ASSESSMENT OF CADMIUM, LEAD AND VANADIUM STATUS
OF LARGE ANIMALS AS RELATED TO
THE HUMAN FOOD CHAIN

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ABSTRACT

Cadmium (Cd) and lead (Pb) are of established toxicological significance for humans, and it is important that intakes from foods not cause excessive exposure. Both elements concentrate to high levels in liver and kidneys of large, domestic food animals (cattle, sheep and swine), and Pb also concentrates in bone, which is sometimes used in the form of bone meal as a dietary supplement. Milk is a significant source of Cd and Pb for infants and toddlers. Levels of these elements in domestic food animal tissues and milk are presently within acceptable ranges for human consumption. Vanadium (V) sources to animals may increase and create potential human food problems; however, at the present time meats and milk are safe to consume with respect to this element.

(Key Words: Minerals, Toxicity, Food Animals, Cadmium, Lead, Vanadium.)

Introduction

Restriction of an animal's dietary intake of a toxic element is based on either 1) accumulation of the element to undesirably high levels in tissues consumed by humans or 2) toxicity to the animal, whichever is lower. The maximum tolerable levels of dietary cadmium (Cd) and lead (Pb) for domestic animals (NRC, 1980a) are based on human food residue considerations. The animals of concern here are cattle, sheep and swine. One purpose of this review is to summarize factors important in evaluating the safety of a high level of a metal in a food. For Cd, Pb and vanadium (V), information is reviewed on the toxicity of each element to humans and to domestic animals, standards for tolerable intakes, sources of each element, factors affecting uptake and toxicity and relationships between dose and tissue accumulation. The information in this summary updates the NRC (1980a) reviews for each element and forms the basis for evaluating an animal's status relative to the human food chain.

Mahaffey (1977) described certain aspects of the regulatory role of the Food and Drug Administration (FDA) with respect to mineral concentrations in animal tissues. Much of that information is still relevant. Although each problem of elevated levels of a toxic element in a food may present some unique aspects, the process of evaluating the impact on human health is generally the same.

Since toxic levels of an element in foods may be relatively low, the accuracy of the analyses must be assured at the outset. This is seldom a problem today, but it was a common problem only a few years ago.

For several years the FDA has assessed the background level of several elements in the U.S. diet through analysis of foods. The original design was based on analysis of foods composited by commodity groups. Foods were selected based on a food consumption survey conducted in 1965 by the USDA.

The Food and Agricultural Organization/World Health Organization (FAO/WHO) Expert Committee on Food Additives (1972) established provisional tolerable weekly intakes of Cd and Pb (table 1). They have recently established provisional tolerable intakes for Pb for infants and children, i.e., one-half that of the adult level per kg body weight (FAO/WHO Expert Committee on Food Additives, 1987). When the scientific base for an international standard (or a national standard) is applicable to the U.S., it is used by the FDA in assessing...
TABLE 1. CADMIUM, LEAD AND VANADIUM: INTAKES AND PROVISIONAL TOLERABLE INTAKES OF THE ADULT HUMAN MALE (70 KG) AND MAXIMUM TOLERABLE LEVELS OF DIETARY MINERALS FOR DOMESTIC ANIMALS

<table>
<thead>
<tr>
<th>Item</th>
<th>Cd</th>
<th>Pb</th>
<th>V</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human Intake, μg/d</td>
<td>20^a</td>
<td>41^a</td>
<td>12 to 30^b</td>
</tr>
<tr>
<td>Provisional tolerable intake, μg/d</td>
<td>70 to 84^c</td>
<td>497^c</td>
<td></td>
</tr>
<tr>
<td>Animal Intake, μg/kg diet</td>
<td>50^d</td>
<td>360^d</td>
<td>570^d</td>
</tr>
<tr>
<td>Maximum tolerable levels, μg/kg diet</td>
<td>500^e</td>
<td>30,000^e</td>
<td>50,000^e (10,000)^e</td>
</tr>
</tbody>
</table>

