8–14.
- Burger, I. H. & Smith, P. M. (1987). Amino acid requirements of adult cats. In *Nutrition, Malnutrition and Dietetics in the Dog and Cat*, pp. 49–51 [H. Meyer, E. Kienzle and A. T. B. Edney, editors]. London: British Veterinary Association.
- Czarnecki, G. L. & Baker, D. H. (1984). Urea-cycle function in the dog with emphasis on the role of arginine. *Journal of Nutrition* **114**, 581–590.
- Da Silva, A. C., Fried, R. & De Angelis, R. C. (1952). The domestic cat as a laboratory animal for experimental nutrition studies. *Journal of Nutrition* **46**, 399–409.
- De Castro, F. T., Brown, R. R. & Price, J. M. (1957). The intermediary metabolism of tryptophan by cat and rat tissue preparations. *Journal of Biological Chemistry* **228**, 777–784.

- Douglass, G. M., Fern, E. B. & Brown, R. C. (1991). Feline plasma and whole blood taurine levels as influenced by commercial dry and canned diets. *Journal of Nutrition* **121**, S179–S180.
- Earle, K. E. & Smith, P. M. (1991). The effect of dietary taurine content on the plasma taurine concentration of the cat. *British Journal of Nutrition* **66**, 227–235.
- Frankel, T. L. (1980). Essential fatty acid deficiency in the cat (*Felis catus* L.). PhD Thesis. Wolfson College, University of Cambridge.
- Frankel, T. L. & Rivers, J. P. W. (1978). The nutritional and metabolic impact of γ -linoleic acid (18:3n-6) on cats deprived of minimal lipid. *British Journal of Nutrition* **39**, 227–231.
- Gershoff, S. N., Andrus, S. B., Hegsted, D. M. & Lentini, E. A. (1957). Vitamin A deficiency in cats. *Laboratory Investigation* **6**, 227–240.
- Hardison, W. G. M., Wood, C. A. & Proffitt, J. H. (1977). Quantification of taurine synthesis in the intact rat and cat liver. *Proceedings of the Society for Experimental Biology and Medicine* **155**, 55–58.
- Hassam, A. G., Rivers, J. P. W. & Crawford, M. A. (1977). The failure of the cat to desaturate linoleic acid; its nutritional implication. *Nutrition and Metabolism* **21**, 321–328.
- Hayes, K. C., Carey, R. E. & Schmidt, S. Y. (1975). Retinal degeneration associated with taurine deficiency in the cat. *Science* **188**, 949–951.
- Hickman, M. A., Morris, J. G. & Rogers, Q. R. (1992). Intestinal taurine and the enterohepatic circulation of taurocholic acid in the cat. *Advances in Experimental Medicine and Biology* **315**, 45–54.
- Ikeda, M., Tsuji, H., Nakamura, S., Ichiyama, A., Nichizuka, Y. & Hayashi, O. (1965). Studies on the biosynthesis of nicotinamide adenine dinucleotide from tryptophan in mammals. *Journal of Biological Biochemistry* **240**, 1395–1401.
- Kettlehut, I. C., Foss, M. C. & Migliorini, R. H. (1978). Glucose homeostasis in a carnivorous animal (cat) and in rats fed a high-protein diet. *American Journal of Physiology* **239**, R115–R121.
- Knopf, K., Sturman, J. A., Armstrong, M. & Hayes, K. C. (1978). Taurine: an essential nutrient for the cat. *Journal of Nutrition* **108**, 773–778.
- Legrand-Defretin, V. & Munday, H. S. (1993). Feeding cats and dogs for life. In *The Waltham Book of Companion Animal Nutrition*, pp. 57–68 [I. H. Burger, editor]. Oxford: Pergamon Press.
- MacDonald, M. L., Rogers, Q. R. & Morris, J. G. (1984). Nutrition of the domestic cat, a mammalian carnivore. *Annual Review of Nutrition* **4**, 521–562.
- Meyer, H. & Kienzle, E. (1991). Dietary protein and carbohydrates: relationship to clinical disease. In *Proceedings of Purina International Symposium* (in association with Eastern States Veterinary Conference, 1991), pp. 13–26. Gainesville: Eastern States Veterinary Association.
- Morris, J. G. (1985). Nutritional and metabolic responses to arginine deficiency in carnivores. *Journal of Nutrition* **115**, 524–531.
- Morris, J. G. & Rogers, Q. R. (1978a). Ammonia intoxication in the near-adult cat as a result of a dietary deficiency of arginine. *Science* **199**, 431–432.
- Morris, J. G. & Rogers, Q. R. (1978b). Arginine: an essential amino acid for the cat. *Journal of Nutrition* **108**, 1944–1953.
- Morris, J. G. & Rogers, Q. R. (1989). Comparative aspects of nutrition and metabolism of dogs and cats. In *Nutrition of the Dog and Cat. Waltham Symposium 7*, pp. 35–66 [I. H. Burger and J. P. W. Rivers, editors]. Cambridge: Cambridge University Press.
