OPEN ACCESS

SHORT COMMUNICATION

Hair arsenic level in rice-based diet-fed Staffordshire bull terriers

Sarah Rosendahl, Johanna Anturaniemi, Anna Hielm-Björkman

Abstract

Background There have been concerns related to inorganic arsenic (iAs) in rice and the risk of chronic toxicity in human beings, especially children. Rice is a common constituent of pet food, and dogs often eat the same food on a continual daily basis for long periods of time. Therefore, the purpose of this study was to assess the risk of chronic iAs exposure in rice-based diet-fed dogs.

Methods Hair iAs level was measured in seven rice-based diet-fed dogs (mean age 3.8 years) and in nine dogs that did not consume any rice (mean age 4.4 years), using inductively coupled plasma mass spectrometry.

Results The hair iAs level was significantly higher (P=0.005) in dogs fed a rice-based diet (mean 0.143 µg/g) than in dogs that did not consume any rice (mean 0.086 µg/g), while age and sex did not show associations with hair iAs level.

Conclusion The results suggest that eating a rice-based diet for long periods of time represents a risk for chronic iAs exposure in dogs.

Arsenic (As) is a metalloid that is ubiquitous in the environment as highly toxic inorganic arsenic (iAs) or as non-toxic organic arsenic (oAs).[1] In human beings, chronic exposure to iAs has been associated with tumours of the skin, bladder and lungs,[2] and with alterations in gastrointestinal, neurological, cardiovascular, immunological, haematological, pulmonary and developmental function.[3] In dogs, research has mainly focused on nephrotoxicity.[4] iAs is mostly found in the water of certain geographical areas and in rice and rice products,[5–7] while oAs is found in fish and seafood.[8] Lately, there have been some recommendations to limit rice intake in human beings, especially children, due to iAs content.[9 10] Rice is often a major constituent of commercial pet foods,[11] and considering that pets often eat the same food on a continual daily basis over long periods of time, this could expose them to long-term iAs intake. In pet foods, higher total As levels have been found in fish-based diets compared with poultry or red meat-based diets.[12 13] Even though the legal safety limit of total As content might not be surpassed in most pet foods, iAs is considered a non-threshold carcinogen and any exposure constitutes a health risk.[14] When measuring low-level dietary exposure to iAs, hair and urine are considered the most useful specimens in dogs.[15] iAs is the predominant form of As in hair.[16] Based on animal studies, oAs does not accumulate in hair,[17] and therefore consumption of fish and seafood should not be a confounding factor when measuring hair As. The aim of this study was to compare hair iAs levels in dogs fed a rice-based diet with dogs that did not consume any rice, to assess the risk of chronic iAs exposure in rice-based diet-fed dogs. The hypothesis was that long-term consumption of a rice-based diet would lead to elevated hair iAs levels in dogs.

The dogs in this study were part of a larger diet intervention study on atopic dermatitis in Staffordshire bull terrier dogs. Only baseline values were used. A total of 45 dogs had baseline values for hair iAs level. Inclusion criteria for the study group were a dog eating a diet of 80 per cent or more of a rice-based (rice as first or second ingredient) dry dog food and for the control group a dog that did not consume any rice. Exclusion criteria for both groups were dogs that had been eating their current diet for less than one year, dogs that were eating mixed diet and dogs that had lacking data. After considering the inclusion and exclusion criteria, 17 dogs were eligible for this study. Since only one was non-atopic, it was furthermore excluded to make the study group more homogeneous. The study group consisted of seven dogs (two males, five females; mean

Veterinary Record (2019) doi:10.1136/ vetrec-2019-105493

Department of Equine and Small Animal Medicine, University of Helsinki, Faculty of Veterinary Medicine, Helsinki, Finland

E-mail for correspondence: Sarah Rosendahl; sarah.rosendahl@helsinki.fi

Provenance and peer review Not commissioned; externally peer reviewed.

