Abstract: The overall goal of our research is to understand how stress disrupts reproductive function and fertility. The impact of stress within our society is widespread; over 75% of Americans report frequently experiencing physical symptoms attributed to stress. In women, stress is considered a major factor in the development of menstrual cycle disorders, amenorrhea, and infertility, affecting 25% of reproductive age women. To date, the neuroendocrine causes of stress-induced infertility are not completely understood. Several pathways within the brain are activated by stress. The hypothalamic-pituitary-adrenal (HPA) axis controls circulating levels of glucocorticoids and is a common and critical response to all stressors. Though glucocorticoids have been considered a key mediator of stress-induced reproductive suppression, little is known about the precise location(s) or mechanism(s) by which glucocorticoids diminish gonadotropin-releasing hormone (GnRH) or gonadotropin secretion, either in response to stress in normal women or in conditions of glucocorticoid excess, such as Cushing’s syndrome. Our recent work suggests that enhanced secretion of corticosterone during stress can disrupt reproductive neuroendocrine function by impairing the regulation of LH pulses and/or the preovulatory LH surge via inhibition of kisspeptin and/or GnRH neuronal activation.

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