

# Fluorosis in Australian wildlife

## Fact Sheet

April 2020

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### Introductory statement

Fluorine is a very reactive element, which in nature is found primarily in the bound form, fluoride. Fluoride-related disease in animals is generally due to chronic exposure through ingestion of contaminated vegetation and water. Fluoride is easily absorbed and has a high affinity for mineralised tissues such as tooth and bone. In general, mammals are more susceptible to fluorosis than birds, amphibians, reptiles and fish. The primary manifestations of excess fluoride exposure in mammals are known as dental and skeletal fluorosis. Chronic fluorosis has been recorded in many mammalian species, but has primarily been studied in livestock. Acute toxicity due to high doses of fluoride is uncommon in wildlife, and is outside the scope of this fact sheet.

### Aetiology

The main natural reservoirs of fluoride in the biosphere are surface rocks and deposits, soil and the oceans <sup>[1]</sup>. Deposits of rocks containing a high level of fluoride can cause a large increase in the fluoride content of water and may lead to endemic fluorosis <sup>[2]</sup>. Volcanic ash deposition in water and on vegetation may also lead to fluorosis in exposed animals <sup>[3]</sup>.

Primary sources of industrial fluoride emissions include production of aluminium, chemical and plastics, agricultural pesticides, glass and ceramics (including brick), manufacture of dye and metal parts and drilling and refining of oil <sup>[4]</sup>.

Elevated levels of fluoride in young animals affect the production of dental enamel, as well as accumulating in mineralised tissues throughout life. A range of characteristic lesions are seen in bone (osteofluorosis) and teeth (dental fluorosis), even though the specific manifestation and severity of lesions may vary between species depending on the age, duration and extent of exposure.

### Affected species

Among livestock and domestic animals, cattle are considered the most sensitive species <sup>[5]</sup>. Dental fluorosis and osteofluorosis have been documented in a range of species, but most frequently in humans and ungulates, including domestic livestock <sup>[6]</sup>, as well as a number of wild species in Europe and the Americas. The latter include cervids, bovids, wild boar (*Sus scrofa*) <sup>[7]</sup> and fruit bats (*Pteropus giganteus*, *P. poliocephalus* and *Rousettus aegyptiacus*) <sup>[8]</sup>.

Dental fluorosis from industrial sources has also been described in several species of small free-ranging terrestrial mammals such as voles (*Microtus agrestis* and *Clethrionomys glareolus*), wood mice (*Apodemus sylvaticus*), and moles (*Talpa europaea*)<sup>[9-11]</sup>.

In Australia, dental and skeletal fluorosis has been reported in eastern grey kangaroos (*Macropus giganteus*), red-necked wallaby (*Notamacropus rufogriseus*), swamp wallaby (*Wallabia bicolor*), koala (*Phascolarctos cinereus*), common brushtail possum (*Trichosurus vulpecular*) and common ringtail possum (*Pseudocheirus peregrinus*) in a localised region of Vic<sup>[12, 13]</sup>.

Metabolic bone disease (MBD) with associated osteofluorosis was reported in captive native frogs (*Leiopelma* sp.) in New Zealand<sup>[14]</sup>.

Seabirds and fish have a higher 'normal' bone fluoride concentration due to dietary intake, and a corresponding higher tolerance, when compared to mammals<sup>[2, 15]</sup>.

## World distribution

Fluorosis can occur in any area of the world where environmental fluoride occurs from natural (e.g. geothermal activity) or industrial sources. Levels of fluoride will also be naturally higher in animals (and plants) that inhabit coastal areas due to sea spray, but not generally manifest as disease. It has been reported from all continents except Antarctica, although very high total bone fluoride concentrations were found in an Antarctic Adelie penguin (*Pygoscelis adeliae*)<sup>[16]</sup>.

## Occurrences in Australia

Murray 1981 [15] investigated industrial fluoride contamination near Newcastle in NSW and reported elevated fluoride levels in pelicans (*Pelecanus conspicillatus*), house mice (*Mus musculus*) and insects. Insects were shown to constitute a significant part of the mice's diet; this exposure pathway is therefore of relevance to insectivorous native Australian mammals.

