Pre-ovulatory Estradiol release induces phasic GnRH release which triggers LH surge

Abstract:

Hypothalamic GnRH neurons initiate a critical hormonal pathway which must function properly in order for the female menstrual cycle to occur properly. Estradiol – upon reaching highly elevated concentration levels during the follicular phase – will no longer inhibit the GnRH neurons but stimulate them and cause them to switch their release mode from tonic to phasic which leads to a surge in GnRH levels. This also causes a subsequent surge in LH levels known as the “LH surge” which allows for ovulation to occur. These processes are supported by an array of experimental data conducted from animal models in which various physiological states were induced to observe the effects upon the pre-ovulatory E2-induced LH surge. Injections of Estradiol into cultured GnRH neurons has shown the dramatic surge of GnRH that is necessary for the LH surge. Animal models in which the ovaries have been completely removed, were still observed to have an LH surge shortly after E2 injections. Any condition that interferes with the buildup of pre-ovulatory E2 levels has usually prevented the LH surge from occurring and ovulation consequently. Progestin injections into mice have shown to inhibit ovulation, as pre-ovulatory E2 levels fail to rise due to increased inhibition of gonadotropins by the injected progestin. E2 is a major regulator of this pathway, whose manipulation will determine whether an LH surge and ovulation could possibly occur.

References:

