miles. One of us (R.W.) traveled the desert road between Gerlach and Summit Lake in the spring of 1957 and did not observe fissures in the 5-mile long area which is now strongly fissured; an alternate road now bypasses this fissured area.

Most of the fissures on the playa of the Smoke Creek Desert, although studied only from the air, appear to be older than those on the Black Rock Desert. The polygonal ground is more widespread on the Smoke Creek Desert, but open fissures were observed at only a few places. The older fissures appear to have been filled either by slumpage, flooding, or wind-blown sand. Lines of vegetation mark the polygonal blocks in one area at the north end of the Smoke Creek Desert. The polygonal ground at this locality looks much like that of the playa of the Animas Valley in New Mexico which was first described by Lang (*2*) and later illustrated by Black (*3*) and Knechtel (*4*).

The large preponderance of orthogonal intersections and the evidence of progressive subdivision, both features of common mud cracks, suggest that the fissures are developing slowly under a progressively increasing stress. The large distance between the fissures indicates that the fissures and the stress producing them extend to considerable depths (*5*). We believe that they are produced by a gradual reduction in volume caused by progressive desiccation of the playa. This is probably the result of a long-term decrease in the supply of surface and ground water from the extensive drainage systems which flow into the Black Rock and Smoke Creek deserts. Rainfall and stream runoff data presented by Antevs (*6*) for the Great Basin indicate that a general period of dry climate commenced between 1910 and 1920, and recent Weather Bureau records show no important reversal in this dry trend.

It is likely that large desiccation fissures will continue to develop on these and other playa lakes of the arid west unless there is a change toward a moister climate (*7*).

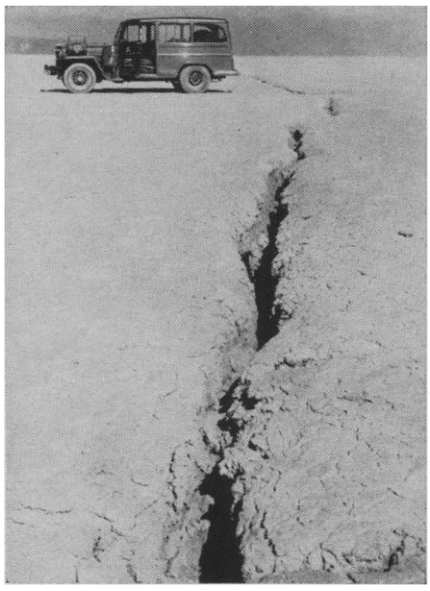

Fig. 2. Open fissure on Black Rock Desert.

RONALD WILLDEN
DON R. MABEY
*U.S. Geological Survey, Menlo Park, California*

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12 December 1960

## Reactivation of Rabies Virus Infection with Adrenocorticotropic Hormones

*Abstract.* A guinea pig inoculated intramuscularly with rabies street virus survived for 5 months, but developed paralysis in the inoculated limb and died of rabies after treatment with adrenocorticotropic hormone.

On 30 November 1959, a group of 34 guinea pigs, being used for a rabies vaccine potency test, was challenged with 0.1 ml of a $1 \times 10^{-2}$ dilution of rabies street virus (salivary gland) given intramuscularly in the right thigh muscle. Twelve of these animals had not been immunized previously with rabies vaccine; they served as controls. The challenge virus was obtained from the submaxillary glands of a spotted skunk, *Spilogale putorius*. The $LD_{50}$ was $1 \times 10^{-4}$ in adult mice and $1 \times 10^{-5.4}$ in infant mice when inoculated intracerebrally (0.015 ml). This virus has not been adapted to brain tissue by intracerebral passage; it was obtained from a naturally infected spotted skunk and subpassed by intramuscular inoculation in spotted skunks (*1*).

One control guinea pig died 23 days after inoculation, and brain material from this animal proved to be negative for rabies virus. Five of the remaining animals developed rabies and died after incubation periods of 37, 37, 40, 47, and 56 days, respectively. The cause of death was proved in each instance to be rabies by subpassage of brain material into mice and by demonstration of Negri bodies in the brain tissue of the infected mice. One other guinea pig exhibited signs of central nervous system stimulation, such as hyperexcitability and muscular hypertonicity, beginning 38 days after inoculation, but recovered.

