In animals that are kept in temperate latitudes and that are seasonal in their breeding activity, nutritional and photoperiodic signals operate in harmony to time the activation of the gonadotrophin-releasing hormone (GnRH) pulse generator in the brain and trigger puberty. These signals ensure that within the constraints imposed by the circannual changes in the environment, body size and time of puberty are appropriate for the overall success of the reproductive process.

Although photoperiodic influences on the timing of puberty in farmed species occur in sheep (Foster et al. 1986), goats (Deveson et al. 1992), red deer (Cervus elaphus; Webster & Barrell, 1985), cattle (Schillo et al. 1992) and pigs (Paterson et al. 1992), it is the short-day breeders (sheep, goats and red deer) that are most photodependent. If the natural timings of their birth during either the increasing daylength of the spring (sheep, goats) or the long days of early summer (red deer) are followed by natural photoperiodic changes and normal growth rates then puberty usually occurs during the declining daylengths of either the first (sheep, goats) or second (red deer) autumn. Recent studies for all three species, and in particular for sheep and deer, have added to our understanding of the interdependence of nutrition and photoperiod in the timing of puberty. These have varied from empirical descriptions of the effects of restricted growth rates and artificial photoperiods on the activation of the GnRH pulse generator in both the female and male, to mechanistic approaches which attempt to identify the biochemical and molecular signals involved in recording and transmitting metabolic information on nutritional status and photoperiod history to the neuroendocrine axis.

Recent research also involves attempts to identify blood-borne metabolites that mediate the nutritional activation of the GnRH pulse generator. In the case of photoperiod, for which there is unequivocal proof that the mediator of this environmental cue in timing puberty is the pineal indoleamine, melatonin (Yellon & Foster, 1986), research effort is now directed to identifying its receptor sites and mode of action. In this regard the recent observations that sheep and deer possess melatonin receptors during fetal life (Helliwell & Williams, 1991; Helliwell et al. 1991) are particularly interesting in view of the demonstration that the photoperiodic signal received by the fetus in utero can alter age at puberty in both species (Adam et al. 1992a; Helliwell et al. 1992), albeit in ways that are specific for each sex.
Spring-born ewe lambs provide an excellent illustration of the interdependence of size and season on the attainment of puberty. Those grown rapidly achieve puberty in their first autumn and at a younger age and heavier weight than those subjected to transitory periods of food restriction. A protracted period of food restriction from mid-summer to mid-winter, followed by ad lib. feeding to achieve the threshold size for puberty in the subsequent spring, delays puberty until the shortening daylengths of the following autumn (Foster et al. 1985). Autumn-born ewe lambs that are grown rapidly invariably fail to achieve puberty in their first winter and have to wait until the following autumn for the acquisition of the photoperiodic information that is needed for the activation of the GnRH pulse generator (Foster, 1981). This dependence on a photoperiodic cue, inevitably leads to pubertal sizes that vary enormously (32–50 kg in the breed used by Foster et al. 1985). Nonetheless, for a specific photoperiodic regimen there is no evidence that the fatness or protein content of the body are important mediators of sexual maturity in the female lamb (Moore et al. 1985), nor indeed is body weight per se (Suttie et al. 1991a). In the early observations of Keane (1974) the threshold body weight for puberty in spring-born ewe lambs grown at different rates declined from 40 kg in early November to 33 kg in late December, again casting doubt on the concept of a uniform threshold body weight for the attainment of puberty, but not excluding the idea of a critical minimum weight for puberty, as exemplified by Hamilton & Blaxter (1980) in farmed red deer hinds kept in a hill environment. However, in the red deer, if this critical weight is exceeded by means of experimentally-advanced (February) birth dates, puberty is expressed in the first rather than the usual second autumn of life (Adam et al. 1992b).

