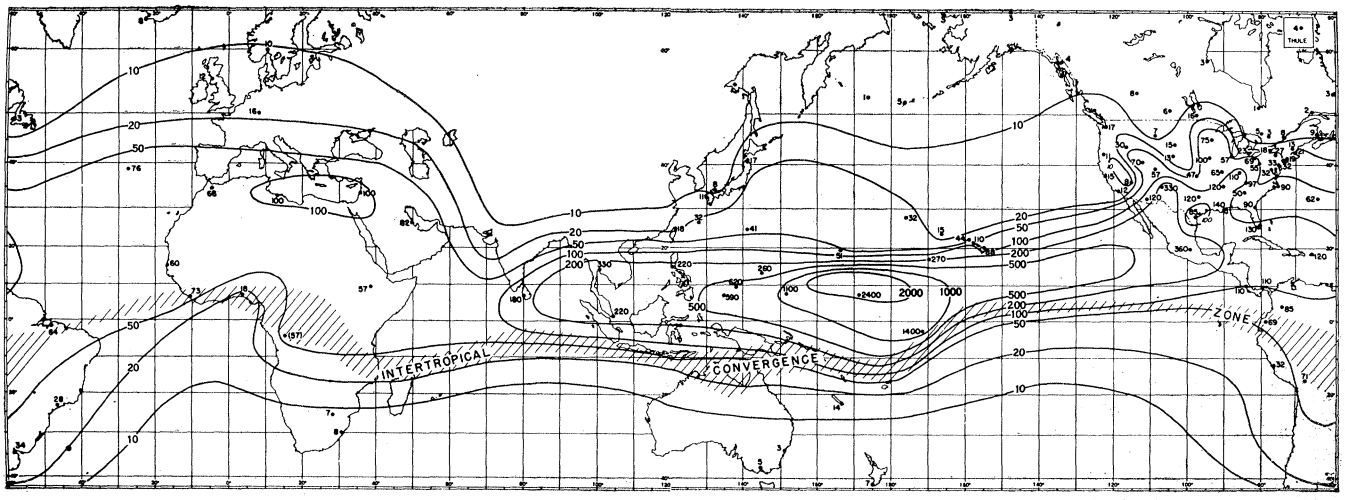

Fig. 5. Total radioactive fallout from the Bravo cloud in the period from 2 to 35 days after detonation, in millicuries per 100 square miles. Hatching indicates approximate March position of the Intertropical Convergence Zone, the belt of low pressure that tends to separate Northern and Southern Hemisphere air near the surface of the earth.

tained at the Southern Hemisphere stations especially during the Mike shot, are immediately evident from the fallout maps. The northern part of the Northern Hemisphere, however, received equally small depositions. The distribution of fallout for the Pacific stations appears to be consistent with the features of the meteorology described, although the branching of the cloud south of Japan in the Mike pattern is based only on scanty observational evidence.

It is apparent that radioactive debris produced by nuclear explosions does not possess all the desired attributes of a tracer for studying global circulations. Information concerning the magnitude and distribution of the radioactivity that remains airborne after the initial fallout is not available. The debris, being particulate, is washed out of the atmosphere and cannot be strictly treated as a conservative property. Thus, for example, the depositions in the Southern Hemisphere may have been low because most of the debris was rained out as it passed southward through the Intertropical Convergence Zone. In addition, the most effective sampling program for the debris provides only the crudest measure of the fallout. Yet, despite these limitations, it appears that the meteorologist can obtain useful information by operating such a network of gummed films during nuclear test periods. Although it is not proposed that special nuclear tests be undertaken for meteorological purposes, it seems reasonable to expect even greater value from future tests using an expanded network and having detonations at other locations and times.

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A. O. Weese, Ecologist

Asa Orrin Weese was born of Canadian parentage in Hutchinson, Minnesota, 7 November 1885, and died in Norman, Oklahoma, 20 November 1955. After graduating from the University of Minnesota in 1909, he taught for 2 years in an academy and, from 1911 to 1922, was a member of the department of biology at the University of New Mexico. He received the degrees of master of arts (1914) and doctor of philosophy (1922) at the University of Illinois, working in ecology under V. E. Shelford. His special interest in community ecology and succession began then and continued throughout his entire career. He was professor of biology at James Millikan University for 2 years and then came in 1924 to the University of Oklahoma as a professor of zoology.

As a student of developing biotic communities, Weese was, of course, interested in all of their components, both plant and animal, but his personal studies turned especially to the insects of the grasslands. In New Mexico he studied also the horned lizard and, at the seashore, annelids and sea urchins, all in relation to the communities to which they belonged. The summers for him were always a time for study, and he took part in the work of various summer laboratories. He was in charge of the course in marine ecology at the Friday Harbor Laboratory in Puget Sound in 1925 and 1929. He and I were among the founders of the Rocky Mountain Biological Laboratory at Gothic, near Crested Butte, Colorado, in 1928, and he succeeded to its presidency in 1938. For many years he was responsible for the instruction in ecology at that laboratory.

He was the secretary of the Ecological Society for 10 years and its president in 1931. He served officially in various other organizations, including the Ecologist's Union, the National Research Council's Committee on Grasslands, Grasslands Research Foundation, and the Oklahoma Biological Survey. In all of these he held important offices and helped shape policies. He played a significant part in the activities of the Oklahoma Academy of

Science, and he was a member of various scientific, academic, and honorary societies. He served on both state and national committees that were concerned with ecology and its applications. As ecologist with the Oklahoma Biological Survey, he participated with his students in many field excursions, and his own published output of scientific contributions was augmented by their work.

Dr. Weese was the recipient of many distinctions within the university, such as the deanship of the graduate college for a period, the chairmanship of the Committee on Faculty Research for some years, and, during the last years of his life, the David Ross Boyd professorship of zoology. He held elective offices in such local organizations as Sigma Xi, the American Association of University Professors, and others.

His most significant trait was his wide and profound knowledge; his reading and study were extensive and not limited to his field of research. He was an able invertebrate zoologist, he was interested in the quantitative aspects of genetics as well as bioecology, and he developed a course in quantitative biology. He was able to penetrate obscure problems and to give advice and illumination on matters both inside and outside his own fields. He contributed greatly to the development of the departmental library at the university and was responsible for many of its acquisitions. Since his death, this library has been named in his honor.

A kindly, interested, and cooperative scholar, he left a moving and permanent impression on all with whom he came in contact. He bore his own burdens simply and fully shared those of his colleagues and his community, which, in a growing university, were often not light. Like so many whose accomplishments remain long to tell their stories, he will never be replaced, and he will live on in the deeds of his students and associates.

A. Richards

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H. H. De Jong, Experimental Neurophysiologist

The history of experimental catatonia is the history of the collaboration by investigators from different countries, united in the love of science and in the enjoyment of scientific research, as well as by the synthesis of data from physiology, biology, and clinical experience—a synthesis indispensable to the future progress of neuropsychiatry.

H. Holland De Jong was born in 1895 in Sneeck, Holland. In 1928 at a meeting of the Société de Neurologie de Paris, he presented a paper on the treatment of tremors by bulbocapnine, an alkaloid that he regarded as an "antagonist of tremors." In the course of plethysmographic investigations of many patients, De Jong had discovered a "vascular rigidity" in catatonics that he failed to find in other patients. He thought of the possibility of the experimental reproduction of this illness, and, on the advice of Magnus, became interested in bulbocapnine. But being unacquainted with clinical catatonia, he had not begun this work.

At that time, I had, with others, undertaken a systematic study of Kahlbaum's catatonia, with a physiological exploration of this illness by new processes and a comparison of the physiological data with precise clinical data based on a study of many patients. I had reached the conclusion that catatonia constitutes a psychomotor syndrome of toxic origin. I approached De Jong, and we agreed to collaborate.

Our first joint experiments were undertaken in Paris in the laboratory of Claude. We compared in detail the symptoms that were produced by bulbocapnine in the cat with the symptoms of human catatonia. We found in the animal, as in human beings, the various manifestations of catalepsy: active and passive negativism, barriers, catatonic hyperkinesis, and neurovegetative disturbances, including salivation and respiratory disturbances. The same parallelism was also found in electromyograhic curves and in disturbances in chronaxie, as in the experiments of Bourguignon and De Jong, and in man by Claude, Bourguignon, and Baruk.

Our next experiments were in Amsterdam in the laboratory of Brouwer. We were able to study in detail the action of bulbocapnine for a wide range of doses and for the entire vertebrate series. In this way, we formulated laws describing the stages that follow increasing doses, from sleep to catalepsy, from catalepsy to negativism, from negativism to hyperkinesis, and finally, with still stronger doses, to epilepsy and the rigidity of decerebration. In similar fashion, the study of the animal series, starting with fish through batrachians, reptiles, and birds to mammals, permitted us to verify the role of the cortex. We described this work in our joint book (1930), which brought us both the Herpin prize of the Academy of Medicine.

De Jong and I were then separated. I remained in Paris, and De Jong went to the United States to teaching posts at Duke University and Johns Hopkins University, where he discovered experimental hormonal catatonia (adrenaline, acetylcholine, and so on), surgical catatonia, mescalinic catatonia, and so on. During this period, I discovered, in Paris, experimental catatonia that was induced by the toxin of *Escherichia coli* (1933), biliary experimental catatonia (with Camus, 1934), and other types of experimental catatonia, induced by cerebral edema, ACTH, chlorpromazine, and so on. During his last years, De Jong investigated the role of intestinal and hepatic factors. His research on catatonia by obliteration of the intestinal lumen and by ligature of the biliary ducts must have led him to consider again conceptions of the role of hepatointestinal factors in catatonia and schizophrenia, conceptions that, after the work of Buscaino and our work on this subject had converged toward our present conclusions, have had important therapeutic results.

Recently, De Jong and I began to think about collaborating again in Paris. Alas, his premature death on 16 February 1956, at the age of 61, in Osawatomie, Kansas, where he was director of research and education at Kansas State Hospital, prevented the realization of this plan. This great misfortune abruptly ended a life dedicated to science and deprived medicine of a scientist of exceptional intelligence and perspicacity, a scientist whose goal it was to discover the causes of mental illness.

H. Baruk

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