^aGartrell et al. (1986b). Calculation based on an energy intake of 2,850 kcal/d.
^cFAO/WHO Expert Committee on Food Additives (1972). The provisional tolerable weekly intakes are calculated for amounts per day.
^dCapar et al. (1978). Typical feed lot diet, dried, containing corn 70%, hay 3%, beet pulp 5%, corn silage 20% and mineral supplement 2%.
^eNRC (1980a). Maximum tolerable dietary values are for cattle, sheep and swine, except that the V value for swine (in parentheses) was obtained by interspecies extrapolation. Values for Cd and Pb are based on human food use considerations.

the potential hazard. The effect of the excess element on high-risk population groups must be evaluated. Infants, young children, pregnant and lactating women and the elderly are most frequently in this category. Low intakes of specific nutrients can increase risk. Clinical toxicity, total body burden and rates of clearance of the element must be taken into account. Another factor is that the intake of the contaminated food, both frequency and amount consumed, must sometimes be estimated for high-risk populations.

Finally a margin of safety needs to be applied to protect against extreme levels of intake and(or) extreme sensitivity to the toxic element. Seldom is the data base as broad as desired, but great strides have been made in the past few years in developing improved data bases (such as those on food intake) and in improving approaches to coordinating the disparate parts of risk assessment. Experts from several disciplines are required in reaching the best final decisions.

Cadmium

Significance for Humans. The toxicological and epidemiological aspects of Cd that are related to human health have recently been summarized (Friberg et al., 1985, 1986). The kidney is the target organ for damage and, when the concentration of Cd in the renal cortex reaches approximately 200 μg/g, damage to the proximal renal tubules occurs and calcium (Ca), phosphorus, glucose, amino acids and small peptides are lost in the urine. These losses can lead to significant bone mineral depletion and fractures. The most severe form of this condition, Itai-itai disease, occurred in Japanese women who consumed elevated levels of Cd in their food and drinking water for approximately 20 yr. It is thought that they constituted a sensitive population because they had borne several children and had low intakes of Ca, iron (Fe), protein, fat and vitamin D.

Absorption of cadmium is typically 3 to 7%, although it may be considerably higher in individuals with low iron stores. It has been estimated that daily intakes of 200 to 390 μg Cd would result in a critical concentration of Cd in the renal cortex by 50 yr of age. The biological half-life of Cd in the human kidney is long and has been estimated to be 10 to 30 yr. Most of the cadmium in the body is bound to metallothionein, a small heat-stable protein found in the subcellular fraction. It has a high content of cysteine and no disulfide bonds, aromatic amino acids and cystine. It can bind zinc (Zn), copper (Cu), Cd and mercury. It typically has...
TABLE 2. DAILY CADMIUM INTAKES (OCTOBER 1980 TO MARCH 1982) OF INFANTS\textsuperscript{a}, TODDLERS\textsuperscript{a} AND ADULT MALES\textsuperscript{b} AS COMPARED WITH THE FAO/WHO PROVISIONAL TOLERABLE INTAKE\textsuperscript{c}

