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**MENSTRUAL ABNORMALITIES AMONG  
EGYPTIAN FEMALE ATHLETES**

**A THESIS**

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**بسم الله الرحمن الرحيم**

**«وعلمك ما لم تكن تعلم**

**وكان فضل الله عليك عظيماً»**

**صدق الله العظيم**

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**INTRODUCTION  
AND  
AIM OF THE WORK**

## INTRODUCTION

Active training and competition may produce many reproductive endocrinological disorders in women. The frequency of oligomenorrhea and amenorrhea in sport women varies in different studies (from eight to eighty percent), but is significantly higher than in ordinary women (*Abraham et al.*, 1982).

Many factors have been postulated to play a role in the development of menstrual irregularities in athletes. This includes weight loss and changes in lean/fat ratio, dietary changes, physical or emotional stress of training and hormonal changes (*Cumming and Belcastro*, 1982).

Because of the increase in the number of women participating in different kinds of sports, it is important to obtain more knowledge about the possible disorders within the reproductive system in women practising different sports.

## **AIM OF THE WORK**

The aim of this work is to study the pattern of menstrual disorders in relation to physical exercise among Egyptian females athletes and ballet dancers in comparison to normal healthy non athletic female controls. This work aimed to study the correlation between menstrual disorders and percentage of body fat.

**REVIEW  
OF  
LITERATURE**

## REVIEW OF LITERATURE

### THE MENSTRUAL CYCLE

The physiological mechanisms involved in the regulation of normal menstrual cycle are the bases for the diagnosis and management of abnormal menstrual function, (*Speroff et al.*, 1989).

The menstrual cycle is a repetitive expression of the operation of the hypothalamic—pituitary—ovarian system, associated with structural and functional changes in the reproductive system, (*Yen*, 1986).

The menstrual cycle can be functionally divided into 3 phases. The follicular phase, which can be subdivided into first half and second half, the ovulatory phase and the luteal phase, (*Hoff et al.*, 1983).

#### Follicular Phase

The follicular phase takes about 10–14 days. The end result of it is one mature follicle ready for ovulation, (*Speroff et al.*, 1989).

The inactive primordial follicles start to grow independent on gonadotropins to the early preantral stage. It is a continuous process throughout approximately 30 years of ovarian function, (*Yen*, 1986).

Folliculogenesis begins in the late luteal phase of the preceding cycle and terminates at the onset of the midcycle gonadotropin surge, (*Goodman and Hodgen, 1983*).

The number of follicles which starts growing each cycle appears to be dependent upon the residual number of inactive follicle, (*Peters et al., 1975*).

It is clear that the follicle which destined to ovulate is recruited in the first few days of the cycle, (*Mais et al., 1986*).

With follicular recruitment the granulosa cells become cuboidal in shape. In response to follicle stimulating hormone (FSH) small gaps develop between the granulosa cells and the oocyte for its nutrition (*Vermesh and Kletzky, 1987*).

The selection of a single follicle destined to ovulate is inherently related to its capacity for oestrogen biosynthesis, (*Hillier et al., 1981*).

*Hsueh et al.* (1983) found that the integrity of follicular oestrogen production depends on the interaction between the theca and granulosa cells.

Ovarian follicular development involves a synergistic interaction between gonadotropins; FSH and Luteinizing hormone (LH) and steroids; androgens and oestrogens on the theca granulosa cells. Through hormonally induced changes of specific receptors within the follicles, (*Yen, 1986*).

### FSH and Granulosa Cells:

In the ovary, granulosa cells of all follicles possess receptors for FSH. Increasing levels of FSH induce an increase in numbers of FSH receptors. This is an early event in the process of follicular growth, (*Dorrington and Armstrong, 1979*).

Also, FSH induces granulosa cells to acquire an aromatizing enzyme. This provides the essential step for oestradiol production from androgens. Oestradiol appears to have the ability to increase the number of its own receptors, (*Hsueh et al., 1983*).

Follicular growth is promoted by the synergistic effect of FSH and oestradiol. This cause intraovarian positive autoregularity feedback mechanism leading to rapid division of granulosa cells, (*Yen, 1986*).

