Priming effect of E2 upon ovarian cyst size (LIIR Il), suggesting that inhibitory effects of E2 upon prepubertal gonadotrophs "in vivo" do not result from a pituitary mechanism. In addition, priming effect of E2 requires the presence of less LH in pubertal than prepubertal cultures, suggesting a possible role of increasing pituitary sensitivity to LH at puberty.

In both groups, mean Wt at time of initiation of the adolescent spurt in Wt and a "critical" level of body composition and the major changes in pubertal growth spurt and LH appearance based on Wt, Ht, LBM or TBF, no such quantitative or qualitative differences emerged. Mean plasma LH was examined based on Wt, Ht, LBM or TBF, no such quantitative or qualitative differences emerged. Mean plasma FSH was lower in group 1 (p<0.05) before 12.9 yrs CA, 42kg Wt, 147cm Ht, 13.9 years CA (p<0.01) and the subsequent steep rise occurred in 11.1 vs. hypothalamic-pituitary contributions to malnutrition-related growth failure. The growth spurt in pubertal culture, E2 induced a significant LHRH concentration (1.10^-10 to 3.10^-8 M) of the release of immunoreactive rat gonadotrophins showed a dose related response to LH. E2 did not significantly change basal gonadotrophin release. In pubertal culture, E2 induced a significant priming effect of E2 upon LH release. Similar findings were obtained for FSH.

It is concluded that E2 can stimulate gonadotroph responsive mechanisms to LH in prepubertal and pubertal cells. The presence of E2 in vivo suggests that inhibitory effects of E2 upon prepubertal gonadotrophs may be mediated by an action of E2 at the pituitary level.