# ABSTRACTS OF THE 9th CONGRESS OF THE INTERNATIONAL STUDY GROUP FOR STEROID HORMONES (Rome, 5-7 December 1979)

## BREAST CANCER

1. Endocrine determinants of risk of breast cancer, R.D. BULBROOK, Imperial Cancer Research Fund, Lincoln's Inn Fields, London

It has been plain for many years that reproductive function is associated with risk of breast cancer. In particular, early age at menarche, late age at first child or nulliparity and late age at menopause, are all associated with enhanced risk. Conversely, oophorectomy even up to age 40 considerably reduces the incidence of the disease. In the 1950's, analytical methods became available for the precise measurement of endocrine function and it might have been expected that rapid progress would be made in the identification of endocrine determinants of risk. This has not been the case. The problem has been remarkably elusive and it is only recently that tentative leads have appeared.

There is tenuous evidence for 3 endocrine abnormalities that are associated with enhanced risk. The first of these is corpus luteum dysfunction. In both benign breast disease and in breast cancer there is a diminished production of progesterone in the luteal phase of the cycle whereas oestrogen levels are normal or raised.

The second abnormality concerns prolactin. Most studies show little or no difference between plasma prolactin levels (measured in the morning) in cases and controls. However, early evening values of prolactin appear to be higher than normal in various risk groups.

Finally, a prolonged prospective study has shown that urinary androgen excretion was sub-normal in a substantial proportion of women (mainly premenopausal) who subsequently developed breast cancer.

The question now to be answered is whether these abnormalities in ovarian, pituitary and adrenal function are inter-related, stemming from a single cause, or whether they occur independently, each one being associated with an incremental enhancement of risk. The resolution of this problem is important because it is now feasible to consider correcting the abnormalities in the hope of eventually reducing the incidence of breast cancer.

2. Effect of diet on estrogen metabolism in women, H. ADLERCREUTZ[1], B.R. GOLDIN[2], J.T. DWYER[2], J. WARREN[2] and S.L. GORBACH[2], [1]Department of Clinical Chemistry, University of Helsinki, Meilahti Hospital, 00290 Helsinki 29, Finland, and [2]Infectious Diseases Service, Department of Medicine, Tufts-New England Medical Center, Boston, Massachusetts 02111, U.S.A.

Epidemiological studies have indicated associations between the consumption of animal fat, estrogen (E) metabolism and breast cancer. The possible relation between diet and E metabolism has therefore been studied by measuring E in plasma, urine and feces by RIA in: 1) young and 2) old omnivors and 3) young and 4) old vegetarians, and in 5) breast cancer patients. Collection of urine and feces was performed over three consecutive days and a plasma pool was formed from three samples drawn in this same period. Young subjects were studied in the mid-follicular phase. This collection scheme is being repeated 4 times over one year. Preliminary results from 2-3 collection periods are available for 29 women belonging to the first four groups. Mean plasma estrone (E1) and estradiol (E2) were 37-83% higher in the omnivors than in the vegetarians (though not statistically significant in the limited number of subjects so far studied). Urinary E excretion was the same in both dietary groups with the exception of immunoreactive estriol-3-glucuronide (E3-3G) which was 35% higher in the young omnivors than in the young vegetarians. However, fecal excretion of unconjugated E1, E2 and E3 was 2.4 to 6.5 times higher in the vegetarians than in the omnivors (statistically significant in both age groups). The lower plasma level of E and urinary E3-3G excretion in vegetarians can be explained by the observed increased fecal elimination of E. The results would suggest that vegetarians reabsorb biliary E to a smaller extent than omnivors, thus making these E more available for intestinal bacterial destruction and fecal elimination. It is concluded that diet significantly affects E metabolism and that this influence may explain some of the epidemiological data obtained.