stances imply. These two, as noted within the past year, are even remarkable:

(A) Excurrent growth of "seed cone" axis—*Sciadopitys verticillata* planted November, 1913, reached a height of about 35 feet, but had some seven feet of the summit broken off by the cyclonic storm of September 21, 1938. The first seed cones were sparingly noted in 1930. By the spring of 1938 various pollinial and seed cones appeared. In this latitude pollination is an early spring event. Later several of the seed cones were found with full-grown proliferate tines of the usual type. While this past spring and summer there appeared a fine seed cone terminating in four tines of the usual foliar type and well-grown as such. No anomaly has been noted in the pollinial cones, which are particularly handsome.

(B) Branched staminate conelets—*Cryptomeria japonica* planted 25 years ago was moved several times before taking on growth. The tree is now 20 feet high. As in the "umbrella pine" pollination occurs in the spring. Several years ago a single excurrently prolonged seed "cone" was seen, and last fall a few clearly branched conelets of the staminate groups were noted. This fall, however, with freer growth of cones, as almost entirely noted toward the base of the tree much better examples of the branched staminate conelets are present. They are finely grown at this writing (December 22); but the ovulate cones are few and not easy to find.

How interpret these recapitulatory growths? For the "umbrella pine," the seed cone axis may regain its original vegetative status, as lost in the leaf and "cone" evolution. The axis is in no sense a true floral axis, but is instead a vegetative branch bearing the bract-seed-scale complex which is floral and inflorescent.

In the *Cryptomeria,* on the contrary, an obverse condition becomes peculiarly instructive. The staminate groups are initially inflorescent in the same order and sense as the excurrent umbrella pine seed cone. The branched pollinial conelets thus become super-inflorescent. Whence they correspond to yet another anomaly especially well seen in the "Chinese black pine," where not infrequently seed cones are to be found intercalated amongst pollinial series.

These striking variations in floral growth require histologic study. That some of them may be brought about under experimental conditions is the near possibility. Finally too, the angiospermous flower appears very old and may have passed the point where the more clearly recessive anomalies appear. That the angiosperm inflorescence often remotely simulates the cones of conifers may be variously interpreted. But the better explanation is remote specialization. Any possible closer relationship between these lines would thus go back near to their defined point of origin in Paleozoic times.

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NATURAL OCCURRENCE OF TULAREMIA IN BEAVER AND ITS TRANSMISSION TO MAN

ON April 10, 1939, the State Game Warden of Wyoming telephoned that he had received a beaver from the northern part of the state which he wished examined to determine cause of death. A nail keg in which the specimen was shipped contained a skinned head wrapped in burlap and a gallon jar containing the viscera. The head was unwrapped, and some of the viscera were touched and turned over with bare hands. Upon discovering a general pyemia the hands were carefully washed in soap and water and rubber gloves used thereafter. Coccoid organisms were found in numerous whitish pin-head-sized or smaller pustules scattered throughout the mesenteries, liver and spleen. Noting an appearance similar to tularemia in ground squirrels, cultures were made from the spleen. The next day in the first transfer cultures, gram-negative rod-like organisms had developed from the coccoid forms, indicating a tentative diagnosis of tularemia.

On April 11, liver pulp was injected intraperitoneally into two young and two adult white rats. The two adult rats died April 14. One young rat died April 15, the other April 16. No lesions of any type were found at autopsy.

On the morning of April 16 the one who had handled the viscera felt dizzy and uncomfortable. This passed in half an hour, but recurred twice during the morning. There was a feeling of lassitude, as if some sort of toxin was in the system, coughed a few times during the morning and each time felt reaction at base of brain similar to severe cold or "flu." Was feeling well by evening. April 20 and 21, felt slightly indisposed. At about 3:30 A.M., April 22, patient awoke feeling very uneasy, a little feverish, but was able to sleep after two hours. Upon arising at 7:30 was light-headed, disinclined to any muscular exertion, eyes sensitive to bright lights. Feeling of uneasiness increased during morning; a noticeable fever by 11:00 A.M. At 1:30 P.M. temperature was 100.5°. Physician diagnosed the case as tularemia. Upon driving an auto, muscular control felt uncertain, and soreness experienced in one shoulder. Rested and felt better by evening. Slept well. April 23rd, a feeling of exhaustion noticeable, sensitive to cold, disinclined to exercise. Temperature sub-normal from 96.7° F. at 8:00 A.M. to 97.9° F. at 8:00 P.M. April 24 at noon temperature 98.5° F, but still slight soreness noted in shoulder muscles and in axillary lymph glands under right arm, and uncertainty of muscular control. Milder recurring attacks, usually at about six-day intervals, took place with diminishing intensity for a period of more than three months.

