ABSTRACT

Polycystic ovarian syndrome (PCOS) is the most common neuro-endocrine disorder of women of reproductive age, characterized by excess androgen, ovulatory dysfunction and polycystic ovaries. PCOS is also linked with several metabolic dysfunctions including type 2 diabetes mellitus, obesity, cardiovascular disorders and psychological co-morbidities, *viz.*, anxiety, depression and mood disorders. Although the prevalence of and the discomfort caused by PCOS is very high, very little is known about its clear patho-etiology. Thereby, the current study was aimed at understanding the status of various regulatory molecules to decipher the neuro-endocrine pathology of PCOS, using rodent model. Letrozole, an aromatase inhibitor, was used for PCOS induction. Results of the present study demonstrate that letrozole is able to mimic reproductive, metabolic and neuro-endocrine characteristics similar to the human PCOS condition. Studies suggest that increased GnRH pulsatility and concurrently elevated LH/FSH ratio may underpin the pathology. Moreover, the pulsatile release of GnRH/LH results from the coordinated actions of steroids, neuro-peptides and neurotransmitters in discrete areas of brain. In this context, the current study clearly demonstrates the involvement of neuropeptides kisspeptin, Neurokinin B, Dynorpin and RFRP3 in steroid-mediated feedback regulation that is hampered in PCOS condition. Furthermore, the current study, for the first time, depicts that along with ovary and adrenal, steroidogenesis is also altered in several areas of the brain, suggesting a putative role of local steroids (neurosteroids) in PCOS pathology. We also aptly demonstrate that increased adrenal androgen, a key feature of PCOS, is due to increased responsiveness of adrenal gland that results into activation of a signalling cascade, leading to overproduction of androgens as well as corticosterone from PCOS adrenals. Further, a neurotransmitter evaluation revealed that the GnRH-stimulatory neurotransmitters are elevated whereas GnRH-inhibitory neurotransmitters are decreased in PCOS condition, which is clearly the cause of increased GnRH/LH release. Additionally, our results indicate that the disease causes a pro-inflamed state of endocrine and neuronal tissues that is linked with altered neuronal signalling and behaviour modulations. Present study concludes that PCOS is associated with an altered brain microenvironment, resulting into neuro-endocrine and psychological complications. ***This is the first study which holistically demonstrates that PCOS is a reproductive disease having clear associations with all other organ systems, thus addressing the different targets which can be explored for a detailed understanding.***

Polycystic ovary syndrome, or PCOS, is the most common endocrine disorder in women of reproductive age. A review of international prevalence of PCOS found that over 2-26% prevalence of PCOS has been documented across various countries. Despite its prevalence, the exact cause of PCOS remains uncertain. Its primary characteristics include hyperandrogenism, anovulation, insulin resistance, and neuroendocrine disruption. The syndrome is named after the characteristic cysts which may form on the ovaries Polycystic Ovary Syndrome, Vaginal smear, albino rats, Testosterone, Pergularia daemia. I. Introduction. The Polycystic Ovary Syndrome (PCOS) is the commonest endocrine disturbance mostly 4% to 12% of the women are affecting in the reproductive age [1,2]. This syndrome has the heterogenous collection of the signs and symptoms that gathered together to the form a spectrum of disorder with the mild representation in some, whilst in others a severe disturbance of reproductive, endocrine and in.Â Polycystic ovary syndrome induction in the rat models can be performed by various methods but hormones are the only active chemical source to induce PCOS [21]. Injecting androgen to the animal is the wise and the successful method for the PCOS induction [22]. Polycystic ovary (PCO) syndrome is a common endocrine disorder of unknown etiology. This condition is a major cause of menstrual irregularity and infertility in women with chronic anovulation. This chapter reviews the literature on methods of producing experimental PCO models in the laboratory rat, and the findings that support their resemblance to the human condition. The principal rat PCO models that have been validated include constant light exposure, hypothalamic lesions, sex steroid-induced models, and the mifepristone (RU486) model. Polycystic Ovary Syndrome / metabolism*. Pregnanediol / metabolism. Pregnanetriol / metabolism.