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## LEADING ARTICLE

# Thiamine deficiency due to sulphur dioxide preservative in 'pet meat' – a case of *déjà vu*

R MALIK<sup>a</sup> and D SIBRAA<sup>b</sup>

This month's Clinical Section features an excellent paper<sup>1</sup> concerning thiamine deficiency in canine patients treated by Rita Singh and colleagues at the North Ryde Veterinary Specialist Centre, Sydney. The series of cases is just what would be expected from a state-of-the-art referral hospital in the first decade of the new millennium: a team approach to investigation and treatment, a timely diagnosis in most patients, utilisation of advanced diagnostics, and outcomes that were generally favourable, even in dogs that presented late with advanced signs.

A range of cases is described, serving to emphasise the spectrum of physical findings that may develop in dogs with this disease. Interestingly, one gets the sense that the authors' diagnostic acumen was refined by their experience with the index case, as a subtle pattern of suggestive clinical findings emerged, facilitating the 'pattern recognition' of subsequent cases. Magnetic resonance imaging (MRI) findings in dogs with thiamine deficiency have been reported on only one occasion previously and the additional data provided from the case so examined suggests that the changes are characteristic, as might be expected from the neuroanatomical distribution of lesions (periventricular brainstem grey matter) in carnivores with this vitamin deficiency (Figure 1). Use of the thiamine pyrophosphate effect test in this case provided unequivocal evidence that thiamine deficiency was the cause of the observed MRI lesions.

In cats, a diagnosis of thiamine deficiency is facilitated by the presence of more characteristic clinical signs, especially bilateral pupillary dilatation and the so-called 'praying sign', in which there is an active spastic ventroflexion of the head and neck. This sign can be precipitated by performing a manoeuvre that accentuates the vestibular dysfunction, namely suspending the cat by its hindquarters and 'nose-diving' the patient towards the floor or tabletop. This is not to be confused with passive ventroflexion of the head and neck, which is seen in association with diseases that cause muscle weakness, such as hypokalaemia and myasthenia gravis.

In dogs, signs of thiamine deficiency tend to be more cryptic. It is important, therefore, to have a high index of suspicion for this diagnosis in canine patients with neural deficits, and sometimes even in dogs presenting with non-specific findings such as depression, vomiting and anorexia. Neurological findings in dogs with thiamine deficiency may include ataxia, vestibular signs, paresis, altered mental state, seizures and combinations of the above, consistent with a multifocal disease process.

The importance of taking a careful dietary history cannot be overemphasised, as typically this provides the vital clue that thiamine deficiency is the correct diagnosis. In Australia, the key historical finding is feeding 'pet mince', 'pet meat' or 'unrefrigerated food rolls' especially when there is a relationship in time between the development of signs and a change in the composition of the diet and/or its supplier. In the distant past, and in other countries, feeding of fresh or canned fish containing thiaminases or inactivation of thiamine by cooking or processing have been implicated in the aetiology, however nowadays commercial canned and dry food produced for cats and dogs is generally supplemented with thiamine. There is a suggestion from Singh's paper, and the historical literature cited, that signs might develop more quickly in young patients, perhaps reflecting increased requirements for thiamine during growth.

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Unfortunately, we were left with extreme frustration after reading this paper! Why, one might ask, when it is such a 'good paper' - well written, presented to an appropriate audience and with acceptable treatment outcomes? The reason is simple: each of these cases was preventable, including the pups that died and the index case that afforded the opportunity to characterise the MRI findings, albeit at considerable expense for the owners, and not without risk to the dog because of the dangers inherent in anaesthetising a patient with likely increased intracranial pressure.