For spring-born ewe lambs, the key features of the natural photoperiod that interact with unrestricted growth to time puberty at about 30 weeks of age are the initial long days of summer followed by the declining daylength of autumn (Foster et al. 1986). Continuous exposure to long days prevents puberty in the first autumn but does not block it indefinitely (Ebling & Foster, 1988) and the same is true in spring-born lambs exposed continuously to short days. For lambs on short days, exposure to a 10-week period of long days starting at 12 weeks of age or a 5-week period at 17 weeks of age, followed in both cases by a return to short days, initiates oestrus cyclicity at the normal age (Foster et al. 1986). Indeed transitory exposure to long days for as little as 1 week at 21 weeks of age also initiates oestrus at the usual age, albeit somewhat abnormally in that there is a high incidence of short luteal phases (Yellon & Foster, 1985). In contrast, when the transitory period of exposure to long days is imposed at an earlier age (5–10 weeks), it is ineffective, leading Foster et al. (1986) to suggest that at very young ages the circadian melatonin rhythm may not be fully entrained to the light–dark cycle. Sequential observations made by Rodway et al. (1985) showing that the night-time rise in melatonin is most pronounced around the time of puberty provide some support for this suggestion. However, more recent observations of Foster et al. (1989a) indicate that it is not the low-amplitude melatonin rhythm of the neonatal lamb that limits the induction of puberty by seasonal light cues but rather immaturity of the reproductive axis or inadequate somatic development.

An age-dependent response also occurs when spring-born female lambs are exposed to artificial short days or are implanted with melatonin to mimic a short-day effect. For example, the insertion of melatonin implants at 19 weeks of age advanced the time of
puberty by 5-2 weeks but was without effect when carried out at 7-5 weeks (Nowak & Rodway, 1985); for ewe lambs implanted at birth, puberty was delayed by 4 weeks (Kennaway et al. 1986). Similarly in red deer, which reach puberty naturally in their second autumn, Webster & Barrell (1985), Adam et al. (1989), Asher (1990) and Fisher et al. (1990) have demonstrated that there is exquisite sensitivity in the timing of melatonin treatment for the induction of precocious puberty.

In the male lamb, nutritional influences play a similar role to that in the female, with those on high planes of nutrition achieving puberty at younger ages and heavier body weights than those on low planes of nutrition. However, compared with the female, photoperiod plays a relatively minor role in the attainment of puberty in the male (Dyrmundsson, 1987). For example, spermatogenesis begins in spring-born ram lambs during mid-summer, approximately 20 weeks ahead of puberty in the female and before the natural decline in daylength. Using the initiation of a sustained increase in luteinizing hormone (LH) secretion as a more sensitive indicator of puberty, Wood et al. (19916) have demonstrated that neuroendocrine sexual maturity occurs in the male at 5-6 weeks of age under natural photoperiod and is only delayed by approximately 3 weeks under reverse photoperiod.

**GESTATIONAL PHOTOPERIOD AND THE TIMING OF PUBERTY**

The lamb develops a circadian rhythm of melatonin in the first few weeks of life that accurately reflects the ambient photoperiod (Claypool et al. 1989) and appears, on the basis of a shift in prolactin concentrations, to be able to discern changes in daylength imposed as early as 7 d following birth (Ebling et al. 1988). However, there is also evidence of an earlier ability to perceive photoperiodic information. In the pregnant ewe, melatonin of maternal origin crosses the placenta and gives rise to a diurnal pattern of circulating melatonin concentrations in the fetus (Yellon & Longo, 1987, 1988; Zemdegs et al. 1988). In so far as melatonin influences prolactin secretion, fetal prolactin concentrations indicate that the fetus can respond to the maternal melatonin signal (Bassett et al. 1989; Serrón-Ferré et al. 1989). Furthermore, specific melatonin-binding sites have been identified in the brains of fetal sheep (Helliwell & Williams, 1991) and red deer (Helliwell et al. 1991) as early as days 40 and 66 of gestation respectively.

