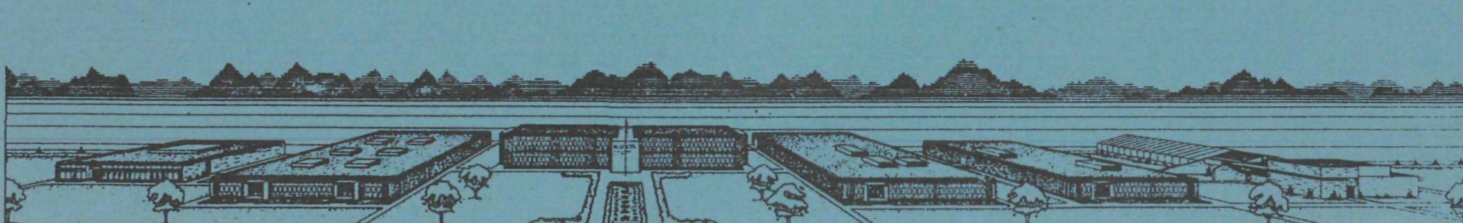
THE JANUARY 1971 SHEEP DEATH INCIDENT NEAR GARRISON, UTAH

by
Radiological Research Program.
Western Environmental Research Laboratory*

ENVIRONMENTAL PROTECTION AGENCY

Published November 1971

This research was performed as a part of the Radiation Effects Program and was supported by the U. S. Atomic Energy Commission under Memorandum of Understanding No. SF 54 373



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*Formerly Southwestern Radiological Health Laboratory, part of the U. S. Department of Health, Education, and Welfare, Public Health Service, Environmental Health Service, Environmental Control Administration, Bureau of Radiological Health.

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ABSTRACT

The acute death near Garrison, Utah, in January, 1971, of some 1,250 sheep from a flock of 2,600 was the object of national attention. The implied cause of either nerve gas from Dugway Proving Grounds or radiation from the Nevada Test Site was the principal newsworthy ingredient used to focus national interest and was the reason used to initiate several investigations to determine the true cause of the deaths.

Based on early accounts of the incident, scientists from the Western Environmental Research Laboratory postulated that weed poisoning, probably *Halogeton glomeratus*, was a probable cause of death. This postulate was subsequently confirmed by the field and laboratory results of several investigative groups. The findings of the investigation in which the Western Environmental Research Laboratory scientists participated are presented and discussed.

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PREFACE

A rapid response to situations involving potential radiation exposures is often decisive in reducing such exposures or in minimizing the apprehension of the general population. Recognizing this fact, the Radiological Research Program of the Western Environmental Research Laboratory has developed and maintains an ad hoc investigative capability which has been used effectively in past incidents. An adjunct capability is contained within the Animal Investigation Program which investigates alleged radiation illness in domestic and game animals.

The investigation described in this report resulted from the application of these capabilities to an incident which occurred in Utah where a large number of sheep had died. The circumstances surrounding the deaths indicated that radiation was not the most probable cause, but prudence required an exact determination.

The contributions of Dr. R. E. Stanley, Mr. K. W. Brown, Mr. E. M. Daley, Mr. D. N. McNelis and Dr. S. C. Black to the success of this study are greatly appreciated.

Special acknowledgement is given to Mr. J. J. Davis for providing the photographs used in this report.

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INTRODUCTION

On January 21, 1971, several news agencies reported that more than a thousand sheep had died suddenly in western Utah, near the small town of Garrison. The news releases implied the possibility of nerve gas or radiation as etiological agents. Because of these implications and the high incidence of sheep loss, the State of Utah, through the State Department of Agriculture, initiated a comprehensive investigation of the cause of the reported losses. In addition to the official investigation by the Utah Department of Agriculture, several other investigative groups were permitted to observe the incident and to conduct independent investigations on a non-interference basis. The findings reported herein are from a limited independent investigation conducted by scientists from the Western Environmental Research Laboratory of the Environmental Protection Agency.

The speculation by the news agencies of the possible connection with environmental contamination by nerve gas or radioactive materials was based on two prior incidents. In 1968, about 6,500 sheep died in Utah following a nerve gas testing operation at the Army's Dugway Proving Grounds. However, nerve gas testing at Dugway was officially stopped later in 1968. On December 18, 1970, an underground test at the Nevada Test Site, code-named Baneberry, accidentally vented and released radioactive material to the atmosphere. While some radioactive material was deposited on certain parts of Utah, the amount measured was several orders of magnitude below that required to produce acute mortality in sheep. Hence, neither of the two causes speculated appeared plausible, so other possible causes were considered by the Western Environmental Research Laboratory scientists. Based on a knowledge of the ecology of the area, the type of livestock involved, and the season of the year, it was suspected that a poisonous plant (Halogeton glomeratus) could be the cause of death.

Because of repeated speculation in the press that the sheep could have been killed by radiation, the Nevada Operations Office of the Atomic Energy Commission (NVOO/AEC) decided to offer assistance with the investigation and requested the participation of Western Environmental Research Laboratory scientists. The Western Environmental Research Laboratory provided a veterinarian and a botantist to the five-man team which was to be sent to the site.

Other members of the team were from the AEC and included an environmental scientist, a health physicist and public information officer.

The team was instructed to observe the investigation conducted by the Utah Department of Agriculture, obtain all pertinent facts surrounding the incident, and to offer any appropriate assistance required to expeditiously investigate the incident and determine the cause of death. Upon arrival at Garrison on January 7, 1971, the team met with a team of investigators from the University of Utah. The two groups proceeded to the site, a sheep range in Antelope Valley some 18 miles southeast of Garrison, Utah.

DESCRIPTION OF HALOGETON

Halogeton glomeratus is a member of the Goosefoot family. It is an annual, growing to a height of 5 to 60 cm. The plant resembles young Russian thistle (Salsola kali var. tenuifolia). The leaves are generally smooth, fleshy and sausage shaped. They are usually from 0.5 to 2 cm long, with a solitary white-colored hair about 3 mm long growing out of the extreme tip. (1)

Young halogeton plants are dark green to blue-green with red stems. Following fall and winter frosts the plant fades and becomes straw colored. Plants growing in dense stands may attain a height of only a few cm as shown in Figure 1; whereas widely spaced plants under favorable moisture conditions may reach a height of 30 - 60 cm. Regardless of its height this plant produces viable seed in relative abundance. (2)

Halogeton thrives in both saline and non-saline soils of semiarid regions. (3) Heavy infestations of halogeton occur in areas where the soil has been disturbed. Prime areas of growth are overgrazed ranges, sheep trails, along railroad beds, road margins, burned-over areas, and abandoned farm lands.

Halogeton probably originated as a contaminant in agricultural seeds which were imported from Russia. It was first recorded in the mid-thirties in Nevada. Since then it has spread over approximately two million acres in Nevada, Utah, Idaho, Wyoming, and Montana. Some occurrences have also been reported in California, Oregon, and Colorado. (4)

Figure 1. Close-up view of *Halogeton glomeratus* on the sheep range at Garrison, Utah. With the exception of the one horsebrush plant in the left background, the remainder of the plants shown are halogeton. The halogeton plants in this area did not exceed a height of 20 cm.

The toxicity of halogeton is based on its oxalate content. This plant becomes more toxic as the growing season advances, and is most toxic when it is frozen and dry, primarily because of the increase in concentration of oxalates particularly in the form of soluble oxalic acid. It is unpalatable while green but it is apparently acceptable to animals during fall and winter.

Sheep are the most frequently poisoned livestock from halogeton, although cattle have been occasionally affected. Losses occur mostly when hungry animals are trailed through or grazed on heavily infested areas.

The amount of halogeton that will kill a sheep varies according to the condition of the plant and is reported to be from 340 to 500 grams.[5] Animals fasted for a day or longer are poisoned by smaller amounts than sheep that have been feeding on other forage. The first symptoms of halogeton poisoning may occur in four to six hours after an animal eats a lethal amount. Poisoned animals have difficulty in breathing, appear weak and drowsy, show drooling and white froth about the mouth, have nasal discharge (usually bloody), lapse into a coma, and die within a few hours.[5]

FIELD EVALUATION

The vegetation of the area was juniper and sagebrush with large stands of halogeton throughout. In certain areas of the range, more than 60 percent of the vegetation present was halogeton. Figure 2 shows a panoramic view of the area and illustrates the vegetation characteristics. The vegetation in the foreground is predominantly halogeton. The light colored areas which are more distant are also halogeton. Of the various ecological factors which could have resulted in excessive halogeton population of the area, overgrazing appeared to be the most probable cause. Apparently, past grazing practices had been sufficient to produce the necessary soil disturbance and to limit the population of competitive non-toxic plants.

Another factor of undefined significance was the limited availability of water. The sheep satisfied their need for water by grazing near the snow

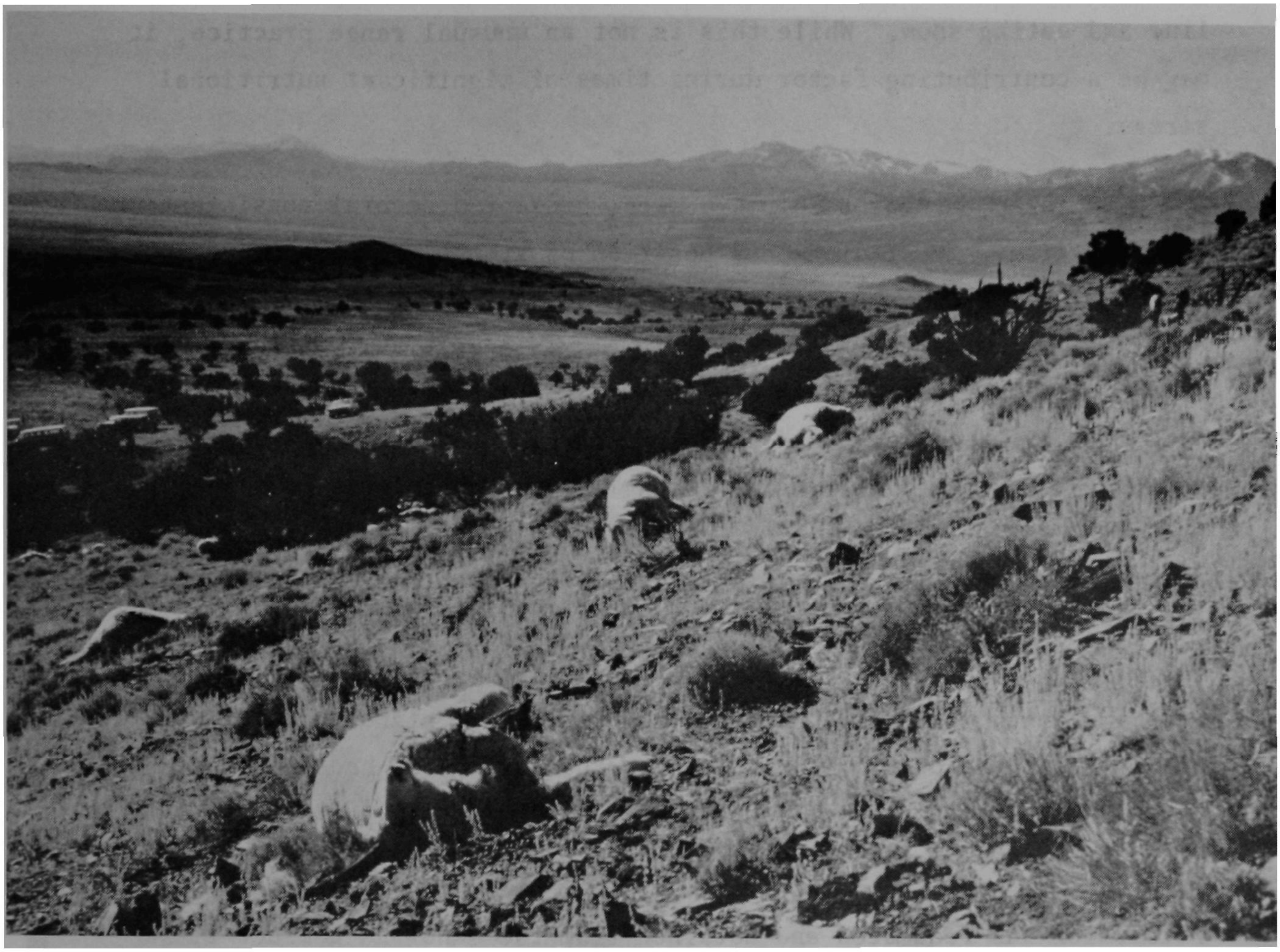

Figure 2. Plant populations on the Garrison range. Halogeton dominates the population in the immediate foreground, with juniper and sage appearing more distant.

line and eating snow. While this is not an unusual range practice, it may be a contributing factor during times of significant nutritional stress.

The dead sheep, pregnant Columbia ewes, presented several consistent external signs. Most died in a position of sternal recumbency with little evidence of severe pain prior to death. Figure 3 shows the carcasses strewn throughout the area. Occasionally some would present evidence of contraction of the neck muscles, since the head was drawn back. All the dead animals had reddish froth from the nose as shown in Figure 4. Interspersed among the carcasses were several animals in various stages of morbidity. All morbid animals exhibited dyspnea. Several were able to walk, but were unsteady and exhibited flexure of the distal joints in both front and rear legs. Other animals in recumbent positions were unable to stand. Although they would attempt to rise with prodding, they would fall before attaining a full standing position. Upon talking with the owner, it was learned that no deaths had occurred prior to January 21. About noon of January 22, approximately 1,250 of the original flock of 2,600 were dead.

Permission was requested and obtained from the owner to collect tissue and ingesta samples from the dead animals. Although the number of samples collected was less than intended, some useful information was obtained. While the history and symptoms along with the widespread distribution of halogeton throughout the area were almost conclusive enough for a diagnosis, an examination of rumen contents further enhanced this viewpoint. Prior to laboratory analysis of the samples gross observation indicated the halogeton content of the engorged rumen to be about 50 percent.

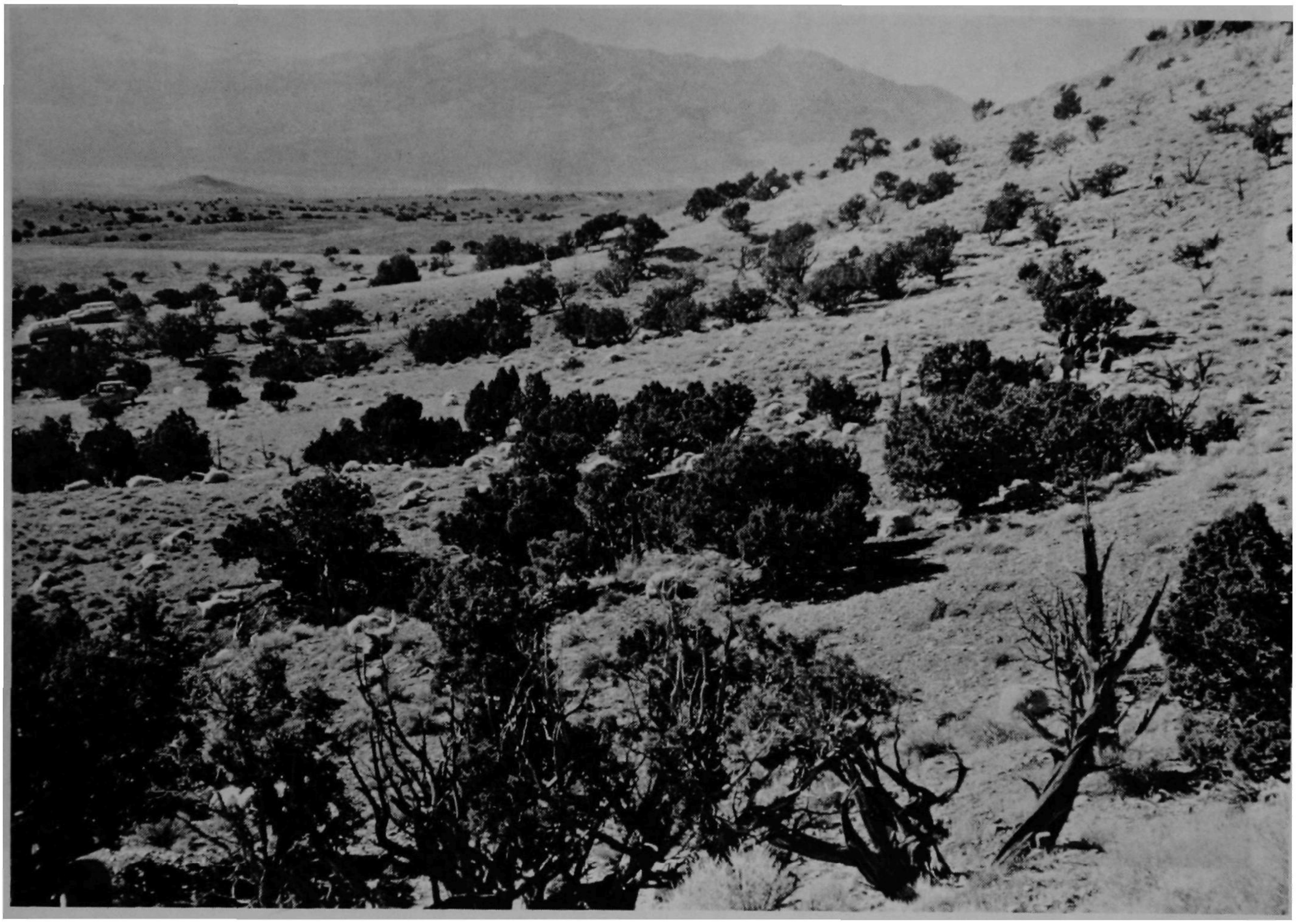

Figure 3. Garrison sheep range strewn with carcasses.

Figure 4. Dead sheep showing blood-tinged frothy exudate from the nose.

