Summary

The neuroendocrine control of the onset of puberty in rodents has been extensively reviewed over the years (1–7). In this chapter, we will provide both a brief account of the basic mechanisms underlying the pubertal process in these animals and an update of some recent developments in the field. Because the rat is the animal most extensively studied, we will discuss mainly results obtained in this species, and when available, we will offer the reader a comparison of these results with those obtained in mice.

Key Words: Hypothalamus; EnRH; Transsynaptic communications; Glia-to-neuron; Signaling; Growth factors; Female puberty.
INTRODUCTION

Although rodent puberty has fundamental differences with puberty in primates, the basic mechanisms underlying the process of sexual maturation are well conserved across species. Such mechanisms include, among many others, those governing the transsynaptic and glial control of gonadotropin-releasing hormone (GnRH), the cellular underpinnings of steroid positive and negative feedback, and the hormonal/neural control of gonadal development. The most obvious difference between rodents and primates (including humans) is the apparent absence in rodents [but see (7)] of the gonad-independent, juvenile reduction in gonadotropin secretion that characterizes primate prepubertal development (8). Notwithstanding this difference, rats—like primates—exhibit a diurnal increase in pulsatile luteinizing hormone (LH) secretion at the end of juvenile development (9), indicating that rodents can serve as useful animal models to identify those primary neuroendocrine events that, by causing diurnal changes in episodic LH release, are ultimately responsible for setting puberty in motion.

BASIC ASPECTS OF FEMALE SEXUAL DEVELOPMENT IN RODENTS

Whereas in humans the neuroendocrine control of gonadotropin secretion (as assessed by the detection of pulsatile LH secretion) appears to be fully functional at birth, this is not the case of rats and mice, which are born very immature—at a developmental stage comparable with ∼150 days of human gestational life (10). The gestational period of the rats and mice lasts 21–23 days. The first ovulation in most laboratory rats occurs 35–45 days after birth, but in mice it is highly variable taking place within several days, or even weeks, after vaginal opening (11,12). Canalization of the vagina, which normally is imperforated before puberty, occurs because of estrogenic stimulation and is the only phenotypic change observed in association with the onset of puberty. In rats, vaginal opening usually occurs a day after the first preovulatory surge of gonadotropins (2,3,13,14), and therefore, it coincides with the day of first ovulation; in mice, however, this association is much less evident. Because the first ovulation in mice may occur several days (4–20) after vaginal opening, assessing only this event is not a good indicator that puberty has actually taken place.

Postnatal development of the female rat can be divided into four phases, described in detail earlier (15). They are a neonatal period comprised between the day of birth and postnatal day 7, an infantile period (days 8–21), a juvenile period that ends around day 30–32, and a peripubertal period that culminates with the first ovulation (usually around day 38–40). The end of the juvenile period can be defined as the time when morning–afternoon differences in pulsatile LH release become established (9). Mouse postnatal development can also be considered as composed of the same stages, but in this case the peripubertal period may extend for several additional days because of the dissociation between vaginal opening and first ovulation (11,12).

PREPUBERTAL CHANGES IN GONADOTROPIN SECRETION

In both rats and mice, serum follicle-stimulating hormone (FSH) levels increase between birth and the second week of postnatal life and decline thereafter as the animal progresses through juvenile development (12,16,17). Plasma LH levels, on the