THE IMPACT OF PARITY ON THE BIOLOGY OF BREAST CANCER: STEROID HORMONE RECEPTORS AND HISTOPATHOLOGICAL PARAMETERS. Susan M.Thorpe, Carsten Rose, Bo V.Petersen, and Birgitte Bruun Rasmussen. The Finsen Institute, The Fibiger Laboratory, and the D.B.C.G. Secretariat, Strandboulevarden 49, 2100 Copenhagen Ø, Denmark.

Tumour tissue from breast cancer patients registered by the Danish Breast Cancer Cooperative Group (DBCG) has been analyzed for estrogen and progesterone receptor (ER and PgR) content using the dextran-coated charcoal (DCC) method. Analysis of 1209 biopsies revealed that non-parous pre/perimenopausal women have significantly higher ($p<0.03$, Wilcoxon Rank Sum Test) PgR content in their tumour tissue than their parous counterparts (98 vs. 44 fmol/mg cytosol protein). This difference between parous and non-parous patients was not found in post-menopausal women. The effect of no, single, or multiple births on steroid hormone receptor content was evaluated. Size of the primary tumour and lymph node involvement has been investigated in approximately 1000 pre/perimonopausal and 1500 post-menopausal women with high risk, primary breast cancer registered by DBCG. In comparisons between parous and non-parous women within each menopausal group, a significant trend ($p<0.001$) toward presentation with larger tumours was found among the non-parous, pre/perimenopausal women; and significantly more ($p<0.003$) positive lymph nodes were found among parous, post-menopausal women.

INHIBITORY EFFECT OF BUTYLATED HYDROXYANISOLE, ETHOXYQUIN AND ACETAMINOPHEN IN THE INDUCTION OF HEPATIC TUMOURS IN THE RAT. Hiroyuki Tsuda, Takao Sakata, Katsumi Imaida and Nobuyuki Ito. First Department of Pathology, Nagoya City University Medical School, Nagoya 467, Japan.

The objective is to evaluate the inhibitory effect of butylated hydroxyanisole (BHA), ethoxyquin (EQ) and acetaminophen (AA) on the induction of preneoplastic and neoplastic lesions in the rat liver after treatments with diethylnitrosamine (DEN) or N-ethyl-N-hydroxyethylnitrosamine (EHEN). Experiment 1: Rats were initially treated by DEN and two weeks later were given 2% BHA, 2% EQ or 1.3% AA mixed in the diet for 6 weeks. All animals were subjected to partial hepatectomy at the end of week 3. Experiment 2: Rats were given 0.1% EHEN for 2 weeks and were fed on basal diet containing 2% BHA, 0.8% EQ or 1.3% AA from week 3 for 29 weeks. The number and area of γ-glutamyltransferase-positive foci per unit area were significantly decreased in rats given BHA, EQ or AA compared with controls in both experiments. Similarly, quantitiative values of hyperplastic nodules (mm^2/cm^2) were significantly decreased in the groups given BHA (3.0), EQ (2.3) or AA (2.3) compared to the control (7.0) in experiment 2. The incidences of hepatocellular carcinoma (HCC) in experiment 2 were: BHA, 2/24 (8.3%); EQ, 3/27 (11.1%); AA, 3/27 (11.1%), whereas that of control was 11/25 (44.0%). These results indicate that antioxidant (BHA, EQ) and antipyretic (AA) have inhibitory effect on the induction of preneoplastic lesions and HCC in the rat liver.

PHALLOIDIN SENSITIVITY OF HEPATOCYTES OF RATS FED 2-AAF OR LIVER TUMOUR PROMOTERS. H.Tsukada and N.Sawada. Department of Pathology, Cancer Research Institute, Sapporo Medical College, Sapporo, Japan.

Changes in cell membrane function of rat hepatocytes induced by 2-AAF or liver tumour promoters were evaluated, using sensitivity to phalloidin of the cells isolated by collagenase-perfusion method and the relationship between the sensitivity and cytochemical GGT-positivity of the cells as parameter. The sensitivity was decreased as 2-AAF feeding was prolonged, and the tumour cells were most resistant to the toxin. GGT-positive cells in preneoplastic lesions and tumours were more resistant than the negative cells. Tumour promoters like phenobarbital (PB), DDT, CPIB or BHT lowered the sensitivity reversibly, while non-promoters like diphenylhydatoin had no effect. The GGT-positive cells seen in response to PB or BHT feeding were more resistant than the negative cells. It is suggested that changes in the cell membranes responsible for the decrease in phalloidin sensitivity relate to the progression of carcinogenesis as well as to GGT-positivity of the cells, and also that liver tumour promoters induce changes in cell membrane functions identical at least in part to those of the cells in carcinogenesis.