The case report of J. A., who skinned the beaver, is interesting. At request of Mr. E. he looked over the beaver situation early in the fall of 1938 on West Pass Creek, Sheridan County, Wyoming. Beaver had put in many caches for the winter and prospects looked good for trapping. He worked as a game warden the first three months of 1939. Went to Pass Creek on April 1, and noticed many untouched caches. Found 21 dead beaver both young and old in one and one-half miles of creek, and believed many more died which he did not find; most of them dead for two months. Dead beaver were also found on East Pass Creek, and from an Indian he learned that beaver had died on Lodge Grass Creek, six or eight years ago, from unknown cause. Heard also of dead beaver on Little Horn River and on Goose Creek. On May 20, the beaver trapper wrote: "Was sick in bed with a high fever immediately following the beaver trapping. Then for a long time felt logy and every move cost a great effort. Only now have I begun to feel natural. Had several boils at the time, and what made me think it was the same malady as the beaver was a gathering under the arm pit. But it receded. A number of these beaver had a pus sac in the same place."

At the writer's suggestion a blood sample was taken on July 12 by Dr. Herbert L. Harvey, of Casper, Wyoming, and forwarded to Dr. R. R. Parker, Director, Rocky Mountain Laboratory, Hamilton, Montana. Dr. Parker's report follows:

Antigen-Bacterium tularense	Serum dilutions						
	1 : 10	1 : 20	1 : 40	1 : 80	1 : 160	1 : 320	1 : 640	1 : 1280
B. Tul. ...	4	4	4	4	4	2	..	

4 + = 75 to 100 per cent. agglutination.
2 + = 25 to 50 per cent. agglutination.
Comment: Blood sample from J. A. agglutinates *Bacterium tularense* at a titer of 1 : 640.

The source of infection is uncertain. However, the following facts are interesting. In response to an inquiry on May 31, 1939, J. A. wrote: "There is a scarcity of rabbits on Pass Creek and some of the natives said they found dead ones last summer. The only parasites on these beaver were fleas, a reddish-brown hard-shelled flea which apparently isn't bothered by submersion. They are an annoyance skinning beaver and are not particular what they feed on. The beaver, by their signs, came out of the creeks and had broad trails into the alfalfa meadows where they fed, and if the rabbits were diseased they could have contracted it there." In the fall of the year beaver move about a good deal and this movement might have aided in spreading the infection. It is also possible that the infection was water borne, as suggested by Hammersland and Joneschild.[1] The fact that practically no beaver escaped the infection indicates that water may have been partly responsible for the character of the outbreak.

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[1] *Jour. Am. Vet. Med. Asn.*, January, 1940.

SYSTEMATIC HISTOPLASMOSIS IN THE UNITED STATES

BETWEEN the years 1906 and 1908 Darling[1] in Panama reported at autopsy three cases of a systemic parasitic infection, the causative agent of which he called *Histoplasma capsulatum*. At first thought to be a tropical disease protozoan in nature and closely related to kala-azar, it has since been proved by de Monbreun[2] to be a fungus infection. Subsequently five[1a] cases have been reported from widely separated areas in the United States.

We have had the opportunity of studying a case of histoplasmosis occurring in a colored male adult who was admitted to St. Philip's Hospital in March, 1939. This is the second known case in which diagnosis was made before death. A detailed report of this case, including complete autopsy findings, will be published later.

Due to the fatal termination of the majority of these cases before diagnosis could be established, much concerning the manner of infection, diagnosis and treatment of the disease is still obscure. We believe that histoplasmosis is much more common than the number of published cases would lead one to believe. It is suggested that the findings of an anemia with leukopenia in a weakened, emaciated individual running a septic temperature should lead one to search the blood smears carefully for the parasite-laden monocytes. The enormous number of parasites seen in bone marrow preparations would indicate that sternal puncture studies are diagnostic.

Diagnosis may also be made by culture of the blood on dextrose or blood agar slants. Other media suitable for the cultivation of fungi would probably be satisfactory as the parasite grows readily. Intraperitoneal inoculation of the growth from blood agar slants into guinea pigs produces a typical infection. At autopsy the parasite can be found in abundance in the infected organs.

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SCIENTIFIC PUBLICATIONS NEEDED IN FRANCE

ALL who can imagine or may remember the isolation

[1] S. T. Darling, *Arch. Int. Med.*, 2: 107, 1908.

[1a] Two additional cases in which diagnosis was made at autopsy have been since added to the literature.

[2] W. A. de Monbreun, *Amer. Jour. Trop. Med.*, 14: 127, 1934.