Received April 3, 2019
Revised September 18, 2019
Accepted October 6, 2019

Table 1 Basic information and hair iAs level of all study dogs (N=16)

Dog and sex	Age (years)	Diet type	Diet group	Hair iAs level (µg/g)
1F	8.5	100% raw	Control	0.06
2M	2.3	100% raw	Control	0.06
3M	1.4	100% raw	Control	0.07
4M	7.7	97% raw	Control	0.07
5F	1.8	90% raw	Control	0.08
6F	2.9	100% raw	Control	0.09
7F	9.7	80% dry	Study	0.10
8M	3.4	100% raw	Control	0.10
9M	6.1	95% dry	Study	0.11
10F	6.0	100% raw	Control	0.12
11F	5.4	90% wet	Control	0.12
12F	2.0	100% dry	Study	0.12
13F	5.5	80% dry	Study	0.14
14F	1.3	80% dry	Study	0.14
15F	1.1	90% dry	Study	0.18
16M	1.1	96% dry	Study	0.21

F, female; iAs, inorganic arsenic; M, male.

age 3.8 years) and the control group of nine dogs (four males, five females; mean age 4.4 years) (table 1). Information about the dogs' diets was collected via a questionnaire which the owners completed before entering into the study. Hair samples were collected from all dogs. The owners were asked not to wash the dogs two weeks before sampling. About 125 mg of hair was cut from the underside of the tail and placed into paper envelopes labelled with the dog's name, age and sex. Samples were analysed at Trace Elements (Texas, USA) for hair iAs using inductively coupled plasma mass spectrometry (Sciex Elan models 6100 and 9000) and microwave digestion method (CEM Mars 5 Plus). Results were given as mg% (milligrams per 100 grams) and converted into µg/g to allow for comparison with previous studies. Data were analysed using SPSS V.25 for Windows (IBM, Armonk, New York, USA). A linear regression model was performed using hair iAs level as dependent variable and diet (rice/no rice), age and sex as independent variables. A critical P value of 0.05 was chosen to denote statistical significance.

According to the linear regression model, the hair iAs level in the study group (mean 0.143 µg/g; minimum 0.10; maximum 0.21; sd 0.039) was significantly higher (P=0.005) than in the control group (mean 0.086 µg/g; minimum 0.06; maximum 0.12; sd 0.024) (figure 1). Age

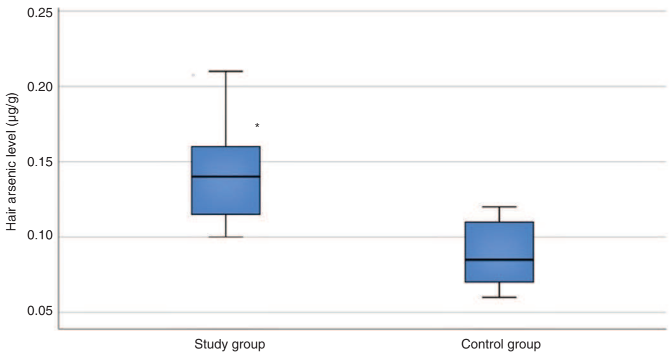

Figure 1 Mean hair inorganic arsenic level in the study group (n=7) and in the control group (n=9), analysed using linear regression model. *P=0.005.

and sex did not show significant association with the hair iAs levels (P=0.128 and P=0.710, respectively). The interanimal variabilities in the study and control groups were 27.6 per cent and 27.5 per cent, respectively.

Albeit statistically significant, the difference in mean iAs level between groups was small and the minimum and maximum values overlapped; thus, the toxicological difference might be small. Anyway, the mean iAs level in the study group was higher than that considered by Neiger and Osweiler[15] as typical background level in unexposed dogs (0.12 µg/g) as well as that in dogs from different areas in Slovakia (0.08–0.11 µg/g),[18] although the differences were small. There is a lack of data on hair iAs levels in dogs, but in human beings levels of 0.1–0.5 µg/g have been indicated for chronic exposure.[19] Even though iAs levels measured in the current study do not indicate toxicity, the role of chronic iAs exposure in dogs needs to be further assessed, taking into consideration adverse health effects observed in human beings such as cancer,[20] diabetes,[21] neurological effects[22] and increased oxidative stress.[23]

In conclusion, dogs eating a diet of rice-based dry dog food had higher hair iAs levels than dogs that did not eat any rice. This suggests that eating a rice-based diet for long periods of time represents a risk for chronic iAs exposure in dogs. These results emphasise the need to establish a less monotonous diet for dogs to minimise the risk of accumulating a certain contaminant. Based on these results, hair mineral analysis can be considered an informative, cheap and non-invasive method in determining iAs exposure in dogs. Future studies should include parallel urine analyses and larger sample size.

Funding This study was funded by Victoriastiftelsen.

Competing interests None declared.

Ethics approval The study protocol was approved by the Animal Experiment Board in Finland (ELLA) (permit number: ESAVI/3244/04.10.07/2013).

Data availability statement All data relevant to the study are included in the article.

