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Review

Cadmium exposure and consequence for the health and productivity of farmed ruminants

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ABSTRACT

This paper reviews Cd exposure and consequences for the health and productivity of farmed ruminants. In farmed ruminants, Cd exposure may be associated with a number of different activities, including industrial processing, mining, and agricultural practices, and is also higher in soils in some geographic regions. Cd kidney concentrations increase with age and Cd exposure. Although Cd toxicity in farmed ruminants has been demonstrated experimentally, there are no published reports of naturally occurring Cd toxicity in farmed ruminants. Clinical signs of Cd intoxication are unlikely with a daily dietary Cd intake of less than 5 mg/kg feed, which is 5–10 times higher than the maximum permitted Cd concentration in ruminant feed in the European Union. In farmed ruminants, Cd levels in tissue are largely dependent on the Cd content of diet. However, many factors affect Cd availability, relating to soils, plants and the presence of other trace elements including Ca, Cu, Fe, Mn, Mo, Se and Zn. Experimental studies have highlighted the ability of Cd to alter trace element status, and the protective effect of good mineral status, however, there remain gaps in knowledge of the impact of these interactions on the health and productivity of farmed animals.

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1. Introduction

Cadmium is considered one of the most toxic elements in the environment, with a wide range of organ toxicity and long elimination half-life (Patrick, 2003). Industrial processing and intensive agricultural practices, resulting in the contamination of forage, feed and water, are sources of Cd exposure for farmed ruminants. In many areas, high soil Cd concentrations are the result of intensified farming methods or industrial processes. However, naturally occurring areas of high Cd concentrations in soil also occur, and some geographical areas are also associated with naturally occurring high Cd concentrations. In Europe, these are found in Ireland (Canty et al., 2011, 2014), Poland, the Goslar district in Germany and southern Sardinia (Pan et al., 2010).

Most ruminants have a low Cd burden at birth. Cd accumulates over time, primarily in kidney and liver (Underwood, 1977; Langlands et al., 1988). Cd has no known biological function but mimics the actions of other divalent metals that are essential to diverse biological functions (European Food Safety Authority, 2009). Bioavailability, retention and consequently toxicity of Cd are affected by several factors such as nutritional status (low body Fe stores) and multiple pregnancies, pre-existing health conditions or diseases (European Food Safety Authority, 2009). Cd has the ability to cross various biological membranes and, once intracellular, to bind to ligands with exceptional affinity. Cd is a known human carcinogen (reviewed by Filipic et al., 2006). However, such effects have not been described in animals and therefore will not be discussed in this paper.

This paper reviews Cd exposure and consequences for the health and productivity of farmed ruminants.

2. Cadmium exposure

2.1. Cadmium sources

Cd is a naturally occurring heavy metal present at higher concentrations in association with Cd-rich soils, including shales, oceanic and lacustrine sediments, and phosphorites. However, more than 90% of Cd in the surface environment is the result of industrial and agricultural processes (Pan et al., 2010). The combustion of coal and mineral oil, smelting, mining, alloy processing and industries that use Cd as a dye (CdS: yellow; $CdSeO_3$: red) in their manufacturing processes (Swarup et al., 2007) are all potential sources of Cd for farmed ruminants, with exposure decreasing with distance from the pollution source (Vos et al., 1988). More generally, air concentrations of Cd of between 0.01 and 0.35 μg/m^3 have been reported (US Department of Health, Education and Welfare, 1966), with the highest concentrations in industrialised cities. Cd is also a pollutant in phosphate fertilisers (Järup, 2003), leading to Cd being added to land through normal farming practice (Roberts et al., 1994; Martelli et al., 2006). The Cd content in phosphate fertilisers varies considerably, depending on source, ranging from 3.6 to 527 mg/kg phosphorous in a study conducted on fertilisers available in Australia (Satarug et al., 2003). The long-term addition of phosphate fertiliser (30 kg P/ha/annum for 31 years) in Ireland has led to a 0.07 mg/kg increase in soil Cd levels (from 0.23 to 0.30 mg/kg) in the top 10 cm of the soil (Department of Agriculture, Food and Rural Development, 2000). In the EU, Cd content in fertilisers is not currently covered by the EU Fertiliser Directive 76/116/EEC. Sewage sludge is also recognised as an important source of Cd contamination (Patrick, 2003).