Moreover, thiamine deficiency was meticulously characterised in Australia many years ago, with the cause in most patients being determined beyond doubt. Perhaps the best Australian studies were conducted by Professor Virginia Studdert and Dr Robert LaBuc in Melbourne,<sup>2</sup> and Dr Robert Steele in Sydney,<sup>3</sup> who demonstrated clearly that thiamine deficiency occurs when cats or dogs are fed exclusively a diet containing high concentrations of sulphur dioxide. Steele's case was especially poignant, as the cat developed fatal iatrogenic thiamine deficiency while being prescribed an elimination diet of kangaroo meat to exclude the possibility of a food-associated dermatosis. The label on the meat fed to the cat indicated it was vacuum-packed, but did not disclose that it contained preservatives. Steele demonstrated further that 54 of 63 meats sold for pet consumption in 1996 contained sulphite preservatives.

Preservatives that liberate sulphur dioxide (220 - sulphur dioxide, 221 - sodium sulphite, 222 - sodium bisulphite, 223 - sodium metabisulphite, 224 - potassium metabisulphite, 225 - potassium sulphite, 228 - potassium bisulphite) are commonly added, in varying degrees, to 'pet meat/mince' to diminish the odour produced by bacteria that multiply in food, and delay the reduction of myoglobin, which results in the meat appearing brown rather than red. Sulphur dioxide rapidly inactivates thiamine present normally in meat and meat by-products, and indeed,

there may be sufficient preservative to inactivate thiamine present in other dietary components fed concurrently, for example, brewers yeast. It is possible to determine the presence of sulphur dioxide in food inexpensively by adding 10 drops of a test solution (0.02% malachite green and 0.02% sodium benzoate) to a test diet; absence of colour after 2 minutes indicates the presence of sulphur dioxide. This test is very sensitive for even small amounts of preservative. In addition to their effects on thiamine, sulphites have been associated with the full range of food intolerance symptoms in people, including headaches, irritable bowel symptoms, behavioural disturbances and skin rashes. They are also well known for their ability to exacerbate asthma in human patients,<sup>4,5</sup> which might be a pertinent consideration when managing cats with 'asthma', or dogs with chronic bronchitis or atopic dermatitis. In relation to this point, it should be noted that sulphites are permitted in very large concentrations (up to 3000 mg/kg) in some foods destined for human (and therefore possibly animal) consumption, for example dried fruits and vegetables.

A trip to a local supermarket or any large pet store or warehouse will support the contention that there are large numbers of suppliers of 'pet meat' and 'food rolls', and that these products seem popular with the public. It would be interesting to know what proportion of the pet food market is catered to by this type of food, and whether such foods are fed exclusively, or as a part of a heterogeneous diet. Previous studies<sup>2,3</sup> have shown that this type of diet may have sufficient sulphur dioxide content to destroy endogenous thiamine present in the ration. A level of 400 mg sulphur dioxide/kg depletes thiamine by 55%, while 1000mg/kg depletes it by 95%.<sup>2,3</sup> Thiamine given as a supplement concurrently is likewise inactivated.<sup>2,3</sup> Considering that these foods may be fed exclusively to cats and dogs it is indeed surprising that thiamine deficiency does not occur more commonly, and the reason for this is worthy of further investigation.

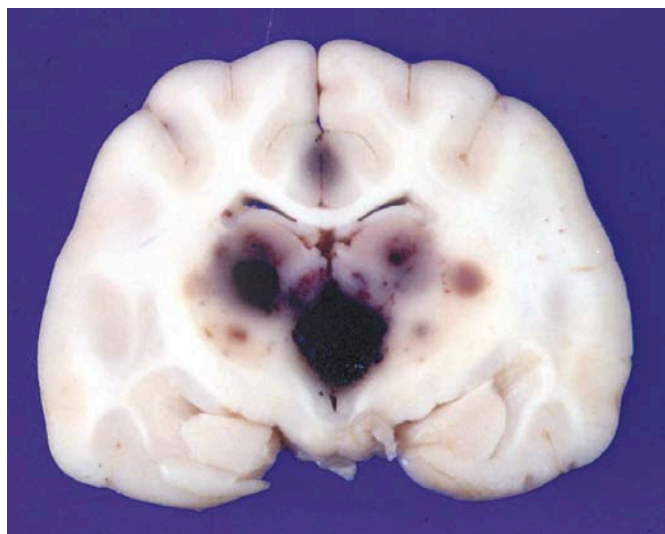
**Table 1. Sulphur dioxide content (in mg/kg) in different food items purchased on 12th April 2005 at a suburban supermarket and a pet food wholesaler. The label claims in relation to preservatives are tabulated also.**