Fluorosis was reported in sheep in Australia in the 1950s that consumed water high in fluoride from Artesian basin bores<sup>[17]</sup>. Similarly, long-lived herbivorous wildlife living close to industrial sources of airborne fluoride emissions are at risk of developing fluorosis.

Chronic lameness in female eastern grey kangaroos (*Macropus giganteus*) living in the buffer zone of the aluminum smelter at Portland Vic, was investigated by Clarke et al. 2006 [18]. Further studies at that location have provided detailed information on fluorosis in six species of Australian marsupials<sup>[12, 13, 19-21]</sup>.

## Epidemiology

Fluoride is taken up from the systemic circulation by bone that is growing or remodelling and bone fluoride concentrations will increase as an animal ages, even in areas of low environmental fluoride<sup>[22]</sup>. However, turnover of bone tissue occurs throughout life via the normal processes of modelling and remodelling, and circulating fluoride is continually incorporated into, and released from, bone<sup>[23]</sup>. It may take many years for the skeletal lesions caused by excessive fluoride exposure to become evident.

Fluoride-related pathological lesions have been shown to vary with the concentration, timing and duration of fluoride exposure <sup>[24-26]</sup>. Reviews of the skeletal lesions associated with fluorosis in various mammals note that the distribution, form and extent of the lesions vary greatly among species and types of bones <sup>[27, 28]</sup>. For example, skeletal lesions were absent in field voles and wood mice, despite very high levels of bone fluoride <sup>[29]</sup>.

Uptake of fluoride by the skeleton in growing animals is close to 100%, and this rate slows down as the bones mature <sup>[30]</sup>. Vikøren and Stuve 1996 [31] concluded that red deer appear to be at risk of developing dental fluorosis if they accumulate between 1000 and 2000 ppm F in their first 1.5 years of life. Death et al. 2018 [13] found that bone fluoride levels in marsupials in a high fluoride environment increased rapidly until approximately one third of the expected lifespan for each species, and then plateaued. Australian marsupials have been shown to develop fluorosis at environmental exposure levels considered safe for livestock, which may be partly attributable to the relative longevity and sedentary nature of the marsupials studied.

If a juvenile animal is exposed to excessive fluoride when their teeth are developing, they may develop abnormal dental enamel even if their subsequent fluoride exposure is normal, so dental fluorosis can be used as a biomonitor of past exposure. Similarly, an animal that is exposed to high levels of circulating fluoride after eruption of the adult dentition will not demonstrate signs of dental fluorosis <sup>[32, 33]</sup>. Negligible amounts of fluoride are transferred via the placenta and milk to the juvenile, therefore the effects of elevated systemic fluoride levels will be seen only in the enamel that develops after weaning <sup>[34-36]</sup>. For this reason, in species where the front molars mineralize before weaning, molars towards the back of the mouth are likely to be more affected than those at the front. Australian marsupials have shown dental lesions considered characteristic of dental fluorosis in eutherian mammals, and the prevalence of lesions increases with environmental fluoride levels. The severity of dental lesions increases as bone fluoride levels increase.

The consistency of a particular bone affects its fluoride concentration. <sup>[20]</sup> described the distribution of fluoride throughout the skeleton of kangaroos and found a positive association between age and bone fluoride concentration and higher bone fluoride concentration in bones with a higher proportion of trabecular bone (versus cortical bone).