Oestradiol initially promotes the ability of FSH to increase and maintain FSH receptors. Later oestradiol enhances the ability of FSH to increase and maintain LH receptors, (*Welsh et al., 1983*).

In the developing follicle while FSH in the presence of oestradiol increases the receptors for FSH and LH in the granulosa cells, LH in association with luteinization induces a reduction of receptors for FSH, LH and oestradiol. This may be important for follicular atresia, (*Richards et al., 1976*).

### LH and Theca Cells:

The theca interna develops specific receptors for LH but not for FSH leading to the biosynthesis of androgen, (Yen, 1986).

The increased androgen produced by the thecal compartment diffuses into the follicular fluid and is aromatized to oestrogen by the granulosa cells, (McNatty *et al.*, 1979).

Fletcher and Greenan (1985) found that not every cell has to contain receptors for the gonadotropins. A signal can transfer by the cells which contain receptors causes protein kinase activation in cells which lack receptors.

The granulosa cells of the preantral follicles possess the ability of converting androstenedione to more potent androgens rather than oestrogen, (McNatty *et al.*, 1979). This is due to the presence of 5  $\alpha$ -reductase, and the formation of non aromatized androgen dihydrotestosterone (DHT) from testosterone, (Hillier *et al.*, 1980).

The fate of the preantral follicle is in delicate balance. At low concentration androgens enhance their own aromatization and leading to oestrogen production. At higher levels aromatization capacity is limited and the follicle become androgenic and atretic, (Erickson *et al.*, 1985).

The success of a follicle depends upon its ability to convert androgen microenvironment to an oestrogen microenvironment, (Chabab *et al.*, 1986).

Under the effect of the combined action of oestrogen and FSH there is an increase in the production of follicular fluid. These occur as the follicle is transformed gradually to the antral stage, (*Speroff et al.*, 1989).

### The Antral Follicle:

In the follicular fluid oestrogen becomes the dominant substance in the presence of FSH, while in its absence androgens predominate, (*McNatty et al.*, 1980).

Antral follicles with the greatest rates of granulosa proliferation contain the highest oestrogen concentrations, the lowest androgen/oestrogen ratios. And are the most likely to contain a healthy oocyte, (*Speroff et al.*, 1989).

The steroids in follicular fluid are present in concentration higher several times than those in plasma. This indicates the functional capacity of the surrounding granulosa and theca cells. Steroid hormones synthesis occur within the follicle by the two-cell mechanism, (*Erickson et al.*, 1985).

The interaction between the granulosa and theca compartment which results in accelerated oestrogen formation appears to be fully functional in the late stage of antral development, (*McNatty et al.*, 1979).

In the antral follicle and under the effect of LH, theca compartment produce androgens, which can be converted to oestrogen in the granulosa compartment through FSH-induced aromatization, (*Speroff et al.*, 1989).

Granulosa cells of large antral follicles have marked aromatase activity which converts the androgens to oestrogens. A shift from the androgenic microenvironment to an oestrogenic microenvironment may be the major determinant for the auto regulation in the selection and maintenance of the dominant follicle, (*Hillier et al.*, 1980).

### Selection of the Dominant Follicle:

*Speroff et al.*, (1989) noted that the selection of the dominant follicle is the result of two oestrogen actions. A local interaction between oestrogen and FSH within the follicle, and the effect of oestrogen on pituitary secretion of FSH. Oestrogen has a positive feedback with FSH within the maturing follicle, and a negative feedback with FSH at the hypothalamic-pituitary level. This lead to withdrawal of gonadotropin support from the less developed follicles. Resulting in limited oestrogen production followed by a decline in FSH-dependent aromatase activity. Interruption of granulosa proliferation and function leading to an androgenic microenvironment and irreversible atretic change. The negative feedback of oestrogen inhibits the growth of all follicles except the dominant follicle. The selected follicle remains dependent upon the FSH and must complete its development in spite of decline in plasma level of FSH. This occur by its own accelerating oestrogen production and due to its high intrafollicular oestrogen concentration leading to enhancement of FSH action induced aromatization.