Evidence that the neonate utilizes the maternal photoperiod in its acquisition of a 'photoperiodic history' comes from the profound influence of prenatal photoperiod on prolactin secretion in the newborn sheep (Ebling et al. 1989b) and red deer (Adam et al. 1992c). Thus, while circulating prolactin concentrations in both species at birth reflect the maternal photoperiodic signal, the subsequent response of the newborn to an alteration in photoperiod is governed by the photoperiodic information received in utero. Although there appears to be no effect of gestational photoperiod on the subsequent growth rates of the lamb (Bassett, 1992) or red deer calf (Adam et al. 1992c), evidence is accumulating in both species for an influence on the rate of sexual maturation. Such an influence has been demonstrated in some rodent species with short intervals to puberty, e.g. the meadow vole (Microtus pennsylvanicus; Lee et al. 1987) and the Djungarian hamster (Phodopus sungorus sungorus; Stetson et al. 1986), but up to now results from large animal species have been equivocal. For example, in the ewe lamb, Sunderland et al. (1990) and Herbosa & Foster (1992) found no effect of prenatal photoperiod on the timing of puberty, whereas Helliwell et al. (1992) reported that prenatal long days could
facilitate the induction of puberty by an early postnatal decrease in daylength. Adam et al. (1992a) have demonstrated advanced puberty in male red deer whose mothers were kept in long days as opposed to short days during pregnancy but the same treatment did not affect puberty in contemporary female offspring (C. L. Adam, C. E. Kyle & P. Young, unpublished results). For autumn-born goats Deveson et al. (1992) reported that exposure to long days for a 2-month period before birth, followed by a return to the ambient photoperiod after birth, delayed the timing of puberty in both males and females. The apparent conflict may be due to the variety of photoperiodic regimens used, ruling out direct comparisons between results. For example, the spring-born female lamb receiving adequate stimulatory photoperiodic cues from its natural postnatal exposure to long days followed by short days may not be reliant on its prenatal photoperiodic history. On the other hand, the photoperiodic experience in utero may be more critical in the male since sexual maturation is initiated much earlier and appears to involve prenatal programming by androgens (Wood et al. 1991a).

ACTIVATION OF THE GnRH PULSE GENERATOR

Unravelling the way in which nutrition and photoperiod initiate the rapid LH pulses that trigger puberty is proving a major research challenge. Although the enhanced LH pulsing merely reflects the increased activity of the hypothalamic GnRH pulse generator, the immediate prepubertal timing of this activity does not occur until long after the hypothalamic–pituitary system is capable of sustaining high-frequency LH pulses. For example, female lambs have the ability to produce high-frequency pulses within a few weeks following birth (Foster et al. 1986) yet fail to do so until nutritional and photoperiodic cues are appropriate. Similarly the ovaries of the young lamb are capable of ovulating in response to exogenously-administered gonadotrophin before the normal time of puberty (Dýr mundsson, 1987); so too are the ovaries of older lambs rendered reproductively inactive by either chronic undernutrition or an inhibitory photoperiod (McShane & Keisler, 1991).

Progress towards a mechanistic understanding of the pubertal activation of the GnRH pulse generator comes from studies on ovariectomized female lambs with or without oestradiol implants. This experimental model demonstrates that ovarian oestradiol, via alterations in its inhibitory feedback action on the hypothalamus, plays an important role in mediating the pubertal stimulation of the GnRH pulse generator by nutrition and photoperiod (Foster et al. 1986). During periods of exposure to inadequate nutrition or an inhibitory photoperiod, failure to achieve puberty arises from the maintenance of a heightened sensitivity of the GnRH pulse generator to the negative feedback effect of oestradiol. In the presence of a permissive photoperiodic cue, improved nutrition is accompanied by a rapid decrease in the negative feedback action of oestradiol and the initiation of rapid LH pulses which trigger puberty (Foster et al. 1986). This ability of the hypothalamic–pituitary axis to respond quickly to an improved nutritional status implies that during periods of undernutrition the ewe lamb is capable of monitoring the important pubertal-dependent components of its photoperiodic environment.