In the Australian marsupials examined, skeletal and degenerative joint disease (DJD or arthritis) lesions occurred to varying extents in all species and lesion distribution further varied with biomechanical differences, with the hind limbs being more affected in the hopping species. The severity of periosteal hyperostosis (i.e. bony lumps) and periarticular osteophytes (i.e. new bone growth around joints) increased with bone fluoride levels. The probability of observing a lesion varied across species, anatomical location, age, and bone fluoride concentration. For example, when controlling for age higher bone fluoride concentrations were associated with more severe DJD in kangaroos, wallabies and possums, but not koalas. The rate of bone fluoride accumulation appears to influence the type of skeletal lesions seen. Periosteal hyperostosis may develop rapidly if bone fluoride levels are sufficiently high in younger individuals, whereas DJD lesions are less common in this cohort and generally develop more slowly in response to bone fluoride levels increasing with chronic exposure and increasing age. Baseline levels of DJD were higher in the koala

irrespective of fluoride levels, and severe cartilage lesions were uncommon in the possums and swamp wallaby.

## Clinical signs

Dental fluorosis manifests as abnormal appearance or quality of dental enamel, or complete absence of enamel <sup>[37]</sup>. Increased rates of tooth wear which can lead to a compromised capacity to chew food and premature loss of teeth <sup>[38, 39]</sup>.

Tooth wear affects the extent of food particle size reduction, leading to increased intake requirements with the associated costs of time and energy <sup>[40]</sup>, ultimately reducing longevity. These dental conditions impact on food consumption, fitness and welfare, due to pain, loss of function and the systemic effects of infection <sup>[41, 42]</sup>.

Skeletal lesions include mild to marked periosteal hyperostosis (which may be localised or generalised) and DJD, which can cause pain and lameness and result in reduced general health, fitness, body condition and reproductive success. Lesions in ungulates are first seen in the metatarsus or metacarpus <sup>[43]</sup>. In macropods, lesions were primarily seen in the hindlimbs. Only in those macropods with the highest bone fluoride concentrations were lesions observed in the front limbs, spine, and ribs. In koalas, the majority of periosteal hyperostosis was seen in the mandibles. DJD was seen primarily in the elbow joints of koalas and possums <sup>[13]</sup> and periosteal hyperostosis was seen equally in both fore and hind limbs of possums and koalas <sup>[12]</sup>.

## Diagnosis

Lesions described in mineralised tissues are characteristic, especially in combination with evidence of systemic accumulation and clinical effects, thus creating a 'syndrome', even though the signs are not diagnostic of themselves <sup>[2, 12, 21, 24, 26]</sup>.

An indication of the lifetime exposure of fluoride, which relates to the likelihood of clinical disease, requires a bone biopsy.

Fluorosis is an unlikely diagnosis in wildlife unless there is a chronic natural or industrial source of fluoride directly influencing their intake of fluoride via food or water. Investigation of the levels of fluoride in the environment (water, soil, natural vegetation, any supplementary feed) is a necessary aspect of the attempt to diagnose the condition in wildlife. It is important to establish "normal" fluoride levels as comparison for a population with suspected fluoride effects, and this may vary with species and location. For example, marine derived food sources are naturally higher in fluoride than terrestrial food sources <sup>[15]</sup> and vegetation fluoride levels in coastal areas are higher due to sea spray deposition <sup>[44]</sup>. In addition, levels of fluoride in bone of affected wildlife need to be analysed and shown to be above "normal" (approximately > 1000 µg F/g). However, in the case of dental fluorosis arising from exposure only as a juvenile, it is possible the bone fluoride levels will not be appreciably high. Bone may be obtained from deceased animals, or via bone biopsy obtained under sedation or anaesthetic (e.g. from a digit or tail vertebrae).

Threshold levels of around 4000 µg F/g dry bone, above which obvious lesions become evident, have been described in various mammalian species <sup>[12, 21, 24, 26]</sup>.

## Clinical pathology

Fluoride levels may be elevated in blood, urine and faeces, but these are generally more reliable when assessed at a population level. These markers are subject to short-term fluctuation as they are closely associated with current fluoride exposure, and they also vary as fluoride is accumulated and excreted from the body.

Changes in haematological and biochemical parameters have not been seen in marsupials and no biomarkers have been reported for other species internationally.

## Pathology

The range of pathology reported in Australian marsupials is consistent with that described in other mammals. Biomechanical differences in marsupials offer some explanation for the degree of interspecific variability in prevalence, type, anatomical location, and severity of the lesions.