Manufacturer	Intended target species	Label description of product	Preservative claim on label	Sulphur dioxide content (mg/kg) <sup>a</sup>
Brand A	Dog (puppies)	Roll; 900 g	-	33
Brand A	Dog	Roll (cooked); 2 kg	No preservatives	18
Brand B	Dog	Roll (beef and lamb with liver, heart, cereal, vegetables); 1.5 kg	-	29
Brand B	Cat	Roll (chicken, lamb, cereal); 365 g	-	119
Brand B	Dog and Cat	Bulk pet mince with kangaroo, beef, lamb, trimming; 2.5 kg	-	640 <sup>b</sup>
Brand B	Cat	Kangaroo, lambs fry, heart; 1 kg	Preservative 220 (minimal) added to retain freshness	738 <sup>b</sup>
Brand C	Cat	Pilchard (chunky, canned); product of Thailand	-	< 10
Brand D	Cat	Kangaroo and brawn; 1kg	-	453 <sup>b</sup>
Brand D	Dog and Cat	Mincing kangaroo; 1 kg	-	356 <sup>b</sup>
Brand D	Dog and Cat	Diced lamb	-	1,058 <sup>b</sup>
Brand E	Cat	Seafood platter (four varieties of canned seafood); Product of Thailand	-	45
Brand E	Cat	Mince (lean kangaroo); 1 kg	Preservative (220) (minimal)	714 <sup>b</sup>
Brand F	Dog	Roll (cooked beef, chicken, vegetables, pasta); 2 kg	Fully cooked	14

<sup>a</sup>mg/kg is equivalent to parts per million (ppm)

<sup>b</sup>These diets have the propensity to cause thiamine deficiency if fed exclusively.

- = Information not apparent on package label



**Figure 1. Photograph of a transversely sectioned, formalin-fixed, brain from a cat that died of thiamine deficiency. Note the multiple, bilaterally symmetrical, focal areas of grey-red discoloration throughout the brainstem. Histologically, such discoloured areas correspond to regions of haemorrhagic malacia. The extent and severity of such lesions would account for the poor prognosis of advanced cases of thiamine deficiency. Kodachrome kindly supplied by Professor BRH Farrow.**

To provide a current estimate of the prevalence of sulphur dioxide in pet meat, pet mince and food rolls, one of the authors (RM) obtained a representative selection of these products from one suburban supermarket and one regional pet food warehouse on the 12<sup>th</sup> April 2005 and submitted them to a commercial laboratory<sup>a</sup> for testing. Specimens were tested by Mr Roger Mooney using AOAC Method 962.16 (modified Monier Williams method).<sup>6</sup> A small number of cans of commercial cat food produced overseas were tested also. The results of these analyses, presented in Table 1, show a high content of sulphur dioxide in approximately half of the food items tested.

Due to the high cost of testing, only a limited number specimens were examined. Even so, certain trends emerged. With one minor exception, refrigerated food rolls (that had been cooked during processing) had low concentrations of sulphur dioxide unlikely to lead to thiamine deficiency. In contrast, all products containing diced or minced meat had dangerous concentrations of sulphur dioxide. Two products that stated preservative 220 was present in 'minimal' quantities had concentrations of sulphur dioxide in excess of 700 mg/kg. Four products that did not advertise the presence of preservatives had dangerous concentrations of sulphur dioxide (ranging from 356 to 1,058 mg/kg). Canned fish foods produced in Thailand had low concentrations of sulphur dioxide, although we did not assay their thiamine content. We were unable to find any unrefrigerated food rolls for testing, which was interesting, as these were said historically to contain especially high concentrations of sulphites.

