

Changes in ovarian hormones, their receptors, and neuroendocrine function as people age

Edward Williamson

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ABSTRACT

The menopause is the irreversible termination of a woman's fertility. The depletion of the ovarian follicle reserve was formerly assumed to be the only explanation for this drastic physiological shift. New evidence from women's studies and animal models leads us to rethink this notion. Multiple pacemakers, according to a growing body of data, play a role in the shift to irregular cycles, decreased fertility, and the timing of menopause. We will present evidence that supports the hypothesis that a dampening and desynchronization of precisely orchestrated neural signals causes miscommunication between the brain and the pituitary-ovarian axis, and that this constellation of hypothalamic-pituitary-ovarian events causes regular cyclicity to deteriorate and heralds menopausal transition. Because the ovarian follicle is not only the source of oocytes but also the primary source of estrogens, which are important for maintaining normal functions such as bone and mineral metabolism, memory and cognition, cardiovascular function, and the frequency of age-related neurodegenerative diseases such as Alzheimer's disease, the end of the

reproductive life span has far-reaching consequences for women. In the face of a relatively stable age of menopause, the significant growth in the average human life expectancy has resulted in an increase in the number of women who would spend a greater part of their lives in a chronic hypoestrogenic condition. As a result, it's more crucial than ever to fully comprehend the systems that regulate the menopausal transition. Furthermore, gerontologists interested in brain ageing will benefit from a better understanding of the mechanisms governing female reproductive ageing, because if the central nervous system is a key pacemaker of reproductive senescence, we may gain a better understanding of the fundamental process of brain ageing. Furthermore, because the female reproductive system undergoes such significant changes early in ageing, we anticipate that this system will allow us to answer crucial questions about the biology of ageing without the confusing pathological alterations that so often sabotage gerontological research.

Key Words: Gerontological research; Hypoestrogenic condition; Alzheimer's disease levels

INTRODUCTION

For many years, it was assumed that the menopause was caused by the exhaustion of the postmitotic endowment of ovarian follicles that is established during embryonic development, and that the hypothalamic/pituitary changes that accompany the menopause were simply a result of declining ovarian function. Several lines of evidence have lately led to the idea that the brain plays a key role in the events that lead to reproductive senescence. Prior to the termination of reproductive cycles, the temporal patterns of brain signals appear to be changed in both people and animal models during middle age, which may contribute to the loss of follicles leading to menopause.

Young women's reproductive axis

The exact functional and temporal integration of hypothalamic, pituitary, and ovarian hormone production results in the pattern of regular ovulatory cycles in normal women. 1 Pituitary production of folliclestimulating hormone (FSH) and luteinizing hormone is stimulated by pulsatile release of gonadotropin-releasing hormone (GnRH) from the hypothalamus (LH). Within the ovary, the two gonadotropin hormones have different but highly intertwined activities. FSH regulates follicular growth and the number of follicles

that reach the preovulatory stage, whereas LH is required for ovulation and the creation of the corpus luteum. LH governs the aromatization of androgenic precursors to estradiol in the granulosa cells, whereas FSH regulates the production of androgens from cholesterol in the theca cells of the growing follicle. Inhibin B and inhibin A, as well as estradiol, are released by growing follicles. Throughout the typical menstrual cycle, LH and FSH are actively and differently controlled. The magnitude and frequency of pulsatile GnRH stimulation, as well as the selective effects of the inhibins on FSH, all play a role in the differential production of LH and FSH from a single cell type inside the pituitary. During the normal menstrual cycle, gonadal steroids dynamically modulate the frequency and quantity of pulsatile GnRH release. Estradiol reduces the amount of GnRH released, but progesterone reduces the frequency of GnRH pulses in the presence of estradiol. The pituitary response to GnRH is further modulated by both gonadal steroids and inhibins. Inhibins, like estradiol and progesterone, play a role in the negative feedback control of the reproductive system by inhibiting FSH secretion selectively. Positive feedback and the LH surge are caused by an exponential increase in estradiol linked with the expansion of the dominant follicle during the follicular phase.

Editorial office, Department of Endocrine Disorder and Surgery, United Kingdom,

Correspondence: Edward Williamson, Editorial office, Department of Endocrine Disorder and Surgery, United Kingdom; E-mail: endocrinesurg@journalsci.org;

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REPRODUCTIVE AGING

Interconnected changes in early ovarian aging

The number of germ cells in the ovary peaks in the middle of pregnancy and then declines dramatically. According to autopsy and surgical specimens, there are 400,000 primary oocytes during puberty, 11,000 at 35 years of age, and just 1100 a decade later. After the age of 35^{6,7}, a decrease in the number of antral follicles observed on transvaginal ultrasonography in normal women is linked to a decrease in fertility. ^{8,9} Early indicators of ovarian age include antimüllerian hormone (AMH), also known as müllerian-inhibiting substance (MIS)^{7,10} and inhibin B^{11,12}. Inhibin B is released constitutively from the granulosa cells of tiny antral follicles¹³, and levels are lower in women 35 years old who have regular ovulatory cycles and FSH levels within the normal range than in women 35 years old who satisfy the same criteria. Although there is little indication that AMH has an endocrine function, inhibin B appears to play a key role in the early phases of ovarian ageing when FSH levels deviate from those of LH. The discovery of reduced levels of inhibin B in combination with greater levels of FSH in women with regular cycles showed that the loss of negative feedback of inhibin B on FSH is linked to ovarian ageing. FSH is regulated not only by inhibin and estradiol, but also by the activin/follistatin system, since activin increases FSH production and secretion while follistatin neutralises it. In some^{14,16} but not all studies, activin.

A levels in blood and follicular fluid are higher in older ovulatory women. ¹⁷ Activin A levels in follicular fluid are likewise higher in older ovulatory women, however this is countered by a shift in follistatin. ¹⁸ Changes in serum activin levels, on the other hand, are uncertain in relation to FSH regulation. Activin A levels rise with age but are unrelated to FSH levels. Furthermore, the highly irreversible binding kinetics of follistatin to activin imply that activin functions in an autocrine/paracrine way rather than as an endocrine hormone. ²⁰ Several research have looked into the maintenance of estradiol levels in the face of other signs of ovarian function decline. Although studies in women older than our cohort demonstrated an increased predisposition to multiple follicle formation, we found no indication of development of more than a single dominant follicle in conjunction with higher estradiol in women older than 35 year. Estradiol concentrations in response to fixed-dose FSH stimulation were identical in older and younger reproductive aged women who had gonadotropin secretion downregulated using a GnRH agonist, leading the authors to conclude that the secretory capacity of recruited follicles is maintained in older reproductive aged women, ²³ and that the higher FSH levels associated with reproductive ageing are responsible for the increased estradiol levels.