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Cimicifuga racemosa extract BNO 1055 inhibits proliferation of the human prostate cancer cell line LNCaP.

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Extracts from black cohosh (Cimicifuga racemosa, CR) exert an anti-proliferative action in human breast cancer cell cultures, which has been attributed to an anti-estrogenic effect. However, CR constituents do not bind to either of the known estrogen receptors. Thus, the anti-tumor effect of CR me be mediated by mechanisms not involving these receptors. Polycyclic aromatic hydrocarbons are toxic environmental pollutants, which indirectly act as anti-estrogens by activating the aryl hydrocarbon receptor (AhR). The AhR is widely expressed in mammalian tissues and tumors. A recent screening study demonstrated activation of the AhR by a variety of herbal extracts, among others, CR. Since activation of the AhR causes inhibition of growth of prostate cancer cells, we addressed the question, whether CR may not only inhibit growth of breast cancer--but also of prostate cancer cells. In the AhR ligand assay, the CR extract BNO 1055 reduced tracer binding to 71% of the control demonstrating interaction of constituents of this extract with the receptor. Under basal as well as under estradiol- and dihydrotestosterone stimulated conditions, the CR extract dose dependently inhibited proliferation of LNCaP cells. A significant reduction of cell growth was observed at a concentration as low as 50 ng/ml. Thus, it is demonstrated for the first time that CR compounds potently inhibit the growth of human prostate cancer cells in vitro. This anti-proliferative effect may be mediated via the AhR.

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