The principal underlying problem is the lack of regulation over the range of meat products and by-products sold for consumption by pets in New South Wales. No requirement currently exists under the NSW Stock Foods Act or the Stock Medicines Act to identify the concentration of sulphite preservatives in meat

destined for pet consumption - this legislation is mostly concerned with food producing species and horses. From our research, there appear to be no standard or legislative requirements specifically in relation to the contents of pet food, apart from the Trade Practices Act which protects against manufacturers making misleading or untrue claims. The industry is currently self regulated via the Pet Food Industry Association of Australia. The web page (<http://www.nswfitc.com.au/a/2680.html>) of this organisation states they set industry standards via a code of practice which apparently incorporates standards for labelling, marketing, nutrition design and claims. Details of this code of practice are not available from the website. Pet food labelling in Australia is governed by both state industry laws and consumer product laws, and in some states there is a legal requirement that the words 'PET FOOD ONLY' appear on the label, and some state legislation makes it mandatory for a picture of the animal species for which the food is intended to feature on the label. A minimum guaranteed analysis declaration is also required stating minimum percentage of crude protein, fat, fibre, moisture, salt and, optionally, other ingredients. There is, however, no legislative control over what is incorporated into pet food, and in particular, whether preservatives are added.

In contrast, the NSW Food Authority carefully regulates butcher shops, delicatessens, supermarkets and other food outlets to ensure that preservatives are not added to meat products for human consumption (except sausages, sausage meat and cooked manufactured meats). Fines of up to \$55,000 for individuals and \$275,000 for corporations apply. In view of the severe adverse consequences that preservatives can have on companion animals, veterinarians have an obligation to lobby that appropriate legislation likewise be drafted to cover food intended for consumption by pets.

The success of the ban on tail docking has demonstrated what this profession is capable of achieving when it presents a coherent unified front. A similar effort is required in relation to the issue of preservatives in pet food. Clearly, we have a responsibility to speak out on such issues in our role as advocates for companion animals, and to provide a scientific basis that underpins our position. For legal and political reasons, this could best be tackled at the state level by appropriate arms of the veterinary profession. In situations where pressure can be brought to bear, it is likely to be most appropriate that those organisations representing a wide body of veterinarians, such as the Australian Veterinary Association and Australian College of Veterinary Scientists, be involved, so that the full force of the profession can be harnessed. It could be effectively argued that sulphite preservatives are actually 'drugs', and that their inclusion in pet mince could be pursued by the Australian Pesticides and Veterinary Medicines Authority. Certainly, the implications of thiamine deficiency are sufficiently severe that they should be considered a life-threatening adverse drug reaction, and in the interim, this may be a fruitful line to pursue.

What advice can we give our clients in the meantime concerning the feeding of raw meat and meat by-products for consumption by cats and dogs? We should advise consumers not to be seduced by the claims on the packaging. Most owners of companion animals wish to do the best they can for their pets. They are told that the contents of 'pet mince' and 'food rolls' are nutritious and provide the best quality meat for the animals. They are often cheaper than fresh meat fit for human consumption, more convenient to buy and sometimes do not require refrigeration. Owners should be informed that because there is no legal requirement to

<sup>a</sup>Silliker Microtech Pty Ltd, Unit 2C, Regents Park Estate, 391 Park Road, Regents Park, New South Wales 2143

state the presence of preservatives in these foods, their presence is not invariably advertised on the label. We should explain that the use of preservatives does not prevent spoilage or putrefaction, but that rather it only masks the most revealing signs, namely the unsavoury odour and brown discolouration of the meat. Finally, we should draw pet owners' attention to the fact that 'pet meat' has not necessarily been subjected to the stringent meat inspection and processing assessment that occurs for meat designated for human consumption.

Perhaps the simplest way to ensure that thiamine deficiency does not occur is to recommend feeding a commercially prepared, nutritionally complete dog food, either canned and/or kibble in type. For those clients and veterinarians convinced of the health benefits of feeding a more natural diet, either exclusively or as a component of a varied diet, it should be recommended that such food be obtained from the local butcher, where appropriate legislation affords protection against the use of preservatives that could give rise to thiamine deficiency.

