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THE EFFECT OF TAMOXIFEN AS ANTIOXIDANT ON MAMMARY TUMOUR AND ON IRRADIATION INDUCED CHANGES IN FEMALE RATS

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INTRODUCTION

Breast cancer

Breast cancer is one of the most common cancers in women. In 1896, Beatson demonstrated that women with advanced Breast cancer show improvement if their ovaries are removed [1]. It is now accepted that one in three premenopausal patients with breast cancer responds to oophorectomy.

Tritium labeled hexestrol and estradiol bind to and is retained by the estrogen target tissues (uterus, breast, vagina, and pituitary gland) of laboratory animals [2]. These findings led to the identification of an estrogen receptor protein in estrogen target tissues and the subsequent development of a subcellular estrogen receptor model by Jensen and Gorski et al [3,4]. This model appeared to be consistent for all species and estrogen target tissues. (Model 1). Another model proposes that the estrogen receptor is a nuclear protein and therefore the steroid must diffuse into the nucleus to form a receptor-complex and initiate estrogen action. The two models are compared in fig. 1[5.6].

It was found that if the estrogen receptor is present in a tissue, estrogen must have a function in the cells. The concept was extrapolated to breast cancer to preselect patients who might respond to endocrine therapy. Different concentrations of estrogen receptors are present in breast cancer[7.8], that can be explained by the heterogenicity of the tumor cell population. The more cells in the tumor that contains estrogen receptors, the higher the overall estrogen receptor content. Approximately, 60% of estrogen receptor positive (receptor rich) patients are responsive to endocrine therapy, whereas only 10 % of estrogen receptor negative