Kisspeptins form a family of related peptides codified by KISS1 gene located at chromosome 1q32.1 (1). Their biological actions are carried through specific receptor called KISS1R that belongs to G protein-coupled receptors rhodopsine-like family (2). Isolated mutations in these genes cause hypogonadotrophic hypogonadism in humans, exposing the role of the system in reproductive function (3,4). In this sense, reproductive deficiencies in KISS1R and KISS1 knockout mice prove their role in maintenance of reproductive axis (5-8). Both humans and modified mice do not exist decreases of GnRH content at hypophysary level. These facts suggest that KISS1/KISS1R system regulate GnRH release at hypothalamic level (9). Two neuronal populations expressing kisspeptins have been identified at hypothalamus (10). One of them is present at arcuate nucleus (11) and, there, kisspeptin is colocalized with neurokinin B and dynorphin, creating a neuron population called KND (12,13). The other one appears at anteroventricular paraventricular nucleus (14). Nevertheless, KISS1R is only expressed in gonadotropin-releasing hormone neurons (15). On the other hand, direct neuronal connections have been shown between arcuate nucleus and anteroventricular parvencular neurons and gonadotropin-releasing hormone neurons (16).

Kisspeptins are powerful agonists for releasing gonadotropin (17,18). Kisspeptin administration causes FSH and LH secretion in mice, rats, monkeys and both males and females. LH release kinetic is faster than FSH release kinetic, but this one is longer in time (19-24).

Direct effect of NKB administration over LH release is not clear because it could be under specific hormonal milieu control or specifc specific response (9). However, FSH is clearly not affected by NKB administration (45). Anyway, any possible effect of NKB over GnRH release is produced before KISS1R activation, as experiments in KISS1R knockouts mice demonstrate (46).

REFERENCES