The organs most profoundly affected were the liver, lungs, testes and adrenals. Without exception, the animals developed liver lesions which varied from a marked fatty change to multi-lobular cirrhosis. Frank cirrhosis occurred in 20 per cent. of rats and affected almost exclusively the left lobes, while the right lobes underwent marked hypertrophy and were fatty. This liver damage in the rat was not without some interest in view of the high incidence of liver cirrhosis in the Bantu.

Lung lesions occurred in the majority of rats and they were chiefly lung abscesses and bronchiectasis.

The testes in all male rats were damaged to a greater or lesser extent. They were soft and, on section, oozed fluid. Giant cells were commonly found, and in one testis associated with complete tubular degeneration a nodule of interstitial cells had developed and, on cytological examination, appeared to be malignant. The prostate and seminal vesicles associated with the atrophic testes were reduced in size. It is worthy of note that the majority of Bantu patients suffering from cirrhosis of the liver had lost their sexual appetite.

The suprarenals presented a variable picture. In the majority of rats, the adrenals were enormously hypertrophied and in five instances adrenal hemorrhage was obviously the cause of death. Hemorrhage occurred only in the left adrenal. In the remaining animals, the adrenals were small and of a gray color.

The thyroid gland was atrophic while the parathyroids were invariably enlarged. The parotid gland and the pancreas did not escape injury. The parotid was always damaged, while the pancreas was affected in only 56 per cent. of cases. In the parotid there was no evidence of metaplasia, but the whole architecture of the gland was profoundly disturbed. The nuclei were extremely enlarged and showed cell degrees of anaplasia. Frequently a single cell contained 6 to 8 giant nuclei, each measuring in some instances as much as 40 microns. The lesion in the pancreas was different from that in the parotid. The granules disappeared from the acinar cells, and those later lost their spheroidal appearance and became arranged in the form of small dilated ducts. These aggregations of ducts were scattered throughout the pancreas and they could be seen with the naked eye as rather white opaque spots no larger than a pin's head.

Enlargement of the heart was found in those rats whose livers were severely damaged, and in two instances the endocardium of the enormously enlarged ventricles was found to be calcified throughout its extent. Cirrhosis of the liver was less common in females and dead embryos were not infrequently found in the uterus. It might be mentioned at this stage that although the skull was very thick, the bone cut easily.

It is thus seen that during the first nine weeks the feeding of corn pap alone leads to an arrest of growth, there being a gain of only 7 to 10 grams. While the addition of fermented milk definitely speeds up growth, it invariably causes the animal to develop extensive lesions, including a damaged liver. In young rats (40 to 50 gm) the livers begin to show fatty change at the end of 20 days, but these do not become very marked until the end of 150 days.

It was impossible to attribute the lesions in the rat to the absence of any specific vitamin. It is true that the testicular damage and the dead embryos in the uterus could be regarded as a manifestation of vitamin E deficiency, but it is very likely, too, that vitamin E deficiency would also be regarded as an expression of liver damage, since in dogs with biliary fistulae Brinkhous and Warner[10] have described the presence not only of muscular dystrophy simulating vitamin E deficiency, but also lesions which could be attributed to a deficiency of vitamins D and K. A damaged liver in the rat also apparently upsets the metabolism of fat-soluble vitamins, although there was no evidence of vitamin K deficiency. The occurrence of a malignant hyperplasia of the interstitial cells of the testes in the rat mentioned above is also of interest, since these tumors can be produced in mice by oestrogen.[11] It seems very likely that damage to the liver affects, amongst other things, not only the metabolism of some of the fat-soluble vitamins but also the metabolism of the steroid hormones. The work is proceeding to discover the factor or factors responsible for the various lesions in rats mentioned.

This simple experiment reveals the widespread lesions that may result from feeding the common Bantu diet to rats; it throws some light, too, on the problem of the high incidence of liver, lung and heart disease in the black people of South Africa; it also indicates that great caution must be exercised in attributing to a racial factor any differences in the incidence of disease amongst the black and white people.

OBITUARY

JAMES OTIS BEASLEY

THE South has lost one of its most valuable agricultural research workers, the nation one of its most promising young cytogeneticists in the passing of James Otis Beasley, who died on September 12, 1943, following wounds received in action in Italy.

Dr. Beasley was born at Wells, Texas, on September 7, 1909. He was educated in the public schools of Texas, graduating from Lufkin High School in

[10] K. M. Brinkhous and E. D. Warner, *Am. Jour. Path.*, 17: 81, 1941.

