

Answer 1:

Bibliographic Information

Inhibition of hamster melanoma growth by estrogen. Schleicher, Rosemary L.; Hitzelberger, M. Helen; Beattie, Craig W. Sch. Med., Univ. Illinois, Chicago, IL, USA. Cancer Research (1987), 47(2), 453-9. CODEN: CNREA8 ISSN: 0008-5472. Journal written in English. CAN 106:96417 AN 1987:96417 CAPLUS (Copyright (C) 2008 ACS on SciFinder (R))

Abstract

A malignant hamster melanoma cell line HM-1 derived from the heterogeneous malignant hamster melanoma MM1 contains a specific, high-affinity binding protein for estrogens. Partial purifn. of the binding protein with $(\text{NH}_4)_2\text{SO}_4$ (40% satn.) increased mean binding content (3.1 fmol/mg protein) 15-fold without any change in affinity (10^{10} M⁻¹). The binding protein sedimented at 8-9S on 10-30% low-salt sucrose gradients and 9-10S in the presence of 20 mM MoO_4^{2-} . Addn. of 0.4 M KCl shifted the 8S peak to 4S. Binding was specific, saturable, and indicative of a single class of high-affinity sites over a concn. range of 0.01-10.0 nM ^3H estradiol. Estradiol ^3H [50-28-2] produced a dose-related inhibition of HM-1 growth in vitro without altering the growth of an addnl. line (HM-2) which did not bind estrogen. The antiestrogen tamoxifen ^3H [10540-29-1] (10^{-7} M) also inhibited HM-1 melanoma growth in vitro, an effect reversed by the addn. of estradiol (10^{-9} M). HM-1 xenografts grew faster in female BALB/c-nu/nu mice than male mice, whereas there was no sex difference in HM-2 growth. Pharmacol. doses of estradiol and the antiestrogen nafoxidine inhibited HM-1 growth without altering tumor incidence or latency. Apparently, HM-1 cell lines bind estrogens specifically and with high affinity and hamster melanoma cells pos. for this binding protein respond to estrogen.