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## Oral Estrogen May Aggravate Metabolic Abnormalities in Women with Growth Hormone Deficiency

BETHESDA, MD -- November 30, 2001 -- Oral administration of estrogen replacement therapy suppresses the biological actions of growth hormones (GH) in GH-deficient women, research suggests.

Findings demonstrate for the first time that the impact of oral estrogen extends beyond effects on circulating insulin growth factor I (IGF-I) levels as GH-induced stimulation of fat oxidation, protein metabolism are also affected.

The findings from twin pair crossover studies appear in the December 2001 issue of the *American Journal of Physiology: Endocrinology and Metabolism*, one of the 14 peer-reviewed journals published by the American Physiological Society (APS).

Growth hormones have recently been approved for replacement treatment in adults in several countries. They play an important role in regulating body composition and physical and psychological wellbeing in adult life. However, there is limited information regarding the interaction of GHs with other hormones during replacement therapy. Accordingly, observations in postmenopausal women raised the question as to whether the traditional oral route of estrogen replacement reduces the biological effects of growth hormones.

Two studies were undertaken comparing the effects of oral and transdermal estrogen administration on the biological actions of growth hormones. Doses employed are those routinely used in the therapy of women with hypopituitarism.

The first study investigated insulin-like growth factor I (IGF-I) responses to three different doses of GH (dose-response study). The second study investigated metabolic effects of growth hormone GH on lipid oxidation and whole body protein metabolism (metabolic study).

Ten hypopituitary GH-deficient women with hypogonadism were recruited from the Endocrine Outpatient Clinic at St. Vincent's Hospital, in Sydney, Australia. They were randomized into two separate studies, which were separated by at least three months. Six subjects participated in both studies.

Growth hormone deficiency was confirmed by a peak GH response of <3 ng/mL during an insulin tolerance test. The duration of hypopituitarism was at least one year, and no subjects had received GH before. All hypopituitary subjects were receiving stable hormone replacement for other deficiencies, except for one subject of postmenopausal age who did not receive sex steroid replacement.

Eight subjects participated in each study. Both studies were of open-label, randomized, crossover design, allowing for differences in treatment effect (i.e., route of estrogen administration) to be compared during estrogen therapy.

Each subject was randomized to 2 mg/day oral estradiol valerate or transdermal estrogen patches delivering 100 µg of 17estradiol daily for eight weeks. The subjects then crossed over to the alternate estrogen treatment for an additional eight weeks.

The estrogen dosages used were based on data indicating equivalent biological activity, as measured by gonadotropin suppression and vaginal cytology. Medroxyprogesterone acetate (10 mg daily) was coadministered on the last 12 days of each four-week cycle of estrogen treatment to induce withdrawal bleeding.

The two randomized, crossover studies show that oral estrogen replacement therapy suppresses the biological actions of growth hormones in GH-deficient women, the researchers concluded.

In the first study, mean IGF-I across all three GH doses was significantly lower, and the rise in IGF-I during oral estrogen was significantly less than that observed during transdermal therapy.

In the metabolic study, postprandial lipid oxidation and leucine incorporation into protein were stimulated by GH treatment but remained significantly lower during the oral estrogen phase.

The route-dependent effects of estrogen on IGF-I, fat oxidation, and protein metabolism were evident even before GH administration. Thus, in GH-deficient, hypogonadal women, oral estrogen exhibits intrinsic metabolic actions that are opposite those of GH and are not overcome by replacement doses of GH currently used in clinical practice, indicating the physiological importance of these observations.

The findings demonstrate for the first time that the impact of oral estrogen extends beyond effects on circulating IGF-I levels in that GH-induced stimulation of fat oxidation and protein metabolism are also affected, the researchers report.

Although fat oxidation was stimulated by GH, it remained suppressed to a greater degree postprandially during the oral estrogen treatment. Similarly, although GH stimulated protein metabolism, leucine incorporation into protein was significantly lower during oral estrogen therapy. These observations in

postmenopausal women strongly suggest that similar detrimental changes may occur with conventional oral estrogen therapy in untreated GH-deficient women.

The researchers conclude that:

- Estrogen at a therapeutic dose exerts significant route-dependent effects on GH action in women with organic GH deficiency.
- Compared with the transdermal route, oral estrogen aggravates metabolic abnormalities of GH deficiency and attenuates the metabolic effects of GH therapy. Thus oral estrogen may worsen the body composition abnormalities of GH deficiency and limit the benefits of GH replacement therapy in GH-deficient women.
- The route of estrogen administration is an important consideration both before and during GH replacement therapy in hypogonadal GH-deficient women.

The authors of the studies, "Oral Estrogen Antagonizes the Metabolic Actions of Growth Hormone In Growth Hormone-Deficient Women," are Troels Wolthers, David M. Hoffman, Ailish G. Nugent, and Ken K. Y. Ho, all from The Garvan Institute of Medical Research, St. Vincent's Hospital and Biomedical Mass Spectrometry Unit, University of New South Wales, Sydney, Australia; and Mark W. Duncan and Margot Umpleby, both from The Endocrine and Diabetic Unit, St. Thomas's Hospital, London.

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