DIET AND DISEASE IN THE BANTU

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THE incidence of disease in the Bantu (South African Negro) has long been known to be very different from that usually described for the Whites or Europeans in South Africa. Cirrhosis of the liver is found in four out of five autopsies performed on male Bantu at the Johannesburg General Hospital.[1] Gallstones and diabetes are rare diseases,[2, 3] while peptic ulcer in the rural Bantu population is almost unknown, but in a series of over 12,000 autopsies it was found to be seven times higher in the whites than in the urban Bantu.[4] Endocrine disorders affecting the pituitary and the thyroid are extremely rare, although colloid goiters are endemic in some areas of this country. Kidney stones in the Bantu are pathological curiosities.[5] Tuberculosis is extremely widespread and almost every second case admitted to hospital dies. Enlarged prostates are infrequent events, but sterility is not uncommon. The most remarkable discrepancy, however, is in the incidence of cancer. On the Witwatersrand Gold Mines, amongst the Bantu recruited from the native territories and Portuguese East Africa, primary carcinoma of the liver, a rare disease in Europeans, accounted for 90.5 per cent. of all cases of cancer. In the urban Bantu population, however, primary carcinoma of the liver was found almost exclusively in young male adults among whom it accounted for 31.9 per cent. of carcinoma of all organs, whereas cancer of the stomach was relatively common amongst males, especially after middle age. In the female, carcinoma of the genital system was responsible for almost 75 per cent. of cancers affecting all organs. Cancer of the stomach is unknown, but urinary bladder cancer is occasionally found.[6]

While it would be easy to attribute this peculiar incidence of disease in the Bantu to a racial factor, it seemed probable that the economic backwardness of the Bantu people might be an equally important factor which could not be overlooked. In 1936, 55 per cent. of wage-earners of all races, of which the Bantu constituted 90 per cent., earned between £9 and £36 (between $36 and $150) per annum. Only 4 per cent. of wage-earners had an income of $600 to $1,000 per annum.[7] In view of this meager wage, the Bantu people are constrained to live almost exclusively on the cheapest available carbohydrate—in this case—corn (maize or mealie meal). This food is partly cooked, and in some areas it is supplemented by fermented cow's milk. Meat is a luxury and is eaten only on ceremonial occasions or when their cattle or sheep die from disease or starvation.[8] It is a natural consequence that deficiency diseases of all kinds, including pellagra, are widespread amongst the black people in South Africa.

In order to ascertain with greater precision the effect of diet on the incidence of disease in the Bantu, it was felt to be of fundamental importance to discover the reactions of laboratory animals fed on corn pap and sour milk. For this purpose, 125 albino rats of the Lister strain and weighing between 40 and 55 gms were fed on liberal quantities of corn pap and fermented milk for a period of two years.

Careful records of the weights of the control rats, fed on a mixed diet, and of the experimental rats showed that at the end of three weeks the control animals had doubled their weights, whereas the experimental rats had not doubled their weights even at the end of seven weeks.[9] At the end of five months, apart from the retardation in growth, there were no gross external abnormalities, except that the coats of some animals assumed a pale rusty color. After 15 months, many rats had lost all their hair, especially in the lower abdomen and over the thighs and back. The majority developed a brown incrustation over the whiskers, around the eyes and the snout. Rats on a normal diet show by slit-lamp microscopy a clear cornea with glistening epithelium. Those on an abnormal diet have pathological lesions in the cornea which take the form of irregular blistered epithelium with evidence of scarring. The transparency of the cornea is further reduced by the ingrowth of capillaries which form a network in the corneal substance. Dental lesions were of frequent occurrence; this expressed itself chiefly in the form of irregular growth of the incisors, some of which grew either into the lower jaw or the top jaw. In one instance only did the incisors lose their orange-red color. In one rat, a hemorrhage occurred into the anterior chamber of the eye. In a few instances, the females gave birth to five or six young, but these were never reared.

[1] A. Sutherland Strachan, personal communication.
[2] C. F. Beyers, *Jour. Med. Asn. S. Afr.*, 1: 606, 1927.
[3] Joseph Gillman, *Am. Jour. Phys. Anthrop.*, 21: 131, 1936.
[4] P. C. Eagle and Joseph Gillman, *S. Afr. Jour. Med. Sci.*, 3: 1, 1938.
[5] V. Vermooten, personal communication.
[6] C. Berman, *S. Afr. Jour. Med. Sci.*, 1: 12, 1937; *ibid.*, 5: 54 and 92, 1940; *ibid.*, 6: 145, 1941.
[7] John Burger, "Black Man's Burden" (Gollancz, London), 1943.
[8] R. Smit, personal communication.
[9] C. Gilbert, J. Gillman, J. Mandelstan, T. Gillman and L. Golberg, *S. Afr. Jour. Med. Sci.*, 1943.

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.