vided in the estimates for the fiscal year 1945–46 for the following grants which are administered by the society: For scientific investigations, £14,000; for scientific publication, £7,000; for scientific congresses, £1,600.

A CORRESPONDENT writes that "the Belgian Government Information Center reports that so far as they know the Congo Museum at Tervueren is in good condition. The Germans have spared this museum, and the same staff is still there."

THE daily press reports that to offset the continued isolation of the universities at Amsterdam, Delft, Groningen, Leyden and Utrecht—all of which are still in Nazi-occupied territory—the Netherlands Ministry of Education has arranged for temporary academic centers in the liberated area.

The Journal of Scientific and Industrial Research, Delhi, India, states that the Government of Bengal has decided to set up a Provincial Statistical Bureau. Each department of the government will be instructed to keep the bureau informed of its activities in statistical matters and to seek the guidance of the bureau when it proposes to introduce new schemes or methods. The function of the bureau will be primarily advisory in character. It will impart technical advice regarding the collection of primary statistics, classification, tabulation and analysis of statistical material and the form of publication of statistical data.

DISCUSSION

AMPLE EXERCISE AND A MINIMUM OF FOOD AS MEASURES FOR CANCER PREVENTION?

As measures for the prevention of human cancer a recent paper by Potter[1] advocated ample exercise, a minimum of food and "proper medical care so that any chronic irritations are eliminated." At the present time there is a paucity of information on the influence of dietary restriction upon the prevention of cancer in human beings. In the absence of data on man we must rely on the results of animal experiments for information on dietary means for delaying the onset of cancer, its partial or complete prevention, and then attempt to evaluate the possible effects of applying such procedures to the human population. Our present concept must, therefore, be largely influenced by this type of information without knowing to what extent it may be applicable to the prevention of cancer in another species, man.

Data from several sources are summarized in Table 1. This table sets forth the results of several dietary regimens that have been effective in decreasing the incidence of spontaneous mammary cancer in susceptible strains of mice and includes other effects which have been noted in the animals subjected to these regimens. Mammary tumorogenesis in mice as influenced by diet has been more extensively studied than any other tumor of spontaneous origin, and for that reason it seems appropriate to make it the basis for this discussion. It will be observed from Table 1 that a variety of nutritional regimens may delay, partially or completely inhibit the development of spontaneous mammary cancer in mice.

A primary effect of the various regimens listed in Table 1 was on body weight. Weanling mice placed on calorie restricted diets failed to gain weight. Adult animals lost from 10 to 45 per cent. in body weight, usually during the first few weeks of the experiment, after which time the animals were able to maintain their weight at the lower level. On the cystine deficient diet young mice were unable to gain in weight, while on the lysine deficiency the diet contained enough lysine to permit a very slow increase in body weight of the young mice, amounting to approximately 20 per cent. of the initial weight of the animals.

The effect on estrus and mammary gland development was equally striking in those experiments in which estrous cycles and mammary glands were studied. Estrus was absent in the weanling mice that failed to increase in body weight and occurred irregularly in the lysine deficient group in which there was some body weight gain. In the calorie restricted and deficient weanling animals in which estrus was absent or occurred occasionally, there was also complete or partial inhibition of mammary gland development. In the parous animals estrus was probably absent, and the mammary glands atrophied.

The most effective regimens in decreasing mammary cancer were those instituted at an early age on weanling mice and continued until death, although mice that were restricted in calories after raising one or two litters also showed a definite decrease in tumor incidence, loss in body weight and atrophy of the mammary glands.

Inhibition of estrus has been observed in rats[2] after a weight loss of only 15 per cent. The loss in weight of the mice as given (Table 1, 5, 6) with the concomitant inhibition of estrus suggests that estrogenic hormone production was one factor that may have been affected. The administration of diethylstilbestrol (Table 1, 9, 6), to a group of cystine deficient mice induced continuous estrus and stimulated development of the mammary glands. The incidence of mammary tumors was increased from 0 to 45 per cent., or to only approximately half that found in the normal controls.

[1] V. R. Potter, SCIENCE, 101: 105, 1945.

[2] M. G. Mulinos, L. Pomerantz, J. Smelser and R. Kurzrok, *Proc. Soc. Exp. Biol. and Med.*, 40: 79, 1939.

The decreased incidence or inhibition of spontaneous mammary tumors in mice by the various dietary regimens listed in Table 1 suggest that the influence of diet on mammary carcinogenesis is not a direct effect of some specific dietary constituents,[3] but is more likely due to the potentialities a dietary procedure has in altering estrogenic and probably other hormonal secretions over a considerable portion of the animal's life span. Obviously, any dietary regimen that in some way inhibits estrus or the development of the mammary gland would be expected to inhibit or delay spontaneous mammary carcinogenesis in mice.