<table>
<thead>
<tr>
<th>Item</th>
<th>Infant</th>
<th>Toddler</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject</td>
<td>Age</td>
<td>Body wt, kg</td>
<td></td>
</tr>
<tr>
<td></td>
<td>.5 to .9</td>
<td>8.2</td>
<td>2</td>
</tr>
<tr>
<td>FAO/WHO provisional tolerable daily intake, \textmu g/kg body wt</td>
<td>1 to 1.2</td>
<td>1.38</td>
<td>1 to 1.2</td>
</tr>
<tr>
<td>Estimated daily intake, \textmu g/kg body wt</td>
<td>1 to 1.2</td>
<td>1.13</td>
<td>1 to 1.2</td>
</tr>
<tr>
<td>Source of intake\textsuperscript{d}, %</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drinking water</td>
<td>8.58</td>
<td>13.82</td>
<td></td>
</tr>
<tr>
<td>Whole milk</td>
<td>54.51</td>
<td>30.10</td>
<td></td>
</tr>
<tr>
<td>Other dairy products\textsuperscript{e}</td>
<td>4.85</td>
<td>1.01</td>
<td></td>
</tr>
<tr>
<td>Dairy products</td>
<td>4.55</td>
<td>12.34</td>
<td>9.65</td>
</tr>
<tr>
<td>Meat, fish, poultry</td>
<td>7.96</td>
<td>17.12</td>
<td>36.02</td>
</tr>
<tr>
<td>Grain and cereal products</td>
<td>1.52</td>
<td>6.46</td>
<td>24.06</td>
</tr>
<tr>
<td>Potatoes</td>
<td>12.57</td>
<td>5.60</td>
<td>9.50\textsuperscript{f}</td>
</tr>
<tr>
<td>Vegetables</td>
<td>.94</td>
<td>4.17</td>
<td></td>
</tr>
<tr>
<td>Fruits and fruit juices</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fruits</td>
<td>.62</td>
<td>3.57</td>
<td>3.20</td>
</tr>
<tr>
<td>Oils and fats</td>
<td>.05</td>
<td>1.81</td>
<td>3.16</td>
</tr>
<tr>
<td>Sugar and adjuncts</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beverages</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>99.98</td>
<td>99.99</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Gartrell et al. (1986a).
\textsuperscript{b}Gartrell et al. (1986b). Cd intake was calculated for an energy intake of 2,850 kcal/d.
\textsuperscript{c}FAO/WHO Expert Committee on Food Additives (1972). The provisional tolerable weekly intakes are calculated for amounts per day.
\textsuperscript{d}Due to differences in consumption patterns, food groups varied with age, e.g., drinking water was included in beverages for adults.
\textsuperscript{e}Includes substitute dairy products.
\textsuperscript{f}Leafy vegetables 6.45%, legume vegetables 1.19% and root vegetables 1.86% of total intake.
\textsuperscript{g}Garden fruits 4.54% and fruits 1.66% of total intake.

a very high metal content, 5 to 10% by weight. Once Cd accumulates in tissues it cannot be removed safely by chelation therapy without causing kidney damage. Cd levels in foods, therefore, merit careful consideration and scrutiny.

Information on Cd intakes, standards and dietary sources are shown for infants, toddlers and adults in table 2. On the basis of body weight, the intakes of Cd by the infant and toddler are slightly above and below the FAO/WHO provisional tolerable intake, respectively. Values for the infant apply only to those fed formula; no human milk was analyzed. The highest proportion of Cd for each young age group came from milk, with drinking water; meat, fish and poultry products; grain and cereal products; and vegetables also supplying significant amounts. For the infant, the total Cd intake between 1978 and 1980 was lower than between 1980 and 1982, whereas the intake for the toddler was variable (Gartrell et al., 1986a). The adult male's intake of Cd was well below the FAO/WHO provisional tolerable intake. The greatest amounts came from dairy products, grain and cereal products, potatoes and vegetables. Between 1978 and 1982 the Cd intake of the adult male was relatively constant (Gartrell et al., 1986b).

Pennington et al. (1984) reported the proportions of essential minerals supplied by food groups of the FDA total diet study for
infants, toddlers and the adult male. These included Ca, Cu, Fe and Zn, the principal minerals that interact with Cd (Fox, 1979). Although milk was the main source of Cd for the infant and toddler, it also was the principal source of Ca and Zn. Grain and cereal products supplied significant amounts of Cd for the toddlers and the adult male; however, they were also major dietary sources of Cu, Fe and Zn for most age groups and of Ca for the adult. Fruits and vegetables (particularly potatoes) supplied Cd in the adult male diet; however, this commodity group was a source of Cu, Fe and, to some extent, Zn. Thus for specific age groups, the foods that supplied Cd were also important dietary sources of one or more of the essential minerals that interact with Cd. The occurrence of the essential elements in the gastrointestinal tract with Cd at the time of absorption probably confers additional protection against Cd absorption. The FDA total diet data indicated overall low intakes of Cu, below the estimated safe and adequate daily dietary intake range (NRC, 1980b). The amounts of Fe for the infant and toddler, and of Zn for the toddler and adult male, were less than the recommended dietary allowances.

