INTRODUCTION

The role of prolactin (PRL) is now well established in the induction and promotion of many experimental mammary tumours (Welsch and Nagasawa, 1977). However, the role of PRL and other anterior pituitary hormones in the pathogenesis of human breast cancer is not fully elucidated. PRL has been suspected to play a major role in stimulating the growth of some human breast cancers (l'Hermitte M and l'Hermitte-Baleriaux M, 1988). Jick et al. (1974) reported increased incidence of breast tumours as a result of clinical use of dopamine antagonists such as Rauwolfia drugs and the Phenothazines which leads to elevation of PRL secretion but this finding was contradicted by other studies (Mack et al., 1975, Wagner and Mantel, 1978; Seth, 1982).

Breast may be the target organ for various peptide hormones secreted from the anterior pituitary. PRL is one of the principal hormones regulating the alveolar function of the mammary gland (Anderson R.R., 1974). Breast may also undergo changes in response to the secretion of gonadotrophic hormones (LH and FSH) as well. The changes in leutinizing hormone (LH), pulsing mechanism or the 'firing rate of pituitary hormones' are believed to be responsible for the onset of puberty (Wildt et al., 1980) but whether such changes at the hypothalamic-pituitary-ovarian axis, are responsible for breast stromal development is unknown. Since the breast cells were shown to
possess specific binding sites for estrogens and progesterone (McGuire et al., 1977), the effect of LH and follicular stimulating hormones (FSH) on the breast tissue may be through their effect on the ovarian secretion.

PRL is known to exert several physiologic actions on human breast cells (Shiu and Friesen, 1980) but apart from a few case reports (Brown et al., 1982, Rowe PH, 1984) epidemiologic evidence of breast pathology in patients with hyperprolactinaemia is scant.

Luteal insufficiency which may be caused by hyperprolactinaemia (Seppala et al., 1976) has been reported to be associated with breast disease (Mauvais-Jarvis et al., 1979). A number of studies of plasma PRL level have been performed (Boyns et al., 1973, Kwa et al., 1974; Sheth et al., 1975; Watt Boolsen, 1981; Simkin B, 1982; Walsh et al., 1984b; Wang et al., 1987) but the results of these studies present a mixed picture. This was obvious on account of a very subtle hormonal changes, if any, that exist in these patients. The pulsatile secretion of hormones, diurnal variation in secretion, measurement errors, non standard protocols, etc. made it indeed very difficult to interpret these findings.

The importance of PRL receptors as an index of responsiveness of the tumour tissue has been established in recent years as hormone influence cells by first binding to high affinity receptor protein located either on the cell surface or in the cell interior. Evaluation of the capacity of hormone receptors as an index of response of treatment with either hormone administration or endocrine ablation is clearly of clinical importance. There are several clinical reports of treating benign breast disease by PRL inhibitor and dopamine
agonist drug, bromocriptine (Mansel et al., 1980). Also Leutinizing hormone releasing hormone (LH-RH) analogue has been shown to be effective in treatment of advanced breast cancer (Klign et al., 1985).

PRL binding sites have been reported to be present in breast tumours (l'Hermite-Baleriaux and l'Hermite, 1984; Kumar et al., 1987) but it is unknown whether other peptide hormone LH and FSH have any direct action on the breast tissue. In view of the fact that paracrine like activity takes place in endocrine target organs it was hypothesised that specific binding of peptide hormone of anterior pituitary may be seen in the breast tissue and breast cancer cells. Therefore, in the present study, binding capacity of breast tissue for various peptide hormones in breast were ascertained immunocytochemically and biochemically. Such a knowledge, it is envisaged, will provide basis for further research in this area and help to evaluate newer therapeutic modalities.