in the germinating seeds would result in the hypogeal type of seedling growth, while low auxin and relatively high gibberellic acid concentration would result in the epigeal type of seedling growth. Indeed gibberellic acid is being considered as one of the naturally occurring growth regulators (*7*) since it has been extracted from numerous plants and from various types of plant tissues (*8*).

M. M. LALORAYA
S. A. NAQVI
Department of Botany, University of Allahabad, Allahabad, India

References and Notes

1. F. Skoog and C. Tsui, in *Plant Growth Substances*, F. Skoog, Ed. (Univ. of Wisconsin Press, Madison, 1951), p. 263.
2. M. Wickson and K. V. Thimann, *Physiol. Plantarum* **11**, 62 (1958); K. V. Thimann and M. M. Laloraya, *ibid.* **13**, 165 (1960).
3. G. Kuse, *Botan. Mag. (Tokyo)* **71**, 151 (1958).
4. C. G. Skinner, F. D. Talbert, W. Shive, *Plant Physiol.* **33**, 190 (1958).
5. K. Esau, *Plant Anatomy* (Wiley, New York, 1953), p. 339.
6. P. W. Brain and H. G. Hemming, *Ann. Botany (London)* **22**, 1 (1958).
7. L. J. Audus, *Plant Growth Substances (Plant Science Monographs)* (Leonard Hill, London, ed. 2, 1959), p. 44.
8. We wish to express our grateful thanks to Dr. Kurt Leben of Eli Lilly and Co. for his generous gift of gibberellic acid, to Prof. R. N. Tandon for providing the laboratory facilities, to Dr. S. Ranjan for encouragement and advice, and to Sri R. K. Srivastava and Sri R. M. Pandey for their assistance.

19 December 1960

Giant Desiccation Fissures on the Black Rock and Smoke Creek Deserts, Nevada

Abstract. Open fissures, from 100 to several hundred feet apart, that have produced polygonal patterns on the Black Rock Desert, Nevada, are believed to be giant desiccation cracks resulting from a secular trend toward aridity in the last few decades. Similar features on the Smoke Creek Desert probably have the same origin.

A system of open fissures that divides a part of the playa of the Black Rock Desert into large polygonal blocks was investigated in June 1960 and again October 1960 (*1*). The fissured area was photographed from the air in October 1960 (Fig. 1), when other extensive fissured areas on both the Black Rock and nearby Smoke Creek Deserts were observed.

The system of fissures on the Black Rock Desert that was studied on the ground commences about 20 miles north of Gerlach on the desert road between Gerlach and Summit Lake and extends northward about 5 miles along the northwestern margin of the playa. The system is 1 to 2 miles wide. The fissures range from less than an inch to about 2 feet in width; in general, the narrower fissures are open to the greatest depths (Fig. 2). The fissures appear to be vertical, and some are open to depths in excess of 4 feet. Minor irregularities in the fissure walls prohibit visual estimation of the depth of many of the narrow fissures. The fissures intersect to form an orthogonal network dividing the playa into blocks with edges from 100 to 250 feet long. The widest and oldest fissures, which are generally shallow because of slumpage of material into the opening, form a master network several times as large as the individual rectangular blocks which appear to have developed by progressive subdivision of the larger blocks.

The fissures on the Black Rock Desert are generally confined to the periphery of the playa, and the fissured areas are generally elongated parallel to the edge of the playa. In some fissured areas there is a preferred orientation of master fissures approximately parallel to the edge of the playa (see Fig. 1*A*), but in other areas no preferred orientation was noted.

The fissures are believed to be very recent; some fissures seem to be extending into unfissured ground at the present time (Fig. 1*B*). Faint drainage features in the fissured areas can be identified on 1:20,000-scale aerial photographs of the area northeast of Gerlach taken for the Soil Conservation Service from 10:00 to 10:20 A.M., 21 July 1954, and at 11:06 A.M. on 9 September 1954; however, fissures cannot be identified on these photographs. Although narrow fissures might not have produced wide enough shadows to be resolved on the 1954 photographs, the present fissures were clearly visible from the air between 9:30 and 10:00 A.M., 20 October 1960 (Fig. 1) and could be seen for distances of several