Eastern grey kangaroos formed multiple, large, smooth exostoses over the diaphysis of long bones (especially, tibia, fibula and metatarsi), and the ribs. There were also lesions of degenerative joint disease, including periarticular osteophytosis, articular cartilage erosion/ulceration, synovial hyperplasia and joint capsular fibrosis <sup>[20]</sup>.

Exostoses in eastern grey kangaroos are characterized by trabeculae, formed perpendicular to the bone surface, with some large Haversian systems and in some cases a cartilage cap over the lump. There is clear evidence of active remodelling. There is no increase in osteoblast numbers and no increase in periosteal vascularity or inflammatory cells. Within the cortex of the bone, there may be obvious concentric growth lines and occasionally, some of this new bone growth is more organized, while the newer bone remains woven <sup>[20]</sup>. There is frequent osteoclastic periosteal cortical resorption and relatively dense subchondral bone with occasional clustering of chondrocytes within the articular cartilage. Mild articular cartilage change is common with the appearance of white spots on the cartilage surface, usually approximately 2-3 mm in diameter, which correspond histologically to small cysts within the superficial cartilage. As the severity of gross cartilage damage increases, there is uneven staining of the articular cartilage and greater chondrocyte clustering, with cells becoming increasingly plump, progressing to clear fibrillation and eventually full-thickness loss of articular cartilage.

All species of marsupial investigated by Death et al. 2017 [12] showed varying degrees of periosteal hyperostosis, ranging from localized and mild to severe and generalised. The gross appearance and texture of the bone varied from chalky, often porous to denser, smooth, yet distorted, cortical bone. Common ringtail possums showed apparent stunting of the long bones. Bowing/deformation of the tibiae and fibulae was seen in swamp wallabies. Sclerosis of the vertebral bodies was noted in some macropodids and possums. A red-necked wallaby displayed severe, generalized hyperostosis of the tail vertebrae.

Dental lesions described in the molars (and incisors) of marsupial species are <sup>[21]</sup>:

1. marked enamel opacity and discoloration (with the fourth molar being the most severely affected molar in all species)

2. enamel surface defects such as hypoplasia and/or post-eruptive flaking and chipping
3. increased molar wear ranging from mild to marked reduction in ridge height.

Frogs with osteofluorosis associated with MBD showed hyperplasia, periosteal growth, and thickening of trabeculae <sup>[14]</sup>.

## Differential diagnoses

Any condition that causes abnormal colouring or wear of the teeth, or changes in gait or bone/joint structure should be considered as a differential diagnosis. In relation to osteofluorosis, conditions include inherited disease (hereditary multiple exostoses), neoplasia, osteomyelitis, vitamin A/D imbalance, endocrine disease and osteoarthritis. In relation to dental fluorosis, conditions include parasitism or malnutrition when juvenile.

## Laboratory diagnostic specimens

Bone sample (+/- blood, faeces and urine). Vegetation, feed and water samples.

## Laboratory procedures

Fluoride levels should be analysed in bone samples using the fluoride ion selective electrode method. It is essential to cross check a proportion of laboratory analysis via validation at another laboratory (ideally one that has been involved in international validation studies using industry standard samples), as results from fluoride analysis are prone to error.

## Treatment

There is no treatment for the effects of fluoride on teeth. Clinical fluorosis may improve over time if animals are removed from the high fluoride source, but any significant reduction in fluoride levels in the body will take several years, and lesions such as dental fluorosis and DJD cannot be reversed. Euthanasia on humane grounds is necessary if animal welfare is impacted (e.g. bone pain or severe osteoarthritis leading to lameness, or excessive tooth wear leading to oral disease and/or reduced body condition).

There is some evidence in humans that reducing/halting exposure to excess fluoride ingestion may result in a gradual reduction in bone fluoride levels and an improvement associated symptoms over several years <sup>[45, 46]</sup>.