In non-polluted environments, a normal and maximum soil Cd concentration of 0.1 and 0.2 mg/kg has been suggested (Brooks, 1998). In contrast, soil Cd levels of 1 mg/kg are regarded as polluted soils (Fay et al., 2007). In plants grown in a variety of naturally and artificially polluted environments (following application of sludge or wastewater, in proximity with a smelter or following application of Cd salts), Cd concentrations were variable, but consistently higher than 0.1 mg/kg. Some plants in the genus *Thalspi* are recognised as hyperaccumulator plants, with Cd concentrations of 100 mg/kg or more in plant tissue, in particular penny-cress (*Thalspi caerulescens*) (Kirkham, 2006).

2.2. Dynamics of cadmium absorption, accumulation and detoxification

The respiratory and digestive systems have both been implicated in Cd absorption. Approximately 10 to 50% of Cd fumes are absorbed by the respiratory system. In contrast, only ~5% of oral Cd is absorbed, which is much less than similar divalent cations such as Zn and Fe. After inhalation, Cd accumulates in the olfactory bulb (Sunderman, 2001) and in cranial lung tissue (Roggeman et al., 2014). Once in the lungs, Cd can pass through alveolar cells and enter the blood stream, in contrast to other heavy metals (Bressler et al., 2004).

Wilkinson et al. (2003) present a detailed review of the accumulation of potentially-toxic metals, including Cd, by farmed ruminants. These animals can ingest Cd by consuming either contaminated herbage or soil, either following natural or artificial contamination. The concentration of Cd in herbage generally reflects that in soil, although a range of factors affect Cd availability, including soil pH, soil organic matter and plant species (Barančíková et al., 2004; Tudoreanu and Phillips, 2004; Kirkham, 2006; Phillips and Tudoreanu, 2011). Cd disperses to most root and shoot tissue, but can also be confined to the meristem. It is well recognised that antagonism between metals substantially influences Cd uptake. There are complex interactions between Cd and Zn, with Cd increasing Zn accumulation (Wilkinson et al., 2003). Further, the gastrointestinal absorption of Cd is strongly influenced by Fe status, suggesting that Cd and Fe are absorbed from the intestinal absorption through a similar mechanism (Öhrvik et al., 2007).

As a non-essential element, it is unlikely that Cd enters the body via a Cd-specific transport mechanism (Roggeman et al., 2014). Rather, Cd crosses various membranes utilising the transport mechanisms of other elements, including Ca (Martelli et al., 2006). Cd is bound to small cysteine-rich peptides, including metallothionein (MT), which are involved in the binding, transport and detoxification of excessive Cd (Nordberg et al., 1994), primarily through high affinity binding. Cd is recognised as the most important metal for MT induction in bovine kidneys (Roggeman et al., 2014). In the bloodstream, Cd is present as Cd-MT and Cd-albumin in plasma and Cd-MT in erythrocytes. Cd-MT is filtered by the kidney and re-absorbed in the proximal tubules where the complex is broken down leading to irreversible damage to tubular cells, particularly when the detoxification system is overwhelmed (Wilkinson et al., 2003). Cd is primarily stored in the liver and kidneys, which account for half of the body's total stores of Cd, with the balance in bone, pancreas, adrenals and placenta (Pope and Rall, 1995). The rate of Cd excretion, primarily in urine, is slower than that of uptake, highlighting the need for animals detoxify and store excessive Cd (George and Coombs, 1977; Wilkinson et al., 2003; Klaassen et al., 2009).

3. Cadmium concentrations in exposed animals

3.1. Blood concentrations

3.1.1. Observational studies

High blood Cd concentrations have been reported from areas of high Cd exposure. In India, cows reared and kept near a steel manufacturing plant had a mean blood Cd concentration of 232 μg/l (ranging from 90 to 410 μg/l). For comparison, a mean blood Cd concentration of 28 μg/l (ranging from not detectable to 50 μg/l) was measured in cows from a non-polluted area (Patra et al., 2005). Patra et al. (2007) reported Cd concentrations of 127 μg/l (ranging from non detectable to 410 μg/l) in cows held near a steel processing plant, compared to 25 μg/l (ranging from non detectable to 50 μg/l) in cows from a non-polluted area. Further work conducted by the same laboratory reported similar blood Cd concentrations in adult cows in another study (cattle near a steel processing plant: 230 μg/l (ranging from 90 to 410 μg/l); cattle