The question arises: To what extent would the dietary regimens found effective in inhibiting mouse mammary cancer be effective in delaying or preventing the same disease in man? Even though they were unduly optimistic to conclude from any experimental evidence available at present that "eating no more than we need" would be a useful guide in decreasing or inhibiting human cancer.

It is further emphasized that the effective dietary regimens discussed herein were applied to a single type of tumor that develops spontaneously only in the females of susceptible strains of mice. That broad generalizations on the effect of nutrition on carcinogenesis should not be attempted, even in animals, from results on a single tumor type are illustrated by results reported from the McArdle Laboratory,[4] where it was observed that well-nourished rats as well as rats consuming an optimum amount of protein (18 per cent. casein) were more resistant to the development of liver tumors following the ingestion

TABLE 1

DIETARY MEASURES FOUND EFFECTIVE IN DECREASING THE INCIDENCE OF SPONTANEOUS MAMMARY CANCER AND OTHER OBSERVED EFFECTS SUCH REGIMENS HAVE ON FEMALE MICE

Strain	Age months		Breeding	Type of regimen	Amount of restriction	Body weight change per cent.		Amount of mammary gland development	Effect on		
	Started	Ended				Control Per cent.	Exp. Per cent.		Estrus	Tumor incidence per cent.	
										Control	Exp.
dba[2]	2.5	20	Nulliparous	Calorie restriction	1/2	+50	−10	.....	.....	38	0
dba[3]	5.5	18	"	Underfeeding	1/3–1/2	+30	−<25	.....	.....	40	2
dba[3]	9.0	24	27 per cent. nulliparous 73 per cent. parous	"	1/3–1/2	+ 7	−>30	.....	.....	30	7
C3H[4]	1	18	Nulliparous	Calorie restriction	1/3	+170	+10	.....	.....	67	0
C3H[5, 6]	1	22	"	" "	1/2	+106	− 8	infantile	complete absence	100	12.5
C3H[5, 6]	4	22	Parous	" "	1/2	+33	−44	atrophy	.....	100	18.2
C3H[7, 6]	1	28	Nulliparous	Lysine deficient	.....	+100	+>20	infantile to moderate	occasional	97.6	25.0
C3H[8, 6]	1	22	"	Cystine deficient	.....	+100	0	infantile	complete absence?	97.4	0
C3H[9, 6]	1	17	"	Cystine deficient + stilbestrol	.....	+100	0	extensive	continuous	92.0	45.0

[2] A. Tannenbaum, *Cancer Res.*, 2: 460, 1942.
[3] *Idem, Am. Jour. Cancer*, 38: 335, 1940.
[4] M. B. Visscher, Z. B. Ball, R. H. Barnes and I. Sivertson, *Surgery*, 11: 48, 1942.
[5] F. R. White, J. White, G. B. Mider, M. G. Kelly and W. H. Heston, *Jour. Nat. Cancer Inst.*, 5: 43, 1944.
[6] F. R. White, *Jour. Nat. Cancer Inst.*, 5: 49, 1944.
[7] F. R. White and J. White, *Jour. Nat. Cancer Inst.*, 5: 41, 1944.
[8] J. White and H. B. Andervont, *Jour. Nat. Cancer Inst.*, 3: 449, 1943.
[9] F. R. White and J. White, *Jour. Nat. Cancer Inst.*, 4: 413, 1943.

partially efficacious, would not some of the other conditions likely to develop, such as failure of normal growth, inhibition of normal mammary gland development or the possible cessation of reproductive capacity completely negate any attempt to restrict the human diet to the extent apparently necessary to obtain prevention of cancer in the mouse? On the other hand, if a relatively small restriction of the food intake would be effective either in delaying or partially decreasing the incidence of cancer without also affecting other body functions it might be of great practical value. Such studies have not yet been carried out with experimental animals, and it seems

[3] H. P. Morris, Symposium No. 28, The American Association for the Advancement of Science, pp. 140, 1945.

of paradimethylaminoazobenzene than were rats consuming this carcinogenic hydrocarbon in nutritionally poor diets such as those containing brown rice and carrots or suboptimum amounts of protein.

