Effect of Furostanol Glycosides from *Trigonella foenum-graecum* on the Reproductive System of Male Albino Rats

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**Trigonella foenum** (TF) has long been used in the traditional Indian systems of medicine for the treatment of various ailments. The objective was to study the anabolic and androgenic activity of the furostanol glycosides fraction of *Trigonella foenum-graecum* (Fenu-FG) in immature castrated male Wistar rats. It was also aimed to investigate the effect of Fenu-FG on testicular histology in non-castrated immature rats. The animals (55 ± 5 g) were castrated. The rats were treated with either vehicle, testosterone (10 mg/kg s.c. bi-weekly) or Fenu-FG (10 and 35 mg/kg p.o.) once daily for 4 weeks. At the end of the study, blood was withdrawn, serum testosterone and BUN were measured. Animals were killed and reproductive organs were excised and weighed.

Fenu-FG (35 mg/kg p.o.) and testosterone (10 mg/kg, s.c. biweekly) increased the weight of the levator ani muscle as well as body weight. Fenu-FG (10 or 35 mg/kg p.o.) did not change the testosterone level in castrated rats. Histopathological examination of the testis of non-castrated rats treated with Fenu-FG (10, 35 mg/kg p.o.) showed normal architecture of the testis. Fenu-FG (35 mg/kg p.o.) showed anabolic activity without androgenic activity. Copyright © 2010 John Wiley & Sons, Ltd.

**Keywords:** furostanol glycosides fraction of *Trigonella foenum-graecum*; androgenic; anabolic; castration; testosterone.

**INTRODUCTION**

Male sexual dysfunction (MED) is a common sexual disorder of desire, arousal, orgasm and sexual pain. It is estimated that about 40 million men suffer from erectile dysfunction, 52% of which are between the age of 40–70 years (Aung et al., 2004). Erectile dysfunction is the most common cause of male sexual dysfunction (Dinsmore and Evans, 1999) and has been shown to compromise the overall quality of life, and also results in depression, anxiety and loss of self esteem (Hedon, 2003; Latini et al., 2002). It may also signal underlying disease including diabetes, hypertension and cardiovascular disease (Behr-Roussel et al., 2005; Kang et al., 2004; Kushiro et al., 2005; Lau et al., 2005; Murray et al., 1992; Papatsoris and Korantzopoulos, 2006).

The so-called male menopause (andropause) is an old age disease associated with the loss of libido, loss of muscle mass and associated with decreased testosterone production (Gould and Petty, 2000; Margolese, 2000; Utiger, 2003). As the male body ages, gonadal function slowly declines with a resulting drop in serum testosterone of approximately 1% per year after age 30, a phenomenon that occurs in both males and females (Morales et al., 2000). This can lead to multiple clinical manifestations such as a decrease in bone mass, learning fractions, erectile functions, haematopoiesis, muscle mass and strength. On the other hand, fat mass increases.

Both medical and surgical treatment modalities are available for treating sexual dysfunction. Androgen (especially testosterone) replacement has been found to be effective in the restoration of these conditions (Adimoejia and Adiakhan, 1997; Antonio et al., 2000; Gauthaman and Ganesan, 2008; Heaton and Varrin, 1994; Mulhall et al., 2004; Schiavi et al., 1997; Tyagi et al., 2008; Yildirim et al., 1997). Androgenic is the term for any compound that stimulates or controls the development and maintenance of masculine characteristics in vertebrates. Anabolic agents increase protein synthesis within cells, which results in the build up of cellular tissue (anabolism), especially in muscles. The term adaptogen is used by herbalists to refer to a natural herb product that is proposed to increase the body’s resistance to stress, trauma, anxiety and fatigue.

However, despite the increasing availability of effective conventional medical treatments, plant-derived and herbal remedies continue to be a popular alternative for men seeking to improve their sex life (Aung et al., 2004; Neychev and Mitev, 2005).

*Trigonella foenum-graecum* Linn (Family: Fabaceae), also known as fenugreek, is an aromatic annual plant, 30–60 cm tall, found wild in Kashmir, Punjab and the upper Gangetic plains and widely cultivated in many parts of India. It is used internally as an abortifacient (Nath et al., 1997), antispasmodic, appetite stimulant, blood cleanser, laxative, tonic (Ziyat et al., 1997) and expectorant (Santillo and Dharmananda, 1984). It is also indicated externally for abscesses, boils.
galactogogue (El-Kamali and Khalid, 1996) and for its demulcent, and emollient (Santillo and Dharmananda, 1984) properties. The seeds contains the diosgenin (Taylor et al., 2002) along with three minor steroidal saponins (similagenin, savsalpogenin and yuccagenin), choline, trimethylamine (a sex hormone in frogs), vitamins (A, B₃, B₆, B₁₂, D), lysine and l-tryptophan rich proteins, mucilaginous fibre, coumarin, fenugreekine, nicotinic acid, sapogenins, phytic acid, scopolin and trigonelline (Billaud and Adrian, 2001), calcium, iron, β-carotene and other vitamins and essential oils (Yadav and Sehgal, 1997).