The meat, fish and poultry category of foods was not a major source of dietary Cd, but these foods were important sources of Cu, Fe and Zn. Liver and kidney are the principal meat sources of Cd, although kidneys per se are eaten less frequently than liver.

In view of the Cd intakes of all age groups in relation to FAO/WHO provisional tolerable intakes, it is considered important that Cd in the food supply does not increase. With respect to domestic animals, milk, liver and kidney are the important source foods. Depending on Cd concentration, a serving of liver could easily double the day's intake of Cd.

Significance for Domestic Animals. Information on intake and tolerable levels of Cd for domestic animals is summarized in table 1. The data for intake are from a careful analytical study of the concentration of 30 elements by four methods (as appropriate) in a feedlot diet and various animal waste and sludge samples. The level of Cd was one-tenth (dry weight basis) that of the maximum tolerable level (NRC, 1980a) but was lower than other control diets. As in humans, Cd accumulates slowly in animal tissues, primarily in the liver and kidney, and has a long biological half-life. At very high dietary levels Cd can cause decreased feed intake and weight gain, anemia, decreased bone mineral and abortions (NRC, 1980a).

The levels of Cd in animal feeds are related to soil and fertilization practices where plant foods are grown. Some phosphate fertilizers and municipal sludges contain significant amounts of Cd. Van Bruwaene et al. (1984) reviewed Cd contamination in agriculture and zootechnology.

The effects of nutrients that interact with Cd in experimental and domestic animals have been reviewed (NRC, 1980a). These include Ca, Cu, Fe, Zn, protein (amount and type) and vitamins C and D. Recently Lamphere et al. (1984) showed reduced concentrations of Cd in blood, liver, kidney cortex and muscle of calves fed supplemental Zn with Cd compared with those fed Cd alone. Pond and Yen (1983) reported decreased toxicity and tissue concentrations of Cd in swine fed clinoptilolite, a natural zeolite. Both of these approaches require further work before practical applications are made to reduce Cd in tissues of food animals.

Telford et al. (1982) fed sheep silage made from corn plants fertilized with municipal sludge. The control and sludge-grown silages contained .29 and 3.88 mg Cd/kg, respectively. After 255 d, the liver and kidneys of sheep fed sludge-grown corn silage contained 11 and 6 times as much Cd, respectively, as those of the controls. Cd in muscle was not affected by treatment. Somewhat similar results were also reported by Telford et al. (1984) for sheep fed a diet containing control and sludge-fertilized hay for 102 d. The effect of treating pastures with anaerobically digested urban sludge was reported in cattle exposed for 8 yr (Fitzgerald et al., 1985). Compared with controls, tissues from experimental cattle contained 7, 5, 2 and 1.5 times as much Cd in liver, kidneys, muscle and milk, respectively. Although data were reported for 64 experimental and 29 control animals, it is not clear whether there were real differences for muscle and milk because there were no statistical analysis. The data for the controls agree well with values reported by Penumarthy et al. (1980) for commercially slaughtered cattle less than 2 yr of age.

Low uptake of dietary Cd into milk and muscle of lactating dairy cows has been thoroughly investigated in recent studies. Sharma et al. (1982) fed 0, 40 or 200 mg Cd (as the chloride) per day, which provided .18, 2.40 and
11.29 mg/kg diet, respectively. After 3 mo, there were dose-related increases of Cd in the liver and kidneys, but none in the blood, milk and muscle. Values for Cd in milk ranged from 12 to 22 ng/g, and in muscle from .02 to .06 µg/g. Cd in the liver and kidneys persisted 3 mo after the Cd was removed from the diet. In two studies with radioactive Cd, Van Bruwaene et al. (1982, 1983) found very low concentrations in a large number of tissues, except for liver and kidney, after 131 d. There were also substantial amounts of Cd in the intestinal tract. Radioactive Cd, fed as intrinsically labeled Zea mays leaves or as the chloride sprayed on the leaves was taken up by the tissues to a lesser extent than CdCl₂ given in a gelatin capsule. The highest proportion of radioactive Cd in the milk was bound to casein, with lesser amounts bound to albumin and lactose, and none was detected in fat.

Medians and ranges of Cd concentrations in liver, kidney and muscle were reported for 86 to 95 swine (less than 6 mo of age) and beef cattle (less than 2 yr of age) slaughtered at midwest Federally-inspected meat plants (Penu-marthy et al., 1980). Cd was measured in all samples of liver and kidney, but in only about two-thirds of the muscle samples (detection limit .005 µg/g). Lowest, median and highest values (µg/g) were as follows: pork liver .015, .051 and .470; beef liver .034, .105 and .430; pork kidney .037, .184 and 2.470; beef kidney .075, .565 and 2.500. The median and highest values (µg/g) for longissimus muscle were .014 and .065 for pork and .006 and .034 for beef. It is obvious that liver and kidney can supply significant amounts of Cd.

**Significance for Humans.** A comprehensive summary of the human health effects of Pb has recently been published (Mahaffey, 1985). Pb can adversely affect heme synthesis, neurological systems and kidney function. Cognitive function in children can be compromised to a small but significant extent by relatively low levels of Pb. Infants and young children are at greatest risk due to high absorption rates (46% for children vs 15% for adults) and intake from environmental sources. Pb accumulates in highest concentrations in liver, kidney and bone. Turnover in the bone is slow but is accelerated by bone remodeling or loss of bone mineral. It is well established that low intakes of Ca and Fe increase Pb absorption. The effects of Ca in milk are less than those of inorganic Ca supplements in reducing Pb absorption. A number of dietary factors appear to influence Pb absorption, so it is difficult to evaluate specific relationships. Fasting markedly increases the amount of Pb absorbed from an aqueous dose in the absence of food.

Sources of dietary Pb in comparison to the FAO/WHO provisional tolerable intakes are shown in table 1. On the basis of body weight, Pb found in the FDA total diet was below the FAO/WHO standard for the infant, toddler and adult male. There did not appear to be any long-term trends in dietary Pb levels between October 1978, and September 1982; however, the lowest values for each age group were reported for 1981–82 (Garrett et al., 1986a,b). Significant amounts of Pb were found in most food groups. For the infant and toddler, milk was an important source of Pb; however, the highest amounts came from fruit and fruit juices. For the adult male, grains and cereal products, vegetables (particularly legume vegetables) and fruits were the principal sources of Pb.

Although the FDA total diet study shows levels of Pb below the FAO/WHO standards, it is considered desirable that Pb levels in foods should not increase. This is particularly important for those infants and young children who are exposed to high levels of environmental Pb from such sources as Pb piping used for drinking water, peeling lead paint, or high lead soil in play areas. The latter problems are expected to persist.

**Significance for Domestic Animals.** Human and domestic animal intakes and tolerable standards of intake for Pb are shown in table 1. Values for animals are much higher than those for humans, and Pb in many animal diets is higher than that shown. The maximum tolerable level for animals was based on considerations of Pb concentrations in edible tissues and on a limited period of consumption.

Effects of Pb toxicity in domestic animals include accumulation of Pb in tissues (particularly liver, kidney and bone), decreased feed intake and growth, rumen stasis, diarrhea, weakness, muscle tremors, anemia, excess salivation, blindness, maniacal excitement (young calves) and death (NRC, 1980a). Calves are the domestic food animal most sensitive to Pb toxicity and the animal most commonly poisoned. Blakley (1984) reported that in 294 cases of Pb poisoning in western Canadian
TABLE 3. DAILY LEAD INTAKES (OCTOBER 1980 TO MARCH 1982) OF INFANTS\textsuperscript{a}, TODDLERS\textsuperscript{a} AND ADULT MALES\textsuperscript{b} AS COMPARED WITH THE FAO/WHO PROVISIONAL TOLERABLE INTAKES

<table>
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<th>Item</th>
<th>Infant</th>
<th>Toddler</th>
<th>Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject</td>
<td>.5 to .9</td>
<td>2</td>
<td>19+</td>
</tr>
<tr>
<td>Body wt, kg</td>
<td>8.2</td>
<td>13.7</td>
<td>70</td>
</tr>
<tr>
<td>FAO/WHO provisional tolerable daily intake, μg/kg body wt</td>
<td>3.6\textsuperscript{c}</td>
<td>3.6\textsuperscript{c}</td>
<td>7.1\textsuperscript{d}</td>
</tr>
<tr>
<td>Estimated daily intake, μg/kg body wt</td>
<td>2.43</td>
<td>2.22</td>
<td>.59</td>
</tr>
<tr>
<td>Source of intake, %</td>
<td></td>
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</tr>
<tr>
<td>Drinking water</td>
<td>4.97</td>
<td>7.16</td>
<td></td>
</tr>
<tr>
<td>Whole milk</td>
<td>18.34</td>
<td>10.58</td>
<td></td>
</tr>
<tr>
<td>Other dairy products\textsuperscript{f}</td>
<td>10.20</td>
<td>3.05</td>
<td></td>
</tr>
<tr>
<td>Dairy products</td>
<td>6.38</td>
<td>5.49</td>
<td></td>
</tr>
<tr>
<td>Meat, fish, poultry</td>
<td>8.79</td>
<td>11.67</td>
<td></td>
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<tr>
<td>Grain and cereal products</td>
<td>5.2</td>
<td>2.38</td>
<td></td>
</tr>
<tr>
<td>Potatoes</td>
<td>10.65</td>
<td>17.09</td>
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</tr>
<tr>
<td>Vegetables</td>
<td>33.72</td>
<td>30.00</td>
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</tr>
<tr>
<td>Fruits</td>
<td>94</td>
<td>1.85</td>
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</tr>
<tr>
<td>Oils and fats</td>
<td>2.42</td>
<td>3.22</td>
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</tr>
<tr>
<td>Sugar and adjuncts</td>
<td>3.05</td>
<td>7.53</td>
<td></td>
</tr>
<tr>
<td>Beverages</td>
<td>99.98</td>
<td>100.02</td>
<td>100.02</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Gartrell et al. (1986a).
\textsuperscript{b}Gartrell et al. (1986b). Pb intake was calculated for an energy intake of 2,850 kcal/d.
\textsuperscript{c}FAO/WHO Expert Committee on Food Additives (1987). The provisional tolerable weekly intakes are calculated for amounts per day.
\textsuperscript{d}FAO/WHO Expert Committee on Food Additives (1972). See footnote c.
\textsuperscript{e}Due to differences in consumption patterns, food groups varied with age, e.g., drinking water was included in beverages for adults.
\textsuperscript{f}Includes substitute dairy products.
\textsuperscript{g}Leafy vegetables 1.67%, legume vegetables 16.25% and root vegetables 1.86% of total intake.
\textsuperscript{h}Garden fruits 7.86% and other fruits 17.70% of total intake.

The cattle exposed for 8 yr to pasturing forage on land fertilized with sewage sludge described above for Cd effects (Fitzgerald et al., 1985) also ingested more Pb than control animals. The Pb concentrations in blood and bone were higher in cattle grazing on sludge-grown plants than in those eating control plants; however, there were no differences in Pb concentrations in milk or five types of soft tissue.

Many nutrients and other dietary components that affect Pb uptake by tissues and toxicity in humans and experimental animals (NRC, 1980a) have not been studied in domestic food animals. Pearl et al. (1983) found that sheep fed 1,000 mg Pb/kg with either .25 or .50% cattle (1968 to 1982), 51.9% occurred in animals 6 mo of age or less. Poisoning was more common in dairy than beef breeds.

Although point-source contamination, some sewage sludges or contaminated feed may supply Pb, the most common sources include Pb-based paints and Pb-containing materials at dump sites (Pb batteries, putty, asphalt roofing, linoleum, etc.). An outbreak of Pb toxicity occurred in dairy cows fed haylage grossly contaminated with Pb shot (Frape and Pringle, 1984). Whereas Pb shot are relatively inert in the ruminant digestive tract, the acid conditions in the haylage solubilized sufficient Pb to be severely toxic.
dietary Ca accumulated the same concentrations of Pb in blood, five types of soft tissue and bone. An exception was the liver, which contained more Pb with lower Ca. Zmudzki et al. (1984, 1985) showed that calves fed an adequate milk replacer diet were very sensitive to Pb toxicity, whereas calves receiving the same amount of Pb in a grain and hay diet appeared normal and had lower Pb concentrations in tissues than milk-fed calves. When 10 or 40% lactose or 40% milk replacer diet was fed with grain and hay, the Pb concentration in blood was higher than with grain and hay fed alone but not as high as with milk replacer diet fed alone (Zmudzki et al., 1986). Similar effects on Pb concentrations were observed in some tissues.

Sharma et al. (1982) fed cows Pb acetate at two supplemental levels to provide dietary concentrations of 3.67 (control), 7.23 and 31.45 mg Pb/kg diet. After 3 mo there were no changes in muscle Pb; however, there were dose-related increases in blood, liver, kidney and bone Pb. In milk, Pb increased only with the highest dietary Pb level. Three months after removing Pb from the diet, Pb concentrations had markedly declined in all tissues except bone, where it was still elevated above the control. Logner et al. (1984) added 500, 1,500 or 4,500 mg Pb/kg to a control diet that contained 1.42 mg/kg. When these diets were fed to calves, all calves fed the highest level and one calf fed the intermediate level died by 6 to 10 d. After 7 wk, kidneys and liver in surviving calves contained dose-related concentrations of Pb, whereas increases in bone, brain and muscle Pb occurred only with the 1,500 mg Pb/kg diet.

Responses to graded levels of Pb were studied in sheep (Fick et al., 1976). Ten, 100, 500 and 1,000 mg Pb/kg were fed as the acetate in a control diet that contained 3.4 mg Pb/kg. For 84 d, all animals grew and developed normally without changes in hemoglobin, hematocrit or blood Pb. Pb concentrations were increased by both 500 and 1,000 mg Pb/kg in liver, bone, brain and spleen, whereas increases occurred in the kidneys, heart and skeletal muscle only with 1,000 mg Pb/kg diet. When sheep were fed 1,000 mg Pb/kg for 75 d followed by the control diet (3 mg Pb/kg) for 180 d, the elevated Pb levels in tissues at 75 d decreased during the period of Pb withdrawal (Pearl et al., 1983). These tissues included brain, spleen, kidney, liver, skeletal muscle, heart and bone. Kidney was the only tissue that returned to baseline Pb concentrations. Bone was the only tissue that accumulated higher amounts of Pb beyond the Pb-feeding period, and the ultimate return toward baseline was proportionately the smallest among the tissues studied.

Penumarthy et al. (1980) determined Pb in four types of tissue and in the blood of healthy swine and cattle slaughtered as described previously for Cd. Most blood, liver and kidney samples and about 70% of muscle samples for both species contained Pb (detection limit .02 μg/g). The median and highest values of Pb in swine tissues (μg/g) were as follows: liver, .06 and .25; kidneys, .06 and 1.04; and longissimus muscle, .06 and 2.60. The median and highest values (μg/g) for cattle were as follows: liver, .11 and .50; kidneys, .19 and 1.83; and muscle, .02 and .34.

Bratton and Zmudzki (1984) reviewed clinical indices that may be useful in diagnosing Pb poisoning and assessing the body burden of Pb. They concluded that blood Pb, erythrocyte aminolevulinic acid dehydratase and circulating Zn protoporphyrin should all be determined if possible. A great deal of work remains to establish the data base needed for precise interpretation of these values in veterinary medicine.

**Vanadium**

**Significance for Humans.** Much less is known about V excess in humans than about the effects of Cd and Pb. The possibility of increasing levels of V in food animal tissues, primarily from mineral supplements and plant fertilizers, led to its consideration here. Data on occurrence in human foods are limited and no standard for tolerable intakes from foods have been established (table 1). Effects of V and its toxicity have been reviewed (Vouk, 1979; Nechay, 1984; Nechay et al., 1986). Toxicity in humans by the oral route appears to be unknown. Industrial exposure to airborne, V-bearing dusts have caused cough and irritation of the respiratory passages, reduced lung function, green coloration of the tongue, metallic sense of taste, conjunctivitis, dermatitis, dizziness, cardiac arrhythmias, anemia and decreased cystine in fingernails. Concentrations of V₂O₅ that produced adverse effects in workers ranged from .2 to 104 mg/m³.

Approximately 1 to 2% of ingested V is absorbed from the intestinal tract. Most of this is rapidly excreted in the urine, and long-term
accumulation does not appear to occur. The total body pool of the "reference human" is about 100 μg, most of which occurs intracellularly. V can exist in oxidation states ranging from -1 to +5. The two forms of importance in biological systems are the vanadate ion, VO₃⁻ (pentavalent) and the vanadyl ion, VO²⁺ (tetravalent), which occur extracellularly and intracellularly, respectively. Vanadate ion is a potent inhibitor of the Na⁺,K⁺-ATPase system as well as an inhibitor or stimulator of many enzymes.

Significance for Domestic Animals. Information on the toxicity of V in domestic animals is somewhat limited (NRC, 1980a). The maximum tolerable level for swine was based on interspecies extrapolation (table 1). Effects of V toxicity include accumulation of V in liver, kidney, bone and other tissues; reduced feed consumption, poor growth, prostration, hemorrhage, nephritis and death. There has been concern about the high levels of vanadium found in some phosphate rock used for animal feeding (Ammerman et al., 1977).

Sheep were fed 10, 100, 200, 400 or 800 mg V (as ammonium metavanadate)/kg control diet, which contained 2.2 mg V/kg (Hansard et al., 1978). After 1 d, animals receiving the two highest levels refused to eat. They were returned to the control diet and recovered within 5 to 8 d. All other sheep grew and developed normally over the 84-d study. On a dry-weight basis, muscle accumulated the least V and kidney the greatest amount, whereas bone and liver had similar concentrations. Sheep fed the diets containing 200 mg V/kg (91 times the control level) had concentrations in the muscle, liver, bone and kidney that were 10, 23, 17 and 48 times the respective control values.

A 100-g serving of fresh liver from the sheep receiving 0, 10, 100 or 200 mg supplemental V/kg diet would provide approximately 3.6, 5.4, 29 and 84 μg V (based on 70% water content of fresh liver). A serving at the higher levels would substantially increase the daily intake of V. Assuming that the adult male consumes approximately 640 g air-dry, fiber-free diet/day (Fox et al., 1981), a 100-g serving of liver with the highest content would be equal to only about 131 μg V/kg air-dry, purified diet. For the domestic species most sensitive to V, the chicken, toxic effects usually start around 10 mg/kg diet (NRC, 1980a). On this basis, it does not appear likely that there is apt to be a problem with V toxicity from animal tissues in the near future. Information is lacking, however, on toxicity in humans exposed to V by the oral route and on bioavailability of V from foods.

Literature Cited