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## BOOK REVIEW

**Domestic Animal Behavior for Veterinarians and Animal Scientists.** 4th edn. Houpt K, Blackwell Publishing, Iowa, 2005, 506 pages. Price \$A137. ISBN 0 8138 0334 9.

One of the guiding principles of my veterinary education was that it was necessary to understand what was normal before attempting to come to grips with the abnormal. However, in books on behavioural problems in animals these fundamentals are often bypassed or minimised in an eagerness to get to the 'clinically relevant' part of the book – descriptions of behavioural problems and their associated therapies. Thankfully, this is not the case with Dr Houpt's book and this remains one of its greatest strengths. Ever since the first edition in 1982, it has emphasised underlying behavioural mechanisms and documenting normal behaviours, thus allowing behavioural problems to be placed in an appropriate context when discussed. To the busy clinician who simply wants to know how to deal with a particular problem, such fundamentals may seem irrelevant, however, by understanding the mechanisms that drive behavioural expression, this provides the therapist with first principles to fall back on when the common 'cookbook' approach to treating behavioural problems fails.

As with previous editions, the book is divided into nine chapters – communication, aggression and social structure, biological rhythms and sleep, sexual behaviour, maternal behaviour, development of behaviour, learning, ingestive behaviour, and miscellaneous behavioural disorders. Each chapter is organised into general principles and a comprehensive summary of what is known for each of seven major domesticated species - horses, dogs, cats, pigs, cattle, sheep and goats (although the information on goats is rather limited). While chapter nine discusses miscellaneous problems, the majority of behavioural disorders are discussed within the appropriate chapters under the heading of the species concerned. This organisation makes the book a pleasure to read, the information easy to find, and the relevance of each behavioural system to specific behavioural disorders obvious and informative.

For those readers familiar with previous editions, there are few surprises, despite the text having been comprehensively updated with contemporary references and new information (over 1800 supporting references of which ~20% are new since the 1998 edition). The reason for this is that much of the core work in applied animal behaviour that is central to the discussions in this book were undertaken 10 to 20 years ago. With only one exception, all the figures can be found in previous editions of the book, thus, while the new additions improve the quality of the information, the need for someone to update from their 2nd or 3rd edition will be more a matter of personal taste than necessity.

So who should buy this book? Anyone working with large animals will find it an excellent resource for its comprehensive summaries of large animal behaviours and associated behavioural problems, information that is often not readily available in other texts. The companion animal practitioner who wants a broader understanding of the behavioural processes they are attempting to modify will also gain much from this edition. I'd also recommend it to people who enjoy being able to answer the obscure questions posed by clients, for example how many times a day should a horse urinate; how long does a sheep sleep and what proportion is REM (but exactly what sheep count when they go to sleep is not discussed); can animals see in colour; the clinical relevance of the curl in a pig's tail or the names and meanings of the 14 different vocalisations a cat makes.

Despite my high praise for the book, I do have a minor criticism. For the next edition I'd recommend that the publisher actually bother reading it (at least to the bottom of the first page). The blurb on the back cover promises: "New to this edition are discussions of progress and research in the relationship between behavior and animal welfare". Unfortunately, the author states very clearly in her preface to the new edition: "Welfare is an important topic that has been covered in several books and many publications, so it is not directly covered here." And indeed it is not. While I can understand the necessity of deferring to more specialised texts, as many of the issues are outside the scope of this book, a small reference to welfare might not have gone astray, in particular, during discussions of vices and stereotypic behaviours that are thought by many to result from poor environmental conditions and are signs of reduced animal welfare. The reason for mentioning this is that many traditional therapies for these problems are based on physical restraint or redirection. It would be wise to warn in these cases that simply addressing the sign, rather than the cause, may 'fix' the behavioural disorder, but not improve the welfare of the animal in conditions where it is compromised.

## MR Low

Matt Low is a member of the animal behaviour chapter of the Australian College of Veterinary Scientists and has recently completed his PhD in behavioural ecology at Massey University.