Twenty weeks after inoculation with rabies virus the six remaining control guinea pigs appeared to be healthy. Blood samples were collected from each of these animals, and the serum was tested, individually, for evidence of neutralizing antibodies against rabies virus. Some degree of protection in a neutralization test in mice was demonstrated by three of the guinea pigs, Nos. 3, 4, and 6. Guinea pigs Nos. 1, 2, and 5 presented no demonstrable neutralizing antibodies to rabies virus.

All of the six control animals were given a course of adrenocorticotropic hormone (H. P. Acthar Gel, Armour and Co., 40 units per cubic centimeter) to determine if this procedure would reactivate rabies virus which might be residing in the host's tissues. Beginning on 20 April 1960 each received 0.25 ml (10 units) of the hormone subcutaneously in the abdominal region at 48-hour intervals.

After three injections of this drug and 7 days after the start of treatment, one guinea pig (No. 2) exhibited weakness in the right hind leg. This was the same limb used for the original introduction of rabies virus. Injections of adrenocorticotropic hormone were continued, and paralysis, of an ascending type, progressed. By the 9th day after the hormone treatment was started, complete paralysis of both hind legs was present. This animal died on 2 May 1960, 154 days after inoculation with rabies virus and 13 days after initiation of the hormone injections.

Portions of the brain, salivary glands, and spinal cord from this animal were excised and prepared for mouse passage. Each tissue was tested in separate groups of eight adult mice. Rabies virus was isolated from the brain, but not the spinal cord or salivary glands. The minimum incubation period in mice was 15 days. Brain smears from inoculated mice were Negri and fluorescent rabies antibody positive.

The remaining five control guinea pigs developed no apparent illness and were killed in the middle of May.

The observation presented here suggests a method whereby rabies virus may be reactivated in the tissues of a host by adrenocorticotropic hormone. Of interest is the fact that the animal that developed rabies after treatment with the hormone had no demonstrable virus-neutralizing antibody in the blood, as determined by the mouse neutralization test.

Other evidence showing that a latent virus infection can be activated by chemical means, in this case adrenalin, has been presented recently by Schmidt

and Rasmussen (*2*). Rabbits, partially immunized against and then challenged with herpes simplex virus, were maintained in an apparently healthy state for periods of 24 to 160 days and then placed upon a course of adrenalin injections: a fatal herpetic encephalitis developed within 2 weeks in six of ten rabbits so treated (*3*).

ORLAND A. SOAVE*
HARALD N. JOHNSON
KOICHI NAKAMURA
*Viral and Rickettsial Disease Laboratory, California State Department of Public Health, Berkeley*

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* Present address: Department of Preventive Medicine, Stanford University School of Medicine, Stanford, Calif.

3 January 1961

## An Advancing Glacier in Canada

*Abstract.* The Commander Glacier, in the Purcell Range of interior British Columbia, advanced 810 ± 50 feet in the 6 years 1954–1960. The advance may be a response to a general cooling trend previously noted elsewhere in the Pacific Northwest.

The glaciers of the Canadian Cordillera have, at least until very recently, been following the world-wide pattern of very rapid shrinkage. The best recent evidence comes from a detailed study of eleven glaciers in the Canadian Rocky Mountains in 1953 (*1*, *2*). All had receded markedly between 1910 and 1950 and were continuing to retreat. In contrast, the Commander Glacier in British Columbia made a significant advance between 1954 and 1960. So far as is known this is the first glacier in interior Canada for which an advance has been noted.

The Commander Glacier lies west of the Canadian Rocky Mountains in the central Purcell Range of British Columbia, 100 miles north of the United States border. A brief glaciological study of this glacier was carried out by one of us in August 1954 (*3*), during the Harvard Mountaineering Club Purcell Range Expedition. The glacier is now primarily a cirque glacier, which occupies a northward-facing valley protected on the south and east by Mount Maye, Mount Commander, Mount Cleaver, and the Guardsmen, all peaks of 10,000 to 11,000 feet elevation. Its greatest length is 2½ miles, and the total area covered by ice is about 4 square miles. Tree-ring dating indicates that the glacier reached its maximum recent extent at about 1820–50, when it extended well into the valley beyond its present cirque basin. The total recession from the 1820–50 terminal moraine to the ice front in 1954 was 6180 ± 100 feet. Most of this recession took place between 1915 and 1947, since photographs taken in 1915 show that the glacier extended nearly to its terminal moraine. The glacier was photographed from the air in 1947 and again in 1953. Little net change in the area covered by ice took place between 1947 and 1954, but in 1954 the ice front was thickened and was quite active.