Frogs showed a reduction in morbidity when fluoride levels of water were reduced (in addition to other measures including diet and UV light).

## Prevention and control

European Food Safety Authority (EFSA) standards have maximum tolerable levels of dietary fluoride in for domestic animals, which are necessary to prevent harm throughout the course of an animal's life <sup>[5]</sup>. The delay between ingestion of elevated levels of fluorides and manifestation of clinical signs adds to the complexity of management.

The standards proposed by Suttie dealt specifically with the prevention of fluorosis in cattle, on the assumption that the protection of this species would afford adequate protection for other domestic livestock <sup>[47]</sup>.

A broad aim should be to keep the bone fluoride levels of exposed mammals below 2000 ppm. Studies support restricting kangaroo access to vegetation <1500 m from the central emission point of the Portland aluminium smelter to achieve this aim. It is important to prevent high peaks (i.e. > 80 ppm per month) of vegetation fluoride exposure in juveniles.

To prevent multifactorial MBD in captive frogs, it is recommended to use defluoridated water, in addition to adequate dietary calcium and increase exposure to natural or artificial (UVB) light <sup>[14]</sup>.

## Surveillance and management

Wildlife populations in the vicinity of industrial fluoride-emitting facilities, or in areas where fluoride is naturally high in water, could be observed for signs of gait abnormality/lameness, and sedated animals or carcasses could be opportunistically examined for dental or skeletal lesions.

Management options include fencing off areas with higher levels of fluoride on the vegetation, or in the water, and habitat alteration to prevent wildlife seeking to inhabit the impacted area (for example, replacing open grassland with shrubs that are unpalatable to grazing macropods, to reduce grazing close to an emission source). Management strategies will have to be adapted to the specific situation.

Wildlife Health Australia administers Australia's general wildlife health surveillance system, in partnership with government and non-government agencies. Wildlife health data is collected into a national database, the electronic Wildlife Health Information System (eWHIS). Information is reported by a variety of sources including government agencies, zoo based wildlife hospitals, sentinel veterinary clinics, universities, wildlife rehabilitators, and a range of other organisations and individuals. Targeted surveillance data is also collected by WHA. See the WHA website for more information <https://wildlifehealthaustralia.com.au/Our-Work/Surveillance> and <https://wildlifehealthaustralia.com.au/Our-Work/Surveillance/eWHIS-Wildlife-Health-Information-System>. We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at [admin@wildlifehealthaustralia.com.au](mailto:admin@wildlifehealthaustralia.com.au).

## Research

The majority of Australian fauna have not been assessed for baseline levels of fluoride or fluorosis. Further description of normal levels of fluoride accumulation in different species, and examination of at-risk populations that inhabit suspected high-fluoride environments (e.g. industrial sites or areas with known high levels of fluoride in the water) would be required to improve our understanding of the range of impacts and risks across species.

## Human health implications

There are no known human health implications due to interaction with or consumption of wildlife that have fluorosis, however if humans are exposed to high levels of fluoride in their diet, or their workplace, disease can occur.

## Conclusions

Fluorosis is recognised in wildlife globally and in a range of Australian species. Fluorosis in wildlife is the result of chronic fluoride ingestion. Excess fluoride ingestion causes abnormal dental development and fluoride is accumulated in bone throughout life and if levels reach certain thresholds, then skeletal disease results. Both dental and skeletal fluorosis has been observed in multiple species of Australian marsupials living in high-fluoride environments, and these long-lived herbivorous native species have been impacted at exposure levels that would have previously been considered “safe” for livestock. Once the clinical manifestations of fluorosis are established in mammals there is no treatment other than reduction in exposure. Investigation of additional native Australian species in known or suspected high-fluoride environments should be undertaken to ensure animal health and welfare.

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*Wildlife Health Australia recognises the Traditional Custodians of Country throughout Australia. We respectfully acknowledge Aboriginal and Torres Strait Islander peoples' continuing connection to land, sea, wildlife and community. We pay our respects to them and their cultures, and to their Elders past and present.*

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