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Ginsenoside-Rb1 Acts as a Weak Estrogen Receptor Agonist Independent of Ligand Binding.

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Ginseng is a medicinal herb widely used in Asian countries, and its pharmacological effects has been demonstrated in various systems such as cardiovascular, central nervous, and endocrine systems. Its effects are mainly attributed to the ginsenosides. We hypothesize that a component of *Panax ginseng*, ginsenoside-Rb1, acts by binding to estrogen receptor. We have investigated the estrogenic activity of ginsenoside-Rb1 in a transient transfection system using estrogen receptors α or β with estrogen-responsive luciferase plasmids in COS monkey kidney cells. Ginsenoside-Rb1 activated both estrogen receptors α and β in a dose-dependent manner (0.5 -100 M). Activation was inhibited by the specific estrogen receptor antagonist ICI 182,780, indicating that the estrogenic effect of ginsenoside-Rb1 is estrogen receptor dependent. Next, we evaluated the ability of ginsenoside-Rb1 to induce estrogen-responsive progesterone receptor gene by semi-quantitative RT-PCR assays. MCF-7 cells treated with 17β-estradiol or ginsenoside-Rb1 exhibited an increased expression of progesterone receptor mRNA. However, ginsenoside-Rb1 failed to displace the specific binding of [3H]17β-estradiol to estrogen receptor in MCF-7 cells as examined by whole cell ligand binding assays, suggesting that there is no direct interaction of ginsenoside-Rb1 with estrogen receptor. Our results indicate that estrogen-like activity of ginsenoside-Rb1 is independent of direct estrogen receptor association.