In ethnobotanical literature, aphrodisiac properties of fenugreek seeds are also reported (Santillo and Dharmananda, 1984). Traditional Chinese herbalists used fenugreek for male reproductive issues (Basch et al., 2003). Fenugreek seeds are also assumed to have restorative and nutritive properties (Moissides, 1939; Rosengarten, 1969) and to stimulate the digestive process. In India, traditionally fenugreek seed ground with the jaggary is given to females after childbirth to develop their muscles and strengthen them (Mittal and Gopaldas, 1986).

In the past, many furostanol glycosides from a variety of plants have been shown to have adaptogenic (Vasil’eva et al., 2003, 2005, 2000), anabolic (Dubinskaia et al., 1998) or androgenic (Park et al., 2006) activity especially as vitalizers to improve the sexual function in men. Furostanol glycosides from Tribulus terrestris and its role in the management of male erectile dysfunction has been shown many times (Gauthaman and Ganesan, 2008; Gauthaman et al., 2003; Neychev and Mitev, 2005) and these effects are attributed mainly to protodioscin and related compounds (Gauthaman et al., 2002, 2003). The defatted seeds are a rich source of saponin-rich fractions (Hardman, 1969).

On one hand, diosgenin (an important precursor for the synthesis of a number of sex hormones including testosterone and estrogens) is reported to be present in Trigonella foenum-graecum (fenugreek) seeds (Aradhana et al., 1992), while on the other hand, saponins (especially protodioscin-like compounds) are also present (Hibasami et al., 2003; Yang et al., 2005). Therefore, a glycoside-rich fraction of fenugreek seeds is worth investigating for possible androgenic and anabolic activity. In this paper, the objectives of the study are to isolate the furostenediol glycoside fraction of Trigonella foenum-graecum (fenugreek) seeds and to evaluate the androgenic and anabolic activity on the male reproductive system in castrated rats. It was also aimed to study the effect of the fraction on the cytoarchitecture of the testis of non-castrated rats for any deleterious effects.

**MATERIALS AND METHODS**

**Animals.** Immature male Wistar rats in the weight range 55 ± 5 g were purchased from the National Toxicology Centre, Pune, India and used for the study. They were maintained at a temperature of 25 ± 1°C and relative humidity of 45% to 55% under a 12:12 h light–dark cycle. The animals had free access to food pellets (Chakan Oil Mills, Pune, India) and water was given *ad libitum*. The experimental protocol was approved by the Institutional Animal Ethics Committee (IAEC) of Poona College of Pharmacy, Pune, India, constituted under the Committee for the Purpose of Control and Supervision of Experiment on Animals (CPCSEA), India.

**Drugs and chemicals.** Testosterone (Himedia Laboratories, India), and anaesthetic ether (TKM Pharma, India) were purchased. The chemicals used for preparation of Fenu-FG were of analytical grade.

**Authentication of plant.** The seeds of fenugreek seeds were authenticated by Dr A. M. Mujumdar, Department of Botany, Agharkar Research Institute, Pune, India and a voucher specimen was deposited at that Institute.

**Preparation of furostanol glycoside fraction (Fenu-FG).** The defatted seeds of fenugreek were extracted with alcohol:water (1:1) mixture, concentrated and dried under a vacuum evaporator. This dry powder was then dissolved in 6 volumes of deionized water to remove insolubles. The clear filtrate was passed through a XAD-8 (Rohm and Haas) column. The column was eluted with methanol: water (1:1) gradiently and the eluents monitored with the TLC system comprising toluene: ethyl acetate: methanol: water (ratio of 7:2:1:0.5). The glycoside fractions were identified using a methanol sulphuric acid spray reagent. The single spot fractions were collected and pooled together, concentrated under vacuum to give a free-flowing powder. The presence of furostanol in the fraction was confirmed by HPLC. The fraction was named as Fenu-FG (also known as Andropeak) and used for biological investigations after making solutions in distilled water.

**HPLC details.** The column was a Kromosil reverse phase C-18 (250 × 4.6 mm with 5 μm particle size). The mobile phase gradient was (A) water and (B) acetonitrile according to the following profile: at 0 min, (75% A and 25% B), at 20 min (65% A and 35% B). Flow rate: 1 mL/min. Detector: UV (205 nm).

**Castration of rats.** Immature male Wistar rats (55 ± 5 g) were castrated by the method described by Ottani et al. (2002). A small transversal incision was made in the skin on the ventral side of the symphysis. The testis in the scrotum was pushed gently into the abdominal cavity. The epididymal fat pad was grasped with the forceps and the testis with the epididymis was pulled out from the abdominal opening. The testis together with the epididymal fat pad was cut. The skin wound was sutured with one or two sutures. The animals were housed individually during the recovery period.

