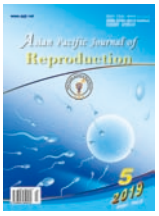

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Hormones in male reproduction and fertility

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Male factor infertility accounts for about half of overall global infertility cases[1]. It involves complex pathophysiological mechanisms[2]. Since male reproductive functions are fine-tuned by orchestrated regulation of reproductive hormones, better understanding of male hormonal imbalance and the influence of their crosstalk with metabolic hormones, neuropeptides, and stress markers, *etc*. will potentiate further research in this direction.

The hypothalamic-pituitary-gonadal (HPG) axis is considered as the parent axis in the regulation of testicular functions. The gonadotropin releasing hormone (GnRH) released in pulsatile fashion from the hypothalamus mediates the synthesis and release of pituitary gonadotropins, namely luteinizing hormone (LH) and follicle-stimulating hormone (FSH). LH and FSH act upon the testicular cells, Leydig cells and Sertoli cells to regulate the main testicular functions, steroidogenesis and spermatogenesis[3]. However, there are several other hormones and factors that act directly on the testis or modulate the actions of the HPG axis, to indirectly influence male reproductive functions. The complex etiopathology of male infertility may be initiated and worsened by any deviation from the normal physiological homeostasis. Inflammation, infectious diseases, varicocele, various metabolic syndrome[4] and conventional hormonal disorders, such as hypothyroidism/hyperthyroidism[5] are the most talked-about pathological conditions that associate with male subfertility/infertility. Factors that lead to the malaise state are again multivariate including both endogenous as well as exogenous factors, most importantly influenced by innumerable lifestyle components[6]. These conditions, either alone or in combination, may lead to disrupted male reproductive hormonal synchronization and thus result in a compromised state of male fertility.

Physiological energy homeostasis finds intricate association with reproductive functions. Energy balance is maintained *via* regulation of food intake behavior, energy expenditure, and storage[7−10]. For example, obese men possess an increased number and size of adipocytes, responsible for abnormally high levels of various adipokines, inflammatory mediators and other hormones. These adipose tissue-derived substances interfere with the concerted regulation of the HPG axis which probably partially explains the mechanism of obesity-induced male subfertility or infertility. Numerous neuropeptides and hormones are involved in the pristine regulation of energy homeostasis as per the endogenous circadian clock. The neurohormones and peptides are in turn regulated by metabolic signals from hormones and several factors of the gastrointestinal system and those from the adipose tissue, to mediate feeding behavior, satiety as well as utilization of energy in other physiological processes[3]. These complex endocrine networks may interfere with hormonal regulation of male reproductive functions, affecting overall semen quality. The mechanism of actions of these hormones are being explored to find link between metabolism and male reproductive functions.

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Individual discussion on the influence of each of the essential hormones on male fecundity is required to provide a complete scenario of endocrine control over male reproduction. In this regard, if metabolic syndrome is considered as one of the most crucial endocrine disruptors; then, leptin may be highlighted first as it acts as a sneaky link between metabolic status and male reproductive axis. It has direct receptor-mediated actions on the Leydig cells and may decrease testosterone synthesis and this may explain why the increased levels of leptin commonly appear in infertile obese men[7]. Moreover, expression of the functional ghrelin receptor in different levels of the HPG axis suggests its influence over the same. It mainly acts to inhibit the secretion of the LH and thereby may hinder proper testicular functions[8]. On contrary, obestatin, an anorectic hormone that aids reversing body weight gain, has been found to be expressed in the male reproductive tissues and reportedly increase testosterone secretions. Thus, obestatin may be a potent endogenous hormone to ameliorate testicular functions in obese men[9]. In addition, adiponectin, a white adipose tissue secretion with antiatherogenic insulin-resistance, and anti-inflammatory properties, is also a potent regulator of the secretion and gene expressions of kisspeptin, GnRH and gonadotropins. Adiponectin thus may be important in the regulations of most of the vital testicular functions, such as steroidogenesis, germ cell proliferation and their coordinated apoptosis, as well as in modulation of testicular redox status and oxidative stress[10].

The diversities in the mode of actions of various hormones and their interactions challenge the understanding of the very basic mechanism of how they alter male fertility parameters. Thus, the present thematic issue aims to provide an updated concept on some of the most important endocrine crosstalk affecting male reproductive functions and their association with male fertility status.

Conflict of interest statement

The authors declare that there is no conflict of interest.

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