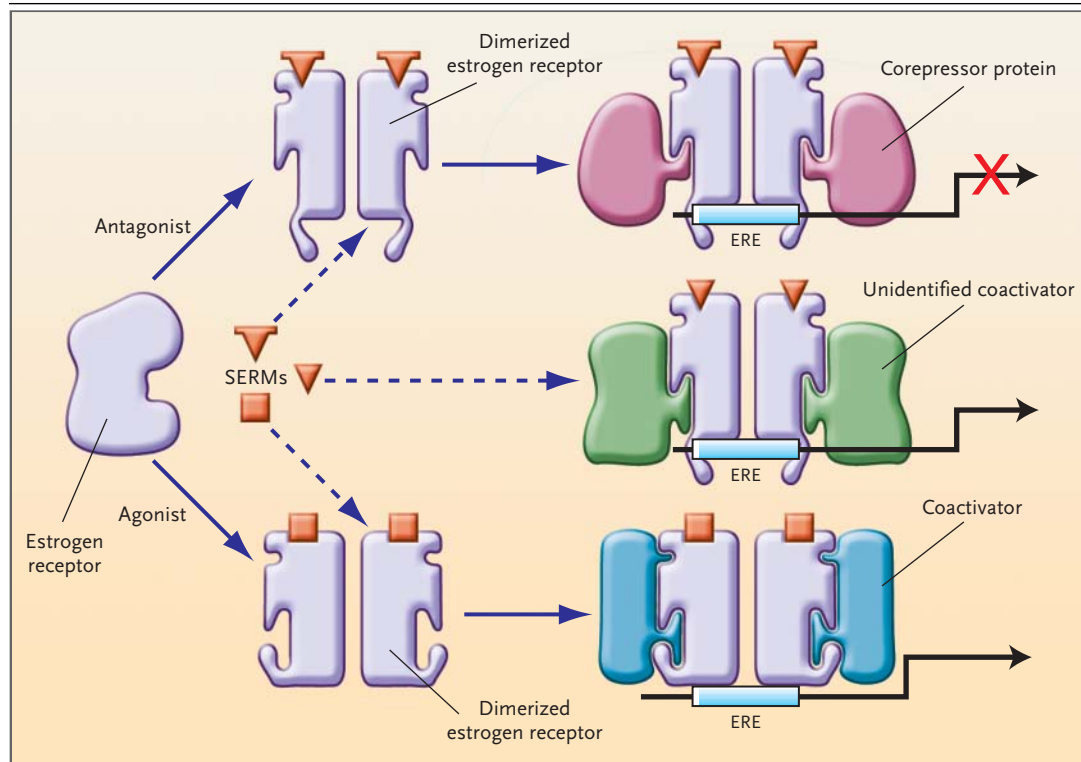


CORRECTIONS

Adefovir Dipivoxil for the Treatment of Hepatitis B e Antigen–Negative Chronic Hepatitis B (February 27, 2003;348:800-7) and Adefovir Dipivoxil for the Treatment of Hepatitis B e Antigen–Positive Chronic Hepatitis B (February 27, 2003;348:808-16). On page 801, in line 10 of the right-hand column, and on page 809, in the first line of the second paragraph, the trade name for adefovir dipivoxil should be “Hepsera” rather than “Preveon.” We regret the error. The Web versions of the articles have been corrected.

Selective Estrogen-Receptor Modulators — Mechanisms of Action and Application to Clinical Practice (February 13, 2003;348:618-29). There was an error in Figure 2; a corrected figure is shown here.



**Figure 2. Estrogen-Receptor Action.**

Each class of SERMs (orange symbols) has a slightly different shape, although all will bind to the estrogen receptor. When it binds to an estrogen, antiestrogen, or SERM, the estrogen receptor undergoes a conformational change that permits its spontaneous dimerization and facilitates the subsequent interaction of the dimer with estrogen response elements (EREs) located within target genes. The estrogen-receptor–ligand complex also leads to binding of various coregulator proteins that vary with its conformational structure. Some estrogen-receptor–SERM complexes favor corepressor recruitment (red) that, in a given target cell, increases its antagonist activity, and others favor coactivator recruitment (blue) that increases its agonist activity. Some SERMs may also facilitate the interaction of the estrogen receptor with yet-to-be-identified coactivators (green) with which estrogens or antiestrogens would not normally couple. It has now been determined that estrogen facilitates the interaction of the estrogen receptor with coactivators. The antagonist-activated estrogen receptor, on the other hand, interacts preferentially with corepressors. The binding of different SERMs to the receptor permits the receptor to adopt conformational states that are different from each other and also distinct from that induced by classic estrogen agonists or antagonists. The implication of this model is that SERM activity will be influenced by the relative levels of expression of the coregulator proteins (corepressors and coactivators) that are expressed in different target cells.