In the chronically undernourished ovariectomized female lamb in which puberty is delayed, exogenously administered GnRH at physiological doses (2.5 mg/kg body weight) induces LH secretion, thus proving pituitary competence (Foster et al. 1989b).
Similarly the hypothalamus has an adequate supply of GnRH which can be released by an intravenous injection of N-methyl-D-aspartate (NMDA) which is an agonist of the stimulatory neurotransmitter, glutamate (Ebling et al. 1990b). Provided the nutritionally-restricted ovariectomized lamb has been subjected to the stimulatory photoperiodic cue of long followed by short days during the period of food restriction, its realimentation is accompanied within 1–2 weeks, and in some individuals within 2 d, by increases in the pituitary synthesis of LH as measured by the amounts of mRNA, and increases in LH secretion as detected by circulating plasma concentrations (Landefeld et al. 1989). Interestingly, parenteral infusion of a dextrose-amino acid mixture is as effective as high-plane feeding in sustaining an established pattern of enhanced LH secretion (Foster et al. 1988).

The gonadectomized model has been used also to demonstrate sexual differentiation in the timing of the responsiveness of the hypothalamic–pituitary axis to oestradiol inhibition. Male lambs castrated at 2 weeks of age and implanted with oestradiol exhibit rapid LH pulses at a time (8–11 weeks of age) that coincides with increased testicular growth rates and the onset of spermatogenesis in intact controls (Claypool & Foster, 1990), and some 18 weeks ahead of the decreased responsiveness to oestradiol that coincides with the onset of ovulatory cycles in the female exposed to a stimulatory photoperiod. However, gonadectomy without oestradiol is accompanied by a higher frequency of LH pulses during sexual development in males than females, implying that sexual differentiation may also occur at a steroid-independent level (Claypool & Foster, 1990). While these results suggest a direct drive by the central nervous system on gonadotrophin secretion this may be an oversimplification. A steroid-independent effect on LH secretion occurs in gonadectomized female lambs reared under an inhibitory photoperiod, but in this case their age-related increase in LH pulse frequency in the absence of oestradiol is not accompanied by a decreased responsiveness of their hypothalamic–pituitary axis to oestradiol inhibition (Ebling et al. 1990a).

Recent attempts to unravel the mechanisms controlling the early activation of the GnRH pulse generator in the male have also used the ‘gonadectomized–oestradiol implant’ model and have provided an intriguing insight into the role of prenatal programming in timing puberty. Female lambs born to pregnant ewes that were injected weekly with 100 mg testosterone cypionate from 30 to 90 d of gestation were gonadectomized at 2 weeks of age and implanted with oestradiol. These lambs exhibited precocious development of their hypothalamic–pituitary axis as exemplified by an early increase in LH secretion which coincided with that observed in untreated contemporary males (Wood et al. 1991a).

In all the preceding studies, either the elevation of LH or the increase in its frequency of episodic release are indicative of an increase in the activity of the GnRH pulse generator and the attainment of puberty. However, the associated changes in follicle-stimulating hormone (FSH) have also been investigated. For example, in the study of Landefeld et al. (1989) involving non-oestradiol-treated ovariectomized lambs, the amounts of FSHβ mRNA as well as LHα and LHβ mRNA were increased following realimentation and so too were the circulating concentrations of immunoreactive FSH. Using this same experimental animal model, Padmanabhan et al. (1989) found that, following a period of nutritionally-imposed growth retardation, realimentation was accompanied by increases of 2- and 3-fold in immunoreactive and bioactive FSH respectively. Furthermore, in contrast to the realimentation-induced increases in LH
pulse frequency, FSH pulse frequency remained constant but pulse amplitude increased, with the bioactive pulse amplitudes being almost 60% higher than those of the immunoreactive. More recently, Padmanabhan et al. (1992) confirmed in ovary-intact lambs that puberty was characterized by higher circulating concentrations of bioactive FSH and these were associated with an increase in the proportion of less acidic FSH isoforms in plasma, thereby implying that alterations in FSH heterogeneity may be involved in initiating puberty. In contrast, the exogenous administration of GnRH to growth-restricted ovariectomized lambs did not alter either pituitary or serum FSH heterogeneity (Hassing et al. 1993).

Whether the natural activation of the GnRH pulse generator at puberty leads to heterogeneity of the gonadotrophins that is critical to the attainment of reproductive competence is still a matter of conjecture. It is clear, however, that in order to attain puberty neither nutritional nor photoperiodic cues take priority but both are required to be stimulatory. The chronically-undernourished female lamb has an unimpaired melatonin mechanism to monitor daylength but can only respond to stimulatory photoperiods when her body size is adequate (Foster & Yellon, 1985). Conversely, the stimulatory effects of increased nutrient intake on LH secretion can be prevented by the inhibitory effects of an inappropriate photoperiodic history (McShane & Keisler, 1991). In many instances the activation of the GnRH system by nutrition is very rapid, implicating the existence of some specific blood-borne metabolite that relays information on the shift in nutritional status directly to the hypothalamus. For photoperiodic information a protracted programming period is required and for this there is unequivocal evidence that the mediator is the pineal indoleamine, melatonin.

PHOTOPERIODIC INFORMATION AND THE MELATONIN SIGNAL

Melatonin (5-methoxy-N-acetyltryptamine) is synthesized and secreted in a precise circadian rhythm. Concentrations in peripheral plasma are elevated during darkness and the duration of their normal elevation is directly proportional to the length of the dark phase, thereby providing the animal with a precise record of its photoperiodic environment (Reiter, 1991). In seasonal breeders the accumulated melatonin signal controls the timing of puberty in both long- and short-day breeders. It is, therefore, neither pro- nor anti-gonadotrophic. Although its mode of action in timing the activation of the GnRH pulse generator is not yet known, melatonin receptors have been identified in the pars tuberalis (PT) of the ovine pituitary during both adult (Morgan & Williams, 1989) and fetal life (Helliwell & Williams, 1991). Within the PT the melatonin-responsive cells are secretory in type (Morgan et al. 1991) and in view of the ability of melatonin to influence the nature of their secretions (Morgan et al. 1992) it has been postulated that these may be involved either directly or indirectly in the timing of the activation of the GnRH pulse generator by a permissive photoperiod (Morgan et al. 1992). Following the demonstration by Helliwell et al. (1992) that the prenatal photoperiod of the ovine fetus influences the timing of puberty, it has been suggested by Skinner et al. (1993) from their finding that melatonin inhibits LH-releasing hormone (LHRH)-stimulated LH release in vitro from the fetal ovine PT, that this may represent a mechanism whereby the melatonin signal influences the timing of puberty in the ewe lamb.
Because of the well-recognized inhibition of the GnRH pulse generator by undernutrition and its rapid activation following realimentation, this model is often used in attempts to identify blood-borne metabolites of nutritional origin that act either directly or indirectly on the neuroendocrine system. Concern that the model may reflect non-specific inputs associated with the psychological stress of food restriction, rather than specific metabolic factors related to current nutrition, has been allayed by the recent studies of Schreihofer et al. (1993a,b). Nonetheless, in interpreting the available information it must be appreciated that acute signalling of nutrient status may differ dramatically from the longer-term signalling that is related to growth (Bronson & Manning, 1988) and that normal control mechanisms may become intertwined with pathophysiological mechanisms arising from long-term undernutrition.

Metabolic cues are likely to be metabolic hormones or products of the pathways that they regulate. The alleviation of undernutrition-suppressed LH secretion by increasing feed intake occurs independently of body weight (Foster et al. 1989b; Suttie et al. 1991a) and can be mimicked by parenteral infusion of glucose and/or a mixture of amino acids (T’Anson et al. 1991) or abomasal infusion of a single amino acid, tyrosine (Hall et al. 1992). Insulin, stimulated by glucose administration and suppressed by undernutrition, may be a candidate for metabolic signalling in that it can also influence the availability of neurotransmitter substrates involved in GnRH secretion (Steiner, 1987) and has, in some experiments, led to enhanced ovulation rates in sheep and pigs (for review, see Robinson, 1990). However, its exact role is unclear since LH secretion in the growth-restricted female lamb was not altered by parenteral (Suttie et al. 1991a) or intracerebroventricular (Hileman et al. 1990) infusion of insulin, although the latter route decreased LH pulsatility in lambs fed ad lib.