- Morris, J. G. & Rogers, Q. R. (1992). The metabolic basis for the taurine requirement of cats. *Advances in Experimental Medicine and Biology* **315**, 33–44.
- Morris, J. G. & Rogers, Q. R., Winterrowd, D. L. & Kamikawa, E. M. (1979). The utilization of ornithine and citrulline by the growing kitten. *Journal of Nutrition* **109**, 724–729.
- Morris, J. G., Trudell, J. & Pencovic, T. (1977). Carbohydrate digestion by the domestic cat (*Felis catus*). *British Journal of Nutrition* **37**, 365–373.
- Mundt, H.-C. & Meyer, H. (1989). Pathogenesis of lactose-induced diarrhoea and its prevention by enzymatic splitting of lactose. In *Nutrition of the Dog and Cat, Waltham Symposium 7*, pp. 267–274 [I. H. Burger and J. P. W. Rivers, editors]. Cambridge: Cambridge University Press.
- National Research Council (1985). *Nutrition Requirements of Dogs*. Washington D.C.: National Academy Press.
- National Research Council (1986). *Nutrient Requirements of Cats*. Washington D.C.: National Academy Press.
- Park, T., Jerkins, A. A., Steele, R. D., Rogers, Q. R. & Morris, J. G. (1991). Effect of dietary protein and taurine on enzyme activities involved in cysteine metabolism in cat tissues. *Journal of Nutrition* **121**, S181–S182.

- Pion, P. D., Kittleson, M. D. & Rogers, Q. R. (1989). Cardiomyopathy in cats and its relation to taurine deficiency. In *Current Veterinary Therapy*, vol. 10, pp. 251–262 [R. W. Kirk, editor]. Philadelphia: W. B. Saunders.
- Pion, P. D., Kittleson, M. D., Rogers, Q. R., & Morris, J. G. (1987). Myocardial failure in cats associated with low plasma taurine: a reversible cardiomyopathy. *Science* **237**, 764–768.
- Rivers, J. P. W., Hassam, A. G. & Alderson, C. (1976a). The absence of ω -desaturase activity in the cat. *Proceedings of the Nutrition Society* **35**, 67A.
- Rivers, J. P. W., Hassam, A. G. Crawford, M. A. & Brambell, M. R. (1976b). The inability of the lion, *Panthera Leo*, to desaturate linoleic acid. *FEBS Letters* **67**, 269–270.
- Rivers, J. P. W., Sinclair, A. J. & Crawford, M. A. (1975). Inability of the cat to desaturate essential fatty acid. *Nature* **258**, 171–173.
- Rivers, J. P. W., Sinclair, A. J., Moore, D. P. & Crawford, M. A. (1976c). The abnormal metabolism of essential fatty acids in the cat. *Proceedings of the Nutrition Society* **35**, 66A.
- Rogers, Q. R. (1963). A study of felinine and its excretion by the cat. PhD Thesis. State University, NY, Buffalo.
- Rogers, Q. R., Morris, J. G. & Freedland, R. F. (1977). Lack of hepatic enzyme adaptation to low and high levels of dietary protein in the adult cat. *Enzyme* **22**, 348–356.
- Rogers, Q. R. & Phang, J. M. (1985). Deficiency of pyrroline-5-carboxylate synthetase in the intestinal mucosa of the cat. *Journal of Nutrition* **115**, 146–150.
- Röhrs, M. (1987). Domestication of wolves and wild cats: parallels and differences. In *Nutrition, Malnutrition and Dietetics in the Dog and Cat*, pp. 1–5 [H. Meyer, E. Kienzle and A. T. B. Edney, editors]. London: British Veterinary Association.
- Rowell, E. V., Carnie, J. A., Wahbi, S. D., Al-Tai, A. H. & Rowell, K. V. (1979). L-serine dehydratase and L-serine-pyruvate aminotransferase activities in different animal species. *Comparative Biochemistry & Physiology* **637**, 543–555.
- Schaeffer, M. C., Rogers, Q. R. & Morris, J. G. (1989). Protein in the nutrition of dogs and cats. In *Nutrition of the Dog and Cat. Waltham Symposium 7*, pp. 159–205 [I. H. Burger and J. P. W. Rivers, editors]. Cambridge: Cambridge University Press.
- Sinclair, A. J., McLean, J. G. & Monger, E. A. (1979). Metabolism of linoleic acid in the cat. *Lipids* **14**, 932–936.
- Sturman, J. A., Gargano, A. D., Messing, J. M. & Imaki, H. (1986). Feline maternal taurine deficiency: effect on mother and offspring. *Journal of Nutrition* **116**, 655–667.
- Sturman, J. A., Rassin, D. K., Hayes, K. C. & Gaull, G. E. (1978). Taurine deficiency in the kitten: Exchange and turnover of [35 S] taurine in brain, retina and other tissues. *Journal of Nutrition* **108**, 1462–1476.
- Turner, R. G. (1934). Effect of prolonged feeding of raw carrots on vitamin A content of liver and kidneys in the dog. *Proceedings of the Society for Experimental Biology and Medicine* **31**, 866–868.