[11] C. K. Hooker and C. A. Pfeiffer, *Cancer Research*, 2: 759, 1942.

1928, from the Agricultural and Mechanical College of Texas in 1932 and receiving the master's degree from the same institution in 1934. During the next two years he served as an assistant in the Texas Agricultural Experiment Station, engaged in studies upon the morphology of the cotton seed. In 1936 he entered Harvard University as a graduate student, completing the requirements for the doctorate in 1939. During parts of 1938 and 1939 he was employed by the U. S. Department of Agriculture in genetic investigations of cotton at Raleigh, N. C. Since 1939, until he entered military service, he was agronomist and cytogeneticist on the staff of the Texas Agricultural Experiment Station. As a First Lieutenant in the Infantry Reserve he was called into active service on March 5, 1942. Feeling that he might be more useful in the Chemical Warfare Service he was, at his own request, transferred to that branch. He landed in Oran in May, 1943, took part in front-line action in the Sicilian campaign and again in the Salerno landing, where he was fatally wounded while leading a small detachment in an attack upon an enemy-held farm house in front of the American lines.

Dr. Beasley is survived by his wife, Dr. Elizabeth Wagner Beasley of Carroll, Ohio, whom he married in 1940, and by his son, John Wagner Beasley, born in 1942.

Since Beasley grew up in a rural community, in a state where cotton is the all-important crop, and since he was by nature a student, it is not surprising that he should have turned his energies at an early age to the study of cotton. As an undergraduate he competed for, and won, a traveling scholarship for the study of cotton, and during the summer of 1932 he visited the principal cotton-growing areas of the United States and the important cotton merchandising and manufacturing centers of Europe. When he entered Harvard as a graduate student, under Dr. E. M. East, he began almost immediately a study of the genetics and cytology of Gossypium species. In spite of the difficulties of growing cotton species in the vicinity of Boston, even in the greenhouse, he succeeded in making considerable progress in a relatively short time.

In his research Beasley showed marked originality not only in devising new methods of attack, but also in adapting the techniques of others to his own problems. By the use of mixed pollinations to prevent the bolls from shedding prematurely and by employing embryo culture techniques, he succeeded in producing species-hybrids not obtainable by ordinary methods. He was one of the first to utilize colchicine extensively in doubling the chromosome number of sterile hybrids to produce fertile allopolyploids. These he investigated cytologically to contribute to an understanding of the origin of cultivated cottons, a subject to which his more important published papers are devoted. He also utilized such hybrids in an attempt to transfer useful genes from wild 13-chromosome species to the 26-chromosome American cultivated cottons. His success at transforming sterile diploids to fertile tetraploids led him to suggest that hybrid vigor in such crops as maize might be perpetuated indefinitely by producing inversions and translocations with x-rays to the point where the F_1 hybrid of two strains should be sterile, then doubling the chromosome number to produce a fertile true-breeding tetraploid hybrid exhibiting the heterosis of the diploid. The proposal, which has never been adequately tested, offers important theoretical possibilities in plant breeding.

Dr. Beasley was an industrious, capable and conscientious scientist motivated by a friendly spirit of cooperation, an intelligent curiosity and an eagerness to contribute to the improvement of cotton and to Southern agriculture in general. For such a task he was superbly fitted both by temperament and training, for he combined to an exceptional degree an appreciation and understanding of the theoretical principles of genetics, with an ability to apply those principles to practical problems of plant improvement. It is difficult to imagine a man of Beasley's type, thoroughly peaceful, scholarly, quiet and reserved, taking kindly to the art of warfare. Yet he was successful as an officer; popular with his men and respected by the officers above him. He has been awarded the Purple Heart posthumously for "military merit" and has been recommended for an additional citation for "continuous devotion to duty." One can not believe, however, that the supreme sacrifice which he was called upon to make as a soldier can compare in effectiveness with the contributions to scientific progress and to human welfare which he almost certainly would have made had his peacetime pursuits been permitted to reach fruition.

P. C. MANGELSDORF

BOTANICAL MUSEUM,
HARVARD UNIVERSITY

DEATHS AND MEMORIALS

DR. DAYTON STONER, since 1932 state zoologist of New York, died on May 8 at the age of sixty years.

STUART BALLANTINE, since 1935 president of the Ballantine Laboratories at Boonton, N. J., known for his work in the field of radio engineering, died on May 7 at the age of forty-six years.

DR. WILLIAM SPENCER CARTER, from 1922 to 1934 dean of the medical faculty of the University of Texas, formerly associate director of the medical sciences of