ORCID iDs
Sarah Rosendahl http://orcid.org/0000-0002-1990-3563
Johanna Anturaniemi http://orcid.org/0000-0002-2766-6254

References

1 Hughes MF, Beck BD, Chen Y, *et al*. Arsenic exposure and toxicology: a historical perspective. *Toxicol Sci* 2011;123:305–32.
2 Hong Y-S, Song K-H, Chung J-Y. Health effects of chronic arsenic exposure. *J Prev Med Public Health* 2014;47:245–52.
3 World Health Organization. Safety evaluation of certain contaminants in food. who food additives series No. 63, 2011.
4 Tsukamoto H, Parker HR, Gribble DH, *et al*. Nephrotoxicity of sodium arsenate in dogs. *Am J Vet Res* 1983;44:2324–30.
5 Sun G-X, Williams PN, Carey A-M, *et al*. Inorganic arsenic in rice bran and its products are an order of magnitude higher than in bulk grain. *Environ Sci Technol* 2008;42:7542–6.

6 Rahman MA, Hasegawa H. High levels of inorganic arsenic in rice in areas where arsenic-contaminated water is used for irrigation and cooking. *Sci Total Environ* 2011;409:4645–55.
7 Davis MA, Signes-Pastor AJ, Argos M, *et al*. Assessment of human dietary exposure to arsenic through rice. *Sci Total Environ* 2017;586:1237–44.
8 Taylor V, Goodale B, Raab A, *et al*. Human exposure to organic arsenic species from seafood. *Sci Total Environ* 2017;580:266–82.
9 U.S Food And Drug Administration. Questions and answers: arsenic in rice and rice products, 2018. Available: https://www.fda.gov/food/foodborneillnesscontaminants/metals/ucm319948.htm [Accessed 25 Feb 2019].
10 Ankarberg Halldin E, Foghelberg P, Gustafsson K, *et al*. Inorganic arsenic in rice and rice products on the Swedish market 2015. Sweden: National Food Agency, 2015: 1–28.
11 Cowell CS, Stout NP, Brinkmann MF. Making of commercial pet foods. In: Hand MS, Thatcher CD, Remillard RL, eds. Small animal clinical nutrition. 4th edn. Marceline, USA: Walsworth Publishing Company, 2000: 127–46.
12 Squadrone S, Brizio P, Simone G, *et al*. Presence of arsenic in PET food: a real hazard? *Vet Ital* 2017;53:303–7.
13 Kim H-T, Loftus JP, Mann S, *et al*. Evaluation of arsenic, cadmium, lead and mercury contamination in over-the-counter available dry dog foods with different animal ingredients (red meat, poultry, and fish). *Front Vet Sci* 2018;5.
14 EFSA Panel on Contaminants in the Food Chain. Scientific opinion on arsenic in food. *EFSA Journal* 2009;7.
15 Neiger RD, Osweiler GD. Arsenic concentrations in tissues and body fluids of dogs on chronic low-level dietary sodium arsenite. *J Vet Diagn Invest* 1992;4:334–7.
16 Yamauchi H, Takahashi K, Mashiko M, *et al*. Biological monitoring of arsenic exposure of gallium arsenide- and inorganic arsenic-exposed workers by determination of inorganic arsenic and its metabolites in urine and hair. *Am Ind Hyg Assoc J* 1989;50:606–12.
17 Vahter M, Marafante E, Dencker L. Metabolism of arsenobetaine in mice, rats and rabbits. *Sci Total Environ* 1983;30:197–211.
18 Kozak M, Kralova E, Sviatko P, *et al*. Study of the content of heavy metals related to environmental load in urban areas in Slovakia. *Bratisl Lek Listy* 2002;103:231–7.
19 Ratnaike RN. Acute and chronic arsenic toxicity. *Postgrad Med J* 2003;79:391–6.
20 Chung C-J, Huang Y-L, Huang Y-K, *et al*. Urinary arsenic profiles and the risks of cancer mortality: a population-based 20-year follow-up study in arseniasis-endemic areas in Taiwan. *Environ Res* 2013;122:25–30.
21 Díaz-Villaseñor A, Burns AL, Hiriart M, *et al*. Arsenic-Induced alteration in the expression of genes related to type 2 diabetes mellitus. *Toxicol Appl Pharmacol* 2007;225:123–33.
22 Vahidnia A, van der Voet GB, de Wolff FA. Arsenic neurotoxicity--a review. *Hum Exp Toxicol* 2007;26:823–32.
23 Jomova K, Jenisova Z, Feszterova M, *et al*. Arsenic: toxicity, oxidative stress and human disease. *J Appl Toxicol* 2011;31:n/a–107.