An effort as mentioned by Potter[1] has been made to correlate the diet and development of cancer in man from statistics on the relation of weight and cancer incidence as obtained from life insurance records. These statistics have been brought together by Tannenbaum.[5] In general this material shows that among people who were overweight at the time of issuance of the insurance policy there was a somewhat greater

[4] J. A. Miller, M. S. Miner, M. D. Rusch and C. A. Baumann, *Cancer Research*, 1: 699, 1941.
[5] A. Tannenbaum, *Arch. Path.*, 30: 509, 1940.

proportion of deaths due to cancer than among persons not overweight at the time the policy was issued. In one insurance report the relationship between overweight and cancer was more marked for cancer of the female genital organs and intestines than for cancer of the breast, stomach, liver or gall bladder. Other statistics did not show any relationship between weight and cancer mortality. The most favorable statistics did not support this relationship very strongly and even if further work substantiates a relationship between overweight and death from cancer, one can not be certain that the relation between cancer and weight is direct; there may be several factors little understood that affect the development of obesity in an individual.

Furthermore, since these weight records were made many years prior to death from cancer it seems undesirable to place too much emphasis on them. Tannenbaum's suggestion[5] of the need of securing more data on the relationship of weight to death from cancer is a good one. Many factors, including composition and amount of diet, should be studied before drawing final conclusions or making specific recommendations.

The role of exercise upon cancer development has not been extensively investigated experimentally. Kline and Rusch[6] have recently reported the results of forced exercise on a transplanted sarcoma in mice. No difference was observed in the number of takes in exercised and non-exercised mice. It seems that if there is any phase of a tumor transplantation experiment which might be classified in the "critical period,"[6] as used by Potter,[1] it would be the period before the transferred tumor cells began to grow in their new host, yet it was only during the growth period of the transplanted sarcoma that forced exercise was observed to slow tumor growth. This would certainly come under the "period of progression" as defined by the above-mentioned authors. It is entirely possible that forced exercise may also play some role in carcinogenesis during this so-called critical period, but the evidence cited seems inadequate. It may be more nearly correct to conclude from the data that forced exercise decreases tumor growth instead of genesis of tumor. Even here such an interpretation is complicated, as Potter also points out, because of the concomitant lowered food intake of the exercised mice.

This survey of the experimental evidence on the genesis of spontaneous mammary tumors in mice leads one to conclude that dietary regimens, so far as are known, which inhibit or delay the disease in animals are too drastic even if applicable to be of practical value as a means of preventing human cancer. The effect of exercise on carcinogenesis has been too inadequately investigated, even in animals, to be used as a guide in drawing definite conclusions about its efficacy as a measure in the prevention of human cancer. On the other hand, nutritionally good diets have been observed to delay carcinogenesis. Therefore, no broad generalization on the effect of nutrition in the prevention of human cancer should be made at this time.

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[6] B. E. Kline and H. P. Rusch, *Cancer Research*, 4: 762, 1944.

FAT CONTENT OF GUINEA-PIG MILK

DURING a search of the literature for data on the guinea pig, I found several references which quoted the fat content of guinea-pig milk as being 45.80 per cent. This figure had been taken from page 470 of "Physiological and Pathological Chemistry" by G. Bunge (2nd Eng. Ed., 1902, Philadelphia, Blakiston). Going back to the original paper, I found that the author was Professor Purdie (Chemical Laboratory of St. Andrews) and that the paper was published in *Chemical News*, Volume 52, page 170, 1884. Actually this paper deals with dolphin milk. The editors of the above-mentioned edition of Bunge's text-book got their information from "Chemie der menschlichen Nahrungs- und Genussmittel," by Dr. J. König, which quotes Purdie's figures for "Meerschwein Milch (Delphinus phocaena)" as being 45.80 per cent. Thus a mistake has been made by translating "Meerschwein" to "guinea pig." The German for guinea pig is Meerschweinchen.

To show that Bunge himself did not believe that guinea pigs had such an excessively fat milk, I would like to refer to Table I, page 142, in his book "Physiologie des Menschen" (1905, Leipzig). Here he quotes the guinea-pig milk as having 7.1 per cent. fat and dolphin milk 43.8 per cent.

Abredhalden in "Physiological Chemistry," page 654, gives the two figures 7.31 per cent. and 6.96 per cent. for fat content of guinea-pig milk. A few samples of guinea-pig milk tested in the laboratory for experimental biology of this department gave about 6.0 per cent. fat. It is known that this figure is a little low, because the last milk was not taken from the nursing guinea pigs.

The above error in translation has resulted in a wide-spread misconception as to the fat content of guinea-pig milk, 45.80 per cent. being the figure most commonly quoted. I feel, therefore, that the matter should be brought to the attention of your readers.

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