Sulfur-related Polioencephalomalacia in Cattle

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Features of PEM

Polioencephalomalacia:
- Polio = gray matter
- Encephalo = brain
- Malacia = softening or tissue death

Clinical signs
- Subacute; blindness and staggering
- Acute; blindness, recumbence, seizures

(Se “blind staggers” and related myths, O’Toole, Vet Path 1996)
Polioencephalomalacia:

- Polio = gray matter
- Encephalo = brain
- Malacia = softening or tissue death

It’s a non-specific lesion –
causes include:

- Lead toxicity
- Salt toxicosis/water deprivation
- Thiamine deficiency
- Excessive sulfur intake
PEM UV Illumination
PEM microscopic lesion

Normal nerve cell

Dead nerve cell
Chronic PEM taken at slaughter

Abnormal

Normal
Potential Sulfur Sources

- Water
- Sulfate accumulation by grasses and weeds
- Cruciferous plants – turnips, rape, kale and oil seed crops / meals
- Mismixed feeds (mineral sulfates), sulfate additives as feed intake limiters or sorting of pelleted supplements
- By-products of corn and sugar processing – sulfur added in process
Experimental Studies at CSU

- Low fiber, high sulfate (≈0.3% S), high CHO diet consistently induces PEM
- Thiamine status is unaltered during development and expression of PEM
- Odor of H₂S in eructated gas when clinical signs begin
Gas Cap Sampling
Gas Cap Sampling
H$_2$S Detector Tubes
Exper Investigations – H$_2$S in Gas Cap

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<tr>
<th>DAYS</th>
<th>ppm H$_2$S x 10$^3$</th>
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<tr>
<td>-4</td>
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+ S$_4$ (median, range)
- S$_4$ (median, range)
Types of Sulfur

- Elemental Sulfur - little toxicity
- Sulfate - little toxicity

but - with ruminal microbial metabolism, sulfur is reduced to

- Sulfide (H₂S) - high toxicity
  (similar to cyanide)

Total S should not exceed 0.3-.5% DM
PEM risk period

McAllister: JAVMA 1997, 211:1275

Days post arrival
Ruminal Gas Cap H$_2$S Production in Feedlot Steers

Loneragan: Bov Pract
2005, 39:16
PEM Risk Period and Ruminal \( \text{H}_2\text{S} \)

Loneragan: Bov Pract. 2005, 39:16
Risk Factors
Dietary [S] incl. H₂O plus risky behavior; (gluttony, sorting, and mixing)

Sulfate-reducing microbes
CHO
pH
Metals?
Thiamine
Prevention?

[\text{H}_2\text{S}]

\text{[H}_2\text{S]}\quad \uparrow

\text{[HS}^{-}\text{]}\quad \uparrow

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What about thiamine treatment of clinical case of PEM?

• Positive response to thiamine therapy does not prove thiamine deficiency is the cause

• Thiamine administration can have a non-specific beneficial effect on an energy deficient brain

Ex. Thiamine provides mitigates nervous signs in calves with lead poisoning