**Anabolic and androgenic activity in castrated rats.** Immature male Wistar rats weighing 55 ± 5 g were anaesthetized by anaesthetic ether and castrated as described above. After a recovery period of 2 days the rats were divided into following groups (I) Vehicle treated, (II) Fenu-FG (10 mg/kg p.o.), (III) Fenu-FG (35 mg/kg p.o.) and testosterone (10 mg/kg in sesame oil suspension, s.c. bi-weekly). After 4 weeks of treatment, the animals were anaesthetized with anaesthetic ether, blood was withdrawn by retroorbital puncture and analysed for biochemical parameters. The animals were killed by an overdose of anaesthetic ether and the

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seminal vesicles, the ventral prostate and the musculus levator ani were carefully dissected and weighed. The body weights of the rats were recorded at the beginning and end of the experiment.

An increase in weight of the seminal vesicles and ventral prostate indicates androgenic activity, whereas a gain in weight of the musculus levator ani was considered to indicate anabolic activity (Eisenberg and Gordan, 1950).

Effect on serum testosterone and blood urea nitrogen. The blood was withdrawn, centrifuged and analysed for blood urea nitrogen (BUN), using a Randox kit, by the kinetic method. Serum testosterone was measured by a chemoluminescence kit (Abbott and DPC).

Effect of Fenu-FG on histopathology of testis in non-castrated rats. Non-castrated male Wistar rats weighing 120–160 g were divided into the following groups: (I) vehicle (distilled water, p.o.) treated, (II) Fenu-FG (10 mg/kg p.o.), (III) Fenu-FG (35 mg/kg p.o.) and (IV) testosterone (10 mg/kg in sesame oil suspension, s.c. bi-weekly). After 4 weeks of treatment, the animals were killed. The testis from each group were removed and placed in 10% formalin solution for 24 h. The organ specimen was subjected to dehydration by placing it in xylene three times (1 h each) and later in 70%, 90% and 100% alcohol respectively, each for 2 h. The infiltration and impregnation was carried out by treatment with paraffin wax twice, each time for 1 h. Paraffin wax was used to prepare paraffin L moulds. Specimens were cut into sections of 3–5μm thickness and stained with haematoxylin and eosin. Mounting of the specimen was carried out using distrene pthalate xylene (DPX).

Statistical analysis. Data for each of the parameters were analysed by one-way ANOVA followed by the Bonferroni post hoc test using Prism software version 4.03 (Graph Pad Inc., USA).

RESULTS

The high-performance liquid chromatogram obtained of Fenu-FG showed the presence of one major peak at 3.2 min (furostanol glycoside saponins, labelled as SAP 1) exhibiting 63.3% purity.

Anabolic activity of Fenu-FG

Treatment with testosterone (10 mg/kg, s.c., bi-weekly for 4 weeks) increased the body weight of rats significantly (p < 0.001) from 151.6 to 254.2 g (68.94% increase), whereas the weight of Fenu-FG (35 mg/kg) treated rats was increased from 151.6 to 193.8 g (28% increase). The increase in body weight of Fenu-FG (10 mg/kg) treated rats was not significant compared with the vehicle control (Fig. 1).

Testosterone significantly increased the seminal vesicle, ventral prostate and levator ani weight but not the penis weight (Fig. 2). Fenu-FG (35 mg/kg, p.o.), on the other hand, significantly (p < 0.001) increased the weights of the levator ani muscle but failed to increase other organ weights (Fig. 2). At the lower dose, Fenu-FG (10 mg/kg) did not increase organ weights.

Effect on serum testosterone of castrated rats

Testosterone treatment caused significant (p < 0.001) increases in serum testosterone level, whereas Fenu-FG (10 or 35 mg/kg p.o.) did not increase the serum testosterone level compared with the control (Fig. 3).

Effect on blood urea nitrogen (BUN)

The BUN in castrated rats was 33.44 mg/dL in the vehicle treated group (Fig. 4). The BUN was found to
be 18.8 mg/dL in both Fenu-FG (35 mg/kg, p.o.) and testosterone (10 mg/kg, s.c. bi-weekly) treatment groups. The Fenu-FG (35 mg/kg p.o.) and testosterone treated groups showed significant decreases in BUN compared with the vehicle group (p < 0.01). However, the lower dose of Fenu-FG (10 mg/kg p.o.) did not cause a significant change in BUN compared with the vehicle treated group (Fig. 4).

**Histology of testis in uncastrated rats**

The testes of rats treated with either vehicle, testosterone or Fenu-FG showed no change in normal features with successive stages of transformation of the seminiferous epithelium into spermatozoa with no sign of atrophy or toxicity with respect to pachytene spermatocytes, germ cells, Leydig cells or Sertoli cells (Fig. 5).

**DISCUSSION**

Usually strength enhancement and an increase in lean muscle mass are the result of increased anabolic activity. One way to increase anabolic activity is to increase endogenous (self-produced or produced naturally
within the living body) androgenic hormone, e.g. testosterone.

Anabolic agents induce positive nitrogen (N) balance in living organisms. Measurements of nitrogen excretion were made in castrated rats fed with a liquid diet and in nitrogen balance. The castrated male rat serves as the most sensitