Diet and Prolactin Release

Sir—Epidemiological data suggest that urbanisation and Westernisation increase the risk of breast cancer but that modification of the life style and/or diet of Caucasian women can lower the risk. Since changes in dietary components and diets high in carbohydrate and low in fat may increase rapid-eye-movement (R.E.M.) sleep, the release of pituitary hormones dependent on or associated with changes in R.E.M./non-R.E.M. sleep patterns could perhaps be altered by dietary factors. Parker et al. reported that the nocturnal release of prolactin was dependent on the R.E.M./non-R.E.M. pattern. Since high-fat diets may be associated with a higher incidence of breast cancer, and since Kwa et al. have suggested that prolactin release in high-risk women may be abnormal, it would be of interest to determine whether the diurnal cycle of prolactin release could be altered by a change of diet. In 4 healthy none-obese nurses (aged 24-37) we found that changing from a Western diet to a vegetarian one was accompanied by a reduction in nocturnal release of prolactin.

The subjects had regular menstrual cycles and no evidence of endocrine abnormalities, cardiovascular disease, or diabetes. They started on a Western diet (carbohydrate 45%, fat 40%, protein 15%, cholesterol 0.5-0.7 g daily) then transferred to a vegetarian diet (carbohydrate 52%, fat 33%, protein 15%, total calories 2100-2300, cholesterol 0.7-0.8 g daily). The dietary intake was determined using the Burke questionary, then transferred to a vegetarian diet (carbohydrate 52%, fat 33%, protein 15%). Plasma-prolactin was determined by radioimmunoassay (RIA), as described by Sinha et al., using N.I.A.M.D.D. Lewis HPRL V-L-5 no. 2. Interassay reproducibility of the plasma-prolactin in reference to plasma-samples was approximately 10% and the precision of the prolactin standard at concentrations of 624 pg and 2.5 ng was 1% and 1.4% respectively. Plasma-luteinising-hormone (L.H.) was determined by R.I.A., as described by Midgley. Cholesterol and triglyceride levels were determined by standard 'Autoanalyser' methods. Statistical analysis was carried out using the paired t test.

The nocturnal increase in plasma-prolactin was significantly less when the nurses were on a vegetarian diet (fig. 1). Interestingly, the L.H. fluctuations seen during the night on a Western diet appeared to be dampened in nurses fed a vegetarian diet (fig. 2). The cholesterol and triglyceride levels were not affected by the change to a vegetarian diet (cholesterol

Fig. 1—Nocturnal release of prolactin in healthy premenopausal women. Average plasma-prolactin levels (4 subjects) during Western diet (solid line) and on vegetarian diet (dashed line).

Fig. 2—Nocturnal release of L.H. in healthy premenopausal women. Plasma-L.H. (mean of 4 subjects and standard error) on Western diet (solid line) and vegetarian diet (dashed line).
151±6.5 mg/dl, triglycerides 62±13 mg/dl on Western diet; cholesterol 160.5±16 mg/dl triglycerides, 73±18 mg/dl on vegetarian diet). No change in weight was evident in any of the nurses.

Wynder et al. 9 and others 14,15 have postulated that the incidence of breast cancer is related to dietary factors, and that environmental changes, mainly dietary, alter the hormone profile. 16 We have previously reported9 that the basal prolactin taken at 9-9.30 A.M. during the menstrual cycle is reduced in nurses fed a vegetarian diet. The present study further suggests that removal of meat and meat products from the diet (these contribute some 40% of the total fat intake of Caucasians) can reduce the nocturnal release of prolactin and dampen the L.H. "spiking" during the night. Whether there is a relationship between the lack of L.H. spiking and the lower release of prolactin18 remains to be determined. Lack of change in the plasma-cholesterol on the vegetarian diet was probably due to increased consumption of eggs and cheese. Since cholesterol is an indicator of adrenal activity and has been reported to increase in breast-cancer patients, 19 reduction of cholesterol intake may be important in cancer patients.

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P. HILL
F. WYNDER

VISUALISATION OF THROMBI WITH TECHNETIUM-99m UROKINASE

Sir,—It was with consternation that we noted the incomplete reproduction of the figure with our article of Aug. 14 (p. 341). While the meaning of the article is clear, it would have been of great benefit to have both halves of the figure produced fully to document the failure to label the thrombus on the scintigram. The scintigram revealed no localised concentration of radioactivity:

It is with great interest that we note the responses of Dr Cox and his colleagues and Dr Millar and Dr Smith (Sept. 11, p. 572). Certainly quality control of radiopharmaceuticals is a difficult problem and very minor changes in technique can result in quite different in-vivo results. We did not have indication of splenic concentration of isotope. This could have escaped our attention, but it seems somewhat unlikely to us that colloidal formation occurred. We will repeat these studies to determine this and will be in contact with both groups of workers to obtain sufficient details of their procedures to allow us to repeat them, and, perhaps, improve our results.

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**We apologise to Dr Weir and his colleagues for omitting the scintigram from their Aug. 14 paper.—Ed. L.

DIETARY IODINE AND CANCER RISK

Sir,—The material cited by Dr Stadel in his hypothesis 1 concerning iodine deficiency and breast cancer risk seems inadequate in light of the large mass of research published on this subject over the past ten years by myself and others. Some of the work cited came from my research; no reference was made to its source in the article.

A complete review of iodine metabolism and breast cancer was presented and published by the New York Academy of Sciences in 1970. Research-work published since then has revealed histopathological changes within breast tissues with iodine deprivation. Additionally, there has been clinical research done in diagnosis and therapy. The action of oestrogen in iodine deprivation has been described and the end-organ responses and intracellular changes represented by oestrogen receptor, cytosol radioactive iodine uptake determinations, and autoradiographic findings have been presented.

Research studies utilising prolactin show histological characteristics that differ from those of iodine deficiency. Interactions between gonadotrophin and hypothyroidism have been described: however, the changes are relative to the production-rate of thyroid hormone. Iodine deficiency does not necessarily induce hypothyroidism because of the ability of the body to conserve available iodine. Research on dietary replacement would seem to make some of Dr Stadel's conclusions inappropriate.

In light of this large volume of laboratory and clinical research that has not been taken into account in the hypothesis, it seems redundant.

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BERNARD A. ESKIN

**This letter has been shown to Dr Stadel, whose reply follows.—Ed. L.

Sir,—Dr Erskin has published extensively on the effects of iodine deficiency on the mammary tissue of rats. 2,3 His work in humans includes the observation that abnormal (dysplastic or neoplastic) breast tissue demonstrates an increased uptake of radioactive iodine as compared with normal breast tissue 6 and the observation that iodine and thyroid supplementation of the diet decreases mammographic evidence of dysplasia. 7

1. Stadel, B. V. Lancet, 1976, i, 89.