The obvious association between growth and puberty has led to the postulate that growth hormone (GH) is involved; however, all the available evidence suggests that such a role is not primary. In humans, GH is normally elevated during puberty but its precise action on the reproductive axis remains unclear (Ogilvy & Shalet, 1992). Whereas, in rats, secretion of both LH and GH is diminished by chronic low nutrition (Sisk & Bronson, 1986), LH secretion is reduced but GH secretion increased in the nutritionally growth-retarded lamb (Foster et al. 1989b); and LH secretion increases and GH secretion decreases in the lamb on realimentation. Moreover, there is apparently no pubertal GH rise in female sheep (Suttie et al. 1991b) and administration of exogenous GH fails to alter the timing of puberty (Bucholtz et al. 1990; Suttie et al. 1991b).

The role of the GH-controlled insulin-like growth factor-1 (IGF-1) deserves some attention since circulating levels are elevated during puberty in rodents and primates (Copeland et al. 1982; Handelsman et al. 1987). Furthermore, Hiney et al. (1991) demonstrated a direct stimulatory effect of IGF-1 on LHRH release when administered to prepubertal rats in the region of the GnRH nerve terminals in the median eminence of the hypothalamus, implying that it may be a metabolic signal involved in regulating sexual maturation. However, there is no evidence for a pubertal rise in IGF-1 in female sheep (Suttie et al. 1991b) and the pubertal breeding season of male deer occurs when peripheral IGF-1 concentrations have decreased following a period of elevation associated with the spring–summer growth spurt of red deer (Suttie et al. 1989) and of reindeer (Suttie et al. 1991c). In contrast, IGF-1 is elevated during the steroid-dependent pubertal growth spurt in humans. The significance of these species and/or sex differences in the
temporal pattern of IGF-1 is not clear. However, IGF-1 may have a different role or have its effects masked in seasonal v. non-seasonal breeders, and there are clearly sex differences in the timing of the initiation as opposed to the culmination of pubertal development (Claypool & Foster, 1990). Perhaps of particular relevance to seasonal breeders are the intriguing observations that IGF-1 concentrations are also sensitive to photoperiod. For example, in the reindeer, a short-day breeder, Suttie et al. (1991c) found elevated IGF-1 concentrations during long days and in the hamster, a long-day breeder, Vriend et al. (1988) reported that injecting melatonin to mimic a short-day effect also increased IGF-1 concentrations. Thus in both species elevated IGF-1 concentrations occurred during their inhibitory photoperiod, making it difficult to dismiss a role for this hormone in the nutritional mechanisms that influence the timing of puberty.

A relationship between thyroid function and growth is well established and both hypothyroidism delay puberty in the ram lamb by reducing hypothalamic GnRH release (Chandrasekhar et al. 1985, 1986). However, there is no further evidence that thyroid secretions provide a primary metabolic cue for puberty, although they do play an active role in terminating the breeding season in female sheep (Nicholls et al. 1988) and in initiating sexual quiescence in red deer stags (Shi & Barrell, 1992) since T4 is essential for ensuring the shift in steroid negative feedback that is required to inactivate the GnRH pulse generator (Webster et al. 1991).

NEUROTRANSMITTER MECHANISMS AND THE PUBERTAL ACTIVATION OF THE GnRH PULSE GENERATOR

A number of neurotransmitters have been implicated in the activation of the GnRH pulse generator but those involved specifically in mediating nutritional and photoperiodic information are still a matter of conjecture. Some amino acids can act directly as neurotransmitters or indirectly, following metabolism, e.g. glutamine and its metabolite γ-aminobutyric acid (GABA), tyrosine and the catecholamines. Opioid peptides also act as neurotransmitters and their location in the diencephalon is in keeping with their role in the control of gonadotrophin secretion (Thiéry & Martin, 1991). Neurotransmitter mechanisms involve both inhibitory and stimulatory pathways and it may well be the changing balance between these that determines the timing of puberty. For example, the opioids are involved in GnRH inhibition, glutamine and aspartate in GnRH stimulation and the catecholamines in both.

Elevation of LH concentrations following the administration of the opiate antagonist naloxone is the accepted bioassay for the opioidergic inhibition of the GnRH pulse generator. It is well established that endogenous opioids are involved in the steroid feedback inhibition of GnRH secretion in non-seasonal breeders (Malven, 1986). They are also involved in steroid-dependent LH inhibition in the seasonally-breeding sheep, but only during the sexually active phase of the annual cycle when daylengths are short (Lincoln et al. 1987). However, in the prepubertal ewe lamb pulsatile LH secretion is inhibited by endogenous opioids with or without ovarian steroid feedback (Ebling et al. 1989a) implying the existence of both direct and indirect effects on the pulse generator. Various workers have attempted to correlate changes in opioid inhibition with the pubertal transition but there is insufficient evidence to suggest a causal role. Whereas Rawlings & Churchill (1990) reported an absence of opioidergic LH suppression in very...
young ewe lambs, Ebling et al. (1989a) found no change in responses to naloxone during reproductive maturation. Similarly no pubertal decline in opioid inhibition has been reported for heifers (Wolfe et al. 1992) and bull calves (Rodriguez et al. 1993). The disappearance of opioid inhibition, i.e. absence of effect of naloxone on LH, in the underfed hypogonadotropic prepubertal ewe (Ebling et al. 1990b; Recabarren et al. 1990) and gilt (Cosgrove et al. 1991) further rules out a role for opiates as 'metabolic messengers' during dietary restriction.

Stimulation of the neurostimulatory amino acids, glutamine and aspartate by the agonist, N-methyl-d-aspartate (NMDA) induces increased LH secretion in the prepubertal ewe lamb (l'Anson et al. 1993). This effect is not blocked during nutritional growth restriction (Ebling et al. 1990b) or by inhibitory photoperiod (Jansen et al. 1991; Lincoln & Wu, 1991). However, NMDA does not consistently stimulate LH release, the response being largely ‘all-or-none’ rather than dose-dependent, leading l’Anson et al. (1993) to postulate that both stimulatory and inhibitory pathways may be activated, with the dominant pathway dictating the level of reproductive activity. Onset of puberty may then be associated with increased direct glutamate–aspartate stimulatory neurotransmitter input to the GnRH pulse generator, and reproductive quiescence with the activation of inhibitory neurotransmitters such as opiates (sheep, Lincoln & Wu, 1991; rhesus monkey, Reyes et al. 1991).

The catecholamines are undoubtedly involved in the control of GnRH release (Thiery & Martin, 1991) but whether their role in timing puberty is inhibitory or stimulatory is not clear. Dopaminergic inhibition of LH release has been demonstrated in the prepubertal lamb (Brango et al. 1990), yet increased dopaminergic neuronal activity in the hypothalamus has been linked with increased LH secretion in the pubertal bull calf (Rodriguez et al. 1993). Noradrenergic inputs have been reported to be stimulatory to LH release (Terasawa et al. 1988) whereas serotonergic pathways are inhibitory (Mondragon et al. 1986). Catecholaminergic systems are sensitive to steroids and may be responsible for mediating ovarian steroid inhibition, while steroid-independent effects appear to be mediated by a serotonergic pathway (Meyer & Goodman, 1986). Clearly there are several catecholaminergic pathways that may be involved in the pubertal process; however, their complex interactions, including those with other neurotransmitters such as opioids, have yet to be unravelled. The identification of the metabolic cues and neurotransmitter pathways that transport nutritional and photoperiodic information to the hypothalamic GnRH pulse generator and, thereby, initiate the pubertal process are now major research challenges.

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