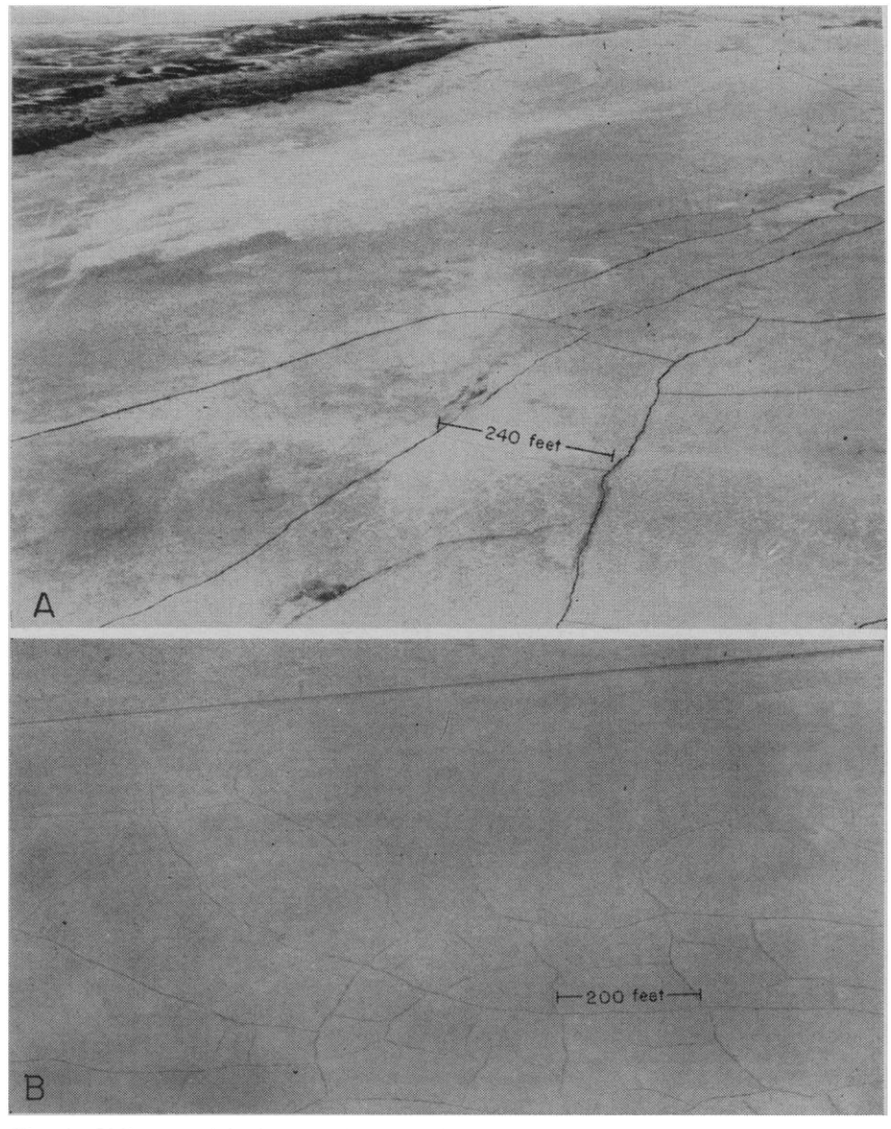

Fig. 1. Oblique aerial photographs of desiccation fissures on the Black Rock Desert, Nevada. Scale is approximate. (*A*) Master fissures approximately parallel to periphery of playa. (*B*) Master fissures extending from polygonal ground into unfissured ground.

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*).

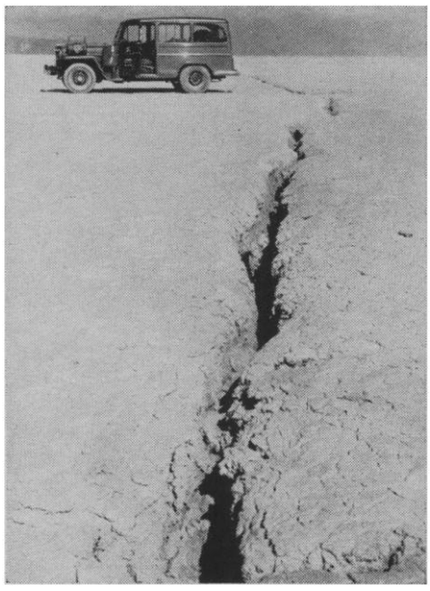

Fig. 2. Open fissure on Black Rock Desert.

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

References and Notes

1. Donald B. Tatlock accompanied the senior author on the second trip, and his assistance is gratefully acknowledged.
2. W. B. Lang, *Science* **98**, 583 (1943).
3. R. F. Black, *Photogram. Eng.* **18**, 123 (1952).
4. M. M. Knechtel, *Bull. Geol. Soc. Am.* **63**, 689 (1952).
5. A. H. Lachenbruch, *U.S. Geol. Survey Profess. Papers No. 400-B* (1960), p. 406.
6. E. Antevs, *Am. Geograph. Soc. Spec. Publ. No. 21* (1938).
7. Publication of this report was authorized by the director of the U.S. Geological Survey.

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 1×10^{-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 1×10^{-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