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Anton C. Beynen

BPA in canned petfood

BPA (bisphenol A) is a component of resins that line the inside of most petfood cans. Quite some dog and cat owners fear that BPA is a danger to their pets. BPA can leach out of the can's coating into the food, while research has identified the synthetic chemical as a disruptor of the body's hormone system, with links to various diseases. In response to the owners' concern, some petfood manufacturers claim that their canned or pouched foods are BPA free.

Ingested BPA that enters the dog's bloodstream is then converted by the liver, resulting in detoxification and excretion with urine. The cat neutralizes BPA much slower than the dog does. A toxicity study in dogs found that feeding a dry diet containing as much as 3000 mg BPA per kg had no negative health effects within 90 days. The BPA content of canned food is not higher than 0.2 mg per kg, which equals about 1 mg per kg food when completely dried. Canned food does not cause BPA intoxication in dogs and cats, at least not within a relatively short period of time.

Experimental research in laboratory rodents has shown that low intakes of BPA can imitate and oppose some of the body's hormones and thereby modulate hormone-regulated processes. It is conceivable that low dietary BPA levels enhance the development of hormone-related diseases of slow progression, revealing themselves at older age. Circumstantial evidence connects BPA with thyroid disease (hyperthyroidism) in cats.

Many dogs and cats receive canned food as part of their total diet. The BPA content of the whole diet does not compromise visible pet health in the medium term. Basic research suggests that low BPA intakes can have negative health effects in the long term, but as yet there is no solid evidence for such impact in dogs and cats.

Chemistry

Bisphenol A (BPA) is a high production volume chemical, synthesized by condensation of acetone (thus the "A") with two phenol equivalents. BPA is used to produce certain plastics and epoxy resins. Polymers coating the inside of food cans and pouches can dissociate so that the BPA component migrates to the can's content (1, 2). These events extend to other bisphenol compounds (3, 4).

Metabolism

In fasted dogs administered BPA by orogastric gavage, about 50% was absorbed, reached the liver through the portal vein and underwent glucuronidation (5). In systemic blood, the ratio of glucuronidated:free BPA was 400:1 (5). Hepatic BPA glucuronidation, which may be more efficient in dogs than in cats (6, 7), probably inactivates the xenobiotic and steers it towards urinary excretion. Nevertheless, BPA feeding raised serum total BPA in dogs (8).

Exposure

Three publications report BPA measurements in canned foods. In 11 dog and 15 cat foods, the ranges were 11 to 206 and 13 to 136 ng BPA/g product (1). Two dog foods were found to contain 12 and 18 ng/g (8) and three dog and three cat foods had <0.2 to 0.3 ng/g (9). The considerable variation may be caused by analytical inaccuracy. The highest value (206 ng/g) corresponds to about 1 ppm in the dietary dry matter.

Toxicity

In a 90-day toxicity study (10), beagle dogs (4/sex/group) were fed dry foods containing 0, 1000, 3000 or 9000 ppm BPA. The four diets were well tolerated with no overt signs of toxicity. There were no BPA effects on body-weight gain, food intake, retina condition, blood and urine values and macroscopically-evaluated tissues. Dogs in the highest dose groups had increased liver weight. Thus, 1000 and 3000 ppm BPA did not induce aberrances.

BPA has been evaluated in three- and two-generation reproductive toxicity studies in rats and mice (11, 12). Each study included a dry food without or with one of five BPA doses, ranging from 0.015 to 7500 ppm for the rats and 0.018 to 3500 ppm for the mice. Highest BPA levels without effect were 75 and 30 ppm for rats and mice.

Gut bacteria

Switching dogs from a dry food to one of two canned foods led to greater serum BPA concentrations, which were associated with fecal microbiome changes (8). There was no BPA-free control treatment, while a dry to wet diet crossover affects gut bacteria anyway. Average serum BPA increased from 0.7 to 2.2 ng/ml after 14 days (8).

Feline hyperthyroidism

Hyperthyroidism is a common endocrine disorder in older cats. Five case-control studies showed that more than 50% wet food in the diet is associated with a three-fold higher risk of developing hyperthyroidism (13-17). Two points twin BPA and the disease. BPA can act as thyroid disruptor (18), possibly leading to derailments in thyroid cells. Consumption of food from cans with easy-open (pop-top) lids further increased risk of hyperthyroidism (15). Such cans have more flexible coatings that may release extra BPA (19).

Low-dose effects

BPA has a complex dose-response curve: biological effects are measurable at low and high doses, but hardly in between (20, 21). High doses cause intoxication of body cells with exceedance of reference values, clinical symptoms and death in the short term. Low doses immediately affect the endocrine system (low-dose effects), with health issues in the long term. Perhaps, BPA accelerates the development of ageing-related diseases such as feline hyperthyroidism.

