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Am J Med. 2006 Feb;119(2):167.e1-8. doi: 10.1016/j.amjmed.2005.08.012.

Progestins affect mechanism of estrogen-induced C-reactive protein stimulation

David B Reuben [1], Shana L Palla, Peifeng Hu, Beth A Reboussin, Carolyn Crandall, David M Herrington, Elizabeth Barrett-Connor, Gail A Greendale

Affiliations

Affiliation

1 Multicampus Program in Geriatric Medicine and Gerontology, David Geffen School of Medicine at University of California, Los Angeles 90095-1687, USA. dreuben@mednet.ucla.edu

PMID: 16443423 DOI: 10.1016/j.amjmed.2005.08.012

Abstract

Purpose: To determine whether the mechanisms of C-reactive protein production differ depending on the presence or absence of a progestin in the regimen.

Subjects and methods: We examined data from the Postmenopausal Estrogen Progestin Intervention (PEPI) study, a 5-group (3 different combined estrogen-progestin regimens, conjugated equine estrogen-only, and placebo) randomized clinical trial. This substudy included 221 postmenopausal women assigned to active treatment groups who took at least 80% of pills and had stored plasma specimens available to assess 12-month changes in estrone, sex hormone binding globulin, interleukin (IL)-6, and C-reactive protein levels.

Results: All treatments resulted in increases in estrone, sex hormone binding globulin, and C-reactive protein at 12 months compared with baseline values. In all progestin-containing groups, 12-month change in IL-6 was positively correlated with 12-month change in C-reactive protein (r between 0.34 and 0.65, each P <.010). By contrast, in the conjugated equine estrogen-only group, 12-month change in IL-6 was negatively correlated with 12-month change in C-reactive protein (r value -0.31, P = .055). In adjusted models predicting 12-month C-reactive protein change, an interaction between change in IL-6 and treatment group was highly significant (P=.0008-P <.0001) for each of the progestin-containing groups compared with the conjugated equine estrogen-only group. In the conjugated equine estrogen-only group, the change in C-reactive protein per unit increase in IL-6 was -0.88, whereas in the progestin-containing groups it ranged from 1.46 to 2.85 (P <.0001 for each comparison with conjugated equine estrogen-only).

Conclusion: Progestins in combination with conjugated equine estrogen potentiate the IL-6 (inflammatory)-mediated stimulation of C-reactive protein. These findings support the hypothesis that progestins plus estrogen, not estrogen alone, generate C-reactive protein through an inflammatory mechanism.

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