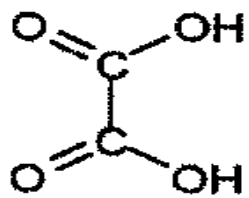
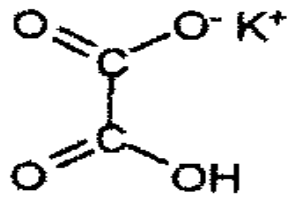
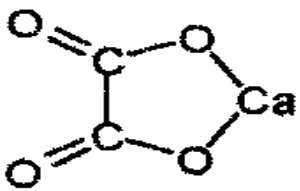
Oxalates



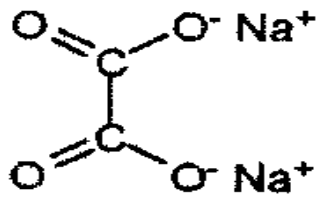
Oxalic acid



Acid potassium oxalate



Calcium oxalate



Sodium oxalate

Oxalate Toxicosis

Types of Oxalate			
Sodium oxalate	C-O-O - Na | C-O-O - Na	sap pH 6-7	Halogeton
Potassium	C-O-O - H | C-O-O - K	sap pH 3-4	Rumex Kikuya grass
Ammonium	C-O-O - NH_3 | C-O-O -NH_3		Grasses
Convert to Toxin	C-O-O\ | Ca C-O-O/		

Toxic Syndromes

1. Hypocalcemia

   Calcium in blood replaces ion on oxalic acid to form Ca-oxalate crystals, resulting in ionic imbalance and shock.

2. Uremia

   Oxalate crystals damage tubles in kidneys – buildup of urea cause poisoning.

3. Disrupt energy metabolism

   Interfere with succinic dehydrogenase and latic dehydrogenase in Krebs Cycle.

Oxalate Toxicology

1. Oxalates increase over the growing season – max in fall (20-36% dw plant)

2. Oxalate level > 18% potentially dangerous

3. Poisoning occurs when oxalate level exceeds the animals ability to detoxify.

    1. Oxalates are readily degraded by rumen microbes. Microbes adapt over 3-4 days to detoxify 75% more.
    2. Oxalates attach to Ca in rumen and excreted in feces.
    3. Oxalates are absorbed.
        a. Flushed out in urine
        b. If reach blood stream, cause damage
            Hypocalcemia
            Uremia
            Energy metabolism

Signs of Poisoning
Acute, Rapid Death

1. Depression
2. Anorexia – lack of appetite
3. Weakness
4. Incoordination
5. Recumbency
6. Blood tinged nasal discharge
7. Coma
8. Death

Treatment of Poisoned Animals

1. Flush with water - excrete oxalates in urine.

2. Di-calcium phosphate drench -
   Ca combine with oxalate in rumen.
   Ca supplement not sufficient to prevent poisoning

3. Intravenous injection of calcium gluconate.
   Maintain blood Ca, but crystals damage kidneys.

4. Recommendation – prevent poisoning

The Distribution of Halogeton in North America

ROBERT W. PEMBERTON

JOURNAL OF RANGE MANAGEMENT 39(3), May 1986

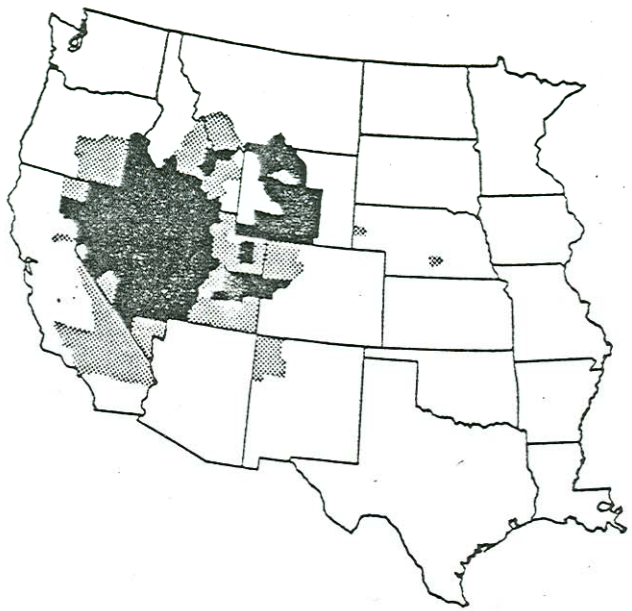


Fig. 1. *The distribution of* Halogeton glomeratus *in 1954 (black zones) and 1980 (black and grey zones). The grey zones are the areas to which halogeton has apparently spread since 1954. This map shows the occurrence of halogeton by county (the level of the survey) and does not intend to imply that halogeton occurs on all range sites within these counties.*

Catastrophic Losses

1942 – Wells NV – 160 sheep
1945 – Twin Falls ID – 275 sheep
1945 – Raft River Valley ID – 750 sheep
1945 – Raft River Valley ID – 1620 sheep
1952 – Park Valley UT – 1200 sheep

Life Magazine “Stock Killing Weed of the West”

Halogeton Act 1952

1. Detect presence of Halogeton
2. Determine its effect on livestock
3. Control, suppress and eradicate it


Acute Halogeton Poisoning of Sheep:
Pathogenesis of Lesions
Kent R. Van Kampen, D.V.M., Ph.D., and Lynn F. James, Ph.D.
Prevention of Fatal Halogeton glomeratus
Poisoning in Sheep
Lynn F. James, Ph.D., and A. Earl Johnson, M.S.
KILLING HALOGETON
WITH
CHEMICALS
ECOLOGICAL AND PHYSIOLOGICAL
FACTORS INFLUENCING
CHEMICAL CONTROL of
HALOGETON GLOMERATUS
Technical Bulletin No. 1325
Agricultural Research Service
UNITED STATES DEPARTMENT OF AGRICULTURE
in cooperation with the
Utah Agricultural Experiment Station
EXTENSION BULLETIN 250
The
HALOGETON Problem
in UTAH
UTAH STATE AGRICULTURAL COLLEGE
EXTENSION SERVICE
Logan, Utah
THE HALOGETON PROBLEM IN UTAH
C. Wayne Cook and L. A. Stoddart

Prevent Poisoning

1. Never turn hungry sheep onto halogeton.

2. Introduce sheep gradually to allow rumen microbes to adjust.

   Graze shadscale first (low oxalates) to allow microbes to build up.

3. Don’t overgraze – maintain range in good condition to prevent invasion.

4. Re-seed infested sagebrush sites.

   Halogeton can’t compete with vigorous perennial grasses and shrubs.