Literature

1. Kang J-H, Kondo F. Determination of bisphenol A in canned pet foods. *Res Vet Sci* 2002; 73: 177-182.

2. Oz F, Seyyar E. Formation of heterocyclic aromatic amines and migration level of bisphenol -A in sous-vide cooked trout fillets at different cooking temperatures and cooking level. *J Agric Food Chem* 2016; 20: 3070-3082.
3. Hammarling L, Gustavsson H, Svensson K, Oskarsson A. Migration of bisphenol-A diglycidyl ether (BADGE) and its reaction products in canned foods. *Food Addit Contam* 2000; 17: 937-943.
4. Uematsu Y, Ogimoto M, Kabashima J, Suzuki K, Kaneko R, Funayama K, Haneishi N, Yasuno T, Ogino S. Simulation of migration from a multi-layer laminated film intended for retort foods. *Shokuhin Eiseigaku Zsshi* 2005; 46: 133-138.
5. Gayrard V, Lacroix MZ, Collet SH, Vigi   C, Bousquet-Melou A, Toutain P-L, Picard-Hagen N. High bioavailability of bisphenol A from sublingual exposure. *Environ Health Perspect* 2013; 121: 951-956.
6. Savides MC, Oehme FW, Nash SL, Leipold HW. The toxicity and biotransformation of single doses of acetaminophen in dogs and cats. *Toxicol Appl Pharmacol* 1984; 74: 26-34.
7. Court MH, Greenblatt DJ. Molecular basis for deficient acetaminophen glucuronidation in cats. *Biochem Pharmacol* 1997; 53: 1041-1047.
8. Koestel ZL, Backus RC, Tsuruta K, Spollen WG, Johnson SA, Javurek AB, Ellersieck MR, Wiedmeyer CE, Kannan K, Xue J, Bivens NJ, Givan SA, Rosenfeld CS. Bisphenol A (BPA) in the serum of pet dogs following short-term consumption of canned dog food and potential health consequences of exposure to BPA. *Sci Total Environ* 2017; 579: 1804-1814.
9. Schechter A, Malik N, Haffner D, Smith S, Harris TR, Paepke O, Birnbaum L. Bisphenol A (BPA) in U.S. food. *Eviron Sci Technol* 2010; 44: 9425-9430.
10. Haighton LA, Hlywka JJ, Doull J, Kroes R, Lynch BS, Munro IC. An evaluation of the possible carcinogenicity of bisphenol to humans. *Regul Toxicol Pharmacol* 2002; 35: 238-254.
11. Tyl RW, Myers CB, Marr MC, Thomas BF, Keimowitz AR, Brine DR, Veselica MM, Fail PA, Chang TY, Seely JC, Joiner RL, Butala JH, Dimond SS, Cagen SZ, Shiotsuka RN, Stropp GD, Waechter JM. Three-generation reproductive toxicity study of dietary bisphenol A in CD Sprague-Dawley rats. *Toxicol Sci* 2002; 68: 121-146.
12. Tyl RW, Myers CB, Marr MC, Sloan CS, Castillo NP, Veselica MM, Seely JC, Dimond SS, Van Miller JP, Shiotsuka RN, Beyer D, Hentges SG, Waechter Jr JM. Two-generation reproductive toxicity study of dietary bisphenol A in CD-1 (Swiss) mice. *Toxicol Sci* 2008; 104: 362-384.
13. Scarlett JM, Moise NS, Rayl J. Feline hyperthyroidism: a descriptive and case-control study. *Prev Vet Med* 1988; 6: 295-309.
14. Kass PH, Peterson ME, Levy J, James K, Becker DV, Cowgill LD. Evaluation of environmental, nutritional, and host factors in cats with hyperthyroidism. *J Vet Intern Med* 1999; 13: 323-329.
15. Edinboro CH, Scott-Moncrieff JC, Janovitz E, Thacker HL, Glickman LT. Epidemiologic study of relationships between consumption of commercial canned food and risk of hyperthyroidism in cats. *J Am Vet Med Assoc* 2004; 224: 879-886.

16. Wakeling J, Everard A, Brodbelt D, Elliott J, Syme H. Risk factors for feline hyperthyroidism in the UK. *J Small Anim Pract* 2009; 50: 406-414.
17. Olczak J, Jones BR, Pfeiffer DU, Squires RA, Morris RS, Markwell PJ. Multivariate analysis of risk factors for feline hyperthyroidism in New Zealand. *New Zealand Vet J* 2005; 53: 53-58.
18. Zoeller RT, Bansal R, Parris C. Bisphenol-A, an environmental contaminant that acts as a thyroid hormone receptor antagonist in vitro, increases serum thyroxine, and alters RC3/neurogranin expression in the developing rat brain. *Endocrinology* 2005; 146: 607-612.
19. Munguía-López EM, Gerardo-Lugo S, Peralta E, Bolumen S, Soto-Valdez H. Migration of bisphenol A (BPA) from can coatings into a fatty-food stimulant and tuna fish. *Food Addit Contam* 2005; 22: 892-898.
20. Richter CA, Birnbaum LS, Farabollini F, Newbold RR, Rubin BS, Talsness CE, Vandenberg JG, Walser-Kuntz DR, Vom Saal FS. *In vivo* effects of bisphenol A in laboratory rodent studies. *Reprod Toxicol* 2007; 24: 199-224.
21. Vandenberg LN, Colborn T, Hayes TB, Heindel JJ, Jacobs Jr DR, Lee D-H, Shioda T, Soto AM, Vom Saal FS, Welshons WV, Zoeller RT, Peterson Myers J. Hormones and endocrine-disrupting chemicals: low-dose effects and nonmonotonic dose responses. *Endocrine Rev* 2012; 33: 378-455.