In August 1960 we remeasured the glacier by using fixed points established in the 1954 survey. The glacier has advanced 810 ± 50 feet since 1954, or an average of about 130 feet per year. Advance has taken place generally along the entire ice front, but is especially pronounced on the west, near the true left (west) lateral moraine. Most of the area underlying the recent advance now appears to be buried beneath at least 100 feet of ice. Blocks of ice were still tumbling from the front of the glacier in 1960, but the activity seemed less pronounced than it was in 1954.

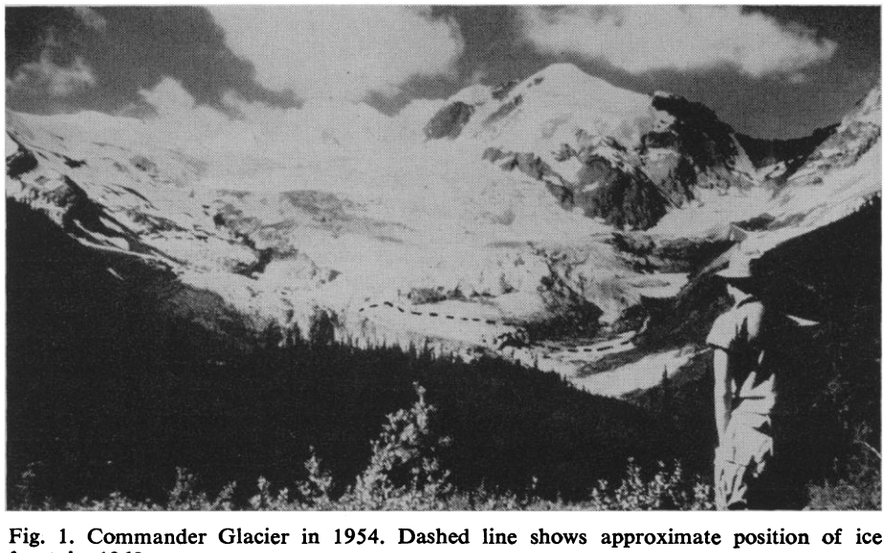

Fig. 1. Commander Glacier in 1954. Dashed line shows approximate position of ice front in 1960.

Glaciers have been generally advancing throughout the Cascade and Olympic mountains of Washington state since 1950 (*4*), although advance may have slowed or ceased since 1958. Increased thickness of glaciers, indicating renewed alimentation, has been noted in the Sierra Nevada in California, in the Wind River Range in Wyoming, and in Glacier National Park in Montana (*5*). Glacier rejuvenation in Washington has been associated with a recent trend toward a cooler and wetter climate in that state (*4*, *6*). This trend is also apparent in Canada. Meteorological data from western Canada show increasing temperature and decreasing precipitation from 1910 to the early 1940's, but a reversal of these trends since about 1945 (*1*, *7*).

The advance of the Commander Glacier probably reflects this climatic change. Although the Commander Glacier is for the present a unique case, other glaciers in the interior ranges of British Columbia seem to show signs of incipient advance. An example is the Hermit Glacier in the Selkirk Range, which has become greatly thickened at the snout since 1950 and appeared to be advancing in 1959 (*8*).

The fragmentary data so far available suggest that glacial advances may be manifested first in interior British Columbia, and only later, if at all, in the more easily accessible glaciers of the Canadian Rockies. However, observations of glaciers throughout the Canadian Cordillera during the next few years should be of special significance.

ROBERT WEST
*Department of Chemistry, University of Wisconsin, Madison*
ARTHUR MAKI
*National Bureau of Standards, Washington, D.C.*

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