RESULTS AND DISCUSSION

Analytical results of the rumen contents collected from the three sheep are shown in Table 1, indicating an average halogeton content of 44 percent. It may be of interest to calculate the halogeton content of the rumens of these sheep for comparison to the amount reported as fatal for sheep. If the capacity of the sheep stomach is assumed to be approximately 15 liters and the capacity of the rumen alone is 80 percent of this value, or 12 liters,[6] the halogeton content of the entire stomach would have been 6.6 kg, with 5.3 kg contained in the rumen. As mentioned the lethal amount varies according to a number of parameters and is reported to be 340 - 500 g.[5] If 420 g is assumed as an average amount to produce acute lethality, these animals had an average rumen content which was about 12 times this amount.

The one thyroid collected weighed 7 grams and contained 350 pCi ^{131}I/g as of January 22. Using reported values [7] for ^{131}I in sheep thyroid; namely, (1) peak activity is attained in 12 - 15 days of daily ingestion, and (2) the effective half-life is about six days; the calculated peak concentration would be about 3,500 pCi/g. This calculated value is in agreement with other thyroid measurements made at a similar time on other animal thyroids collected from the off-site area.

The observations and findings reported here indicate the probable cause of death to be oxalate poisoning resulting from ingestion of fatal amounts of halogeton. This diagnosis was in agreement with that contained in the final report issued by the Utah Department of Agriculture. In the Utah report, "Halogeton Poisoning in Sheep, Antelope Valley (near Garrison), Utah, January 1971," Dr. F. James Schoenfeld, Utah State Veterinarian, and others, detailed all necropsy and laboratory findings and concluded that the cause of death was halogeton poisoning.

Table 1. Analytical Results of Rumen Contents from Three Sheep

Sheep #1

Scientific Name	Common Name	Plant Parts	% Composition
Artemisia arbuscula	Black sagebrush	Stems-Leaves	67
Halogeton glomeratus	Halogeton	Stems-Leaves-Seeds	21
Unidentified grass	Grass	Stems-Leaves	10
Cowania stansburiana	Cliffrose	Stems-Leaves	1
Herbaceous Fragments		Fragments	1
		Total	100 %

Sheep #2

Scientific Name	Common Name	Plant Parts	% Composition
Halogeton glomeratus	Halogeton	Stems-Leaves	47
Artemisia arbuscula	Black sagebrush	Stems-Leaves	45
Unidentified grass	Grass	Stems-Leaves	5
Atriplex confertifolia	Shadscale	Stems-Leaves	2
Herbaceous fragments		Fragments	1
Artemisia tridentata	Big sagebrush	Leaves	Trace
Unidentified shrub		Leaves	Trace
		Total	100 %

Sheep #3

Scientific Name	Common Name	Plant Parts	% Composition
Halogeton glomeratus	Halogeton	Stems-Leaves-Seeds	63
Artemisia arbuscula	Black sagebrush	Stems-Leaves	33
Unidentified grass	Grass	Stems-Leaves	3
Cowania stansburiana	Cliffrose	Stems-Leaves	1
Ephedra	Mormon tea	Stems	Trace
Juniperus osteosperma	Utah juniper	Leaves	Trace
Unidentified shrub		Leaves	Trace
		Total	100 %

REFERENCES

1. J. M. Fenley, How to Live with Halogeton by Limiting Its Spread, Agricultural Extension Service, University of Nevada Bulletin-106, 1952.

2. C. W. Cook and L. A. Stoddart, The Halogeton Problem in Utah:, Bulletin 364, Agricultural Experiment Station, Utah State Agricultural College, November 1953.

3. K. W. Brown and D. D. Smith, The Poisonous Plants of the U. S. Atomic Energy Commission's Nevada Test Site, Nye County, Nevada. SWRHL-33r. December 1966.

4. Poisonous Grassland Plants, Section 4 of a series -- Pasture and Range Plants, Phillips Petroleum Company, Copyright 1957.

5. Farmers' Bulletin No. 2106, United States Department of Agriculture, 16 Plants Poisonous to Livestock in Western States, prepared by the Animal Disease and Parasite Research Division and the Crops Research Division, Agricultural Research Service, Washington, D. C., 1951. Revised June 1963. Slightly revised August 1964. Available from U. S. Government Printing Office.

6. J. D. Grossman and S. Sisson, The Anatomy of the Domestic Animals, W. B. Saunders Company, Philadelphia and London, 1948.

7. L. K. Bustad, E. E. Elefson, E. C. Watson, D. H. Wood, H. A. Ragan, I^{131} in the Thyroid of Sheep and in Food, Thyroid, and Milk of Dairy Cows, Hanford Biology Research Annual Report for 1962, HW-76000, p. 60-62, Hanford Atomic Products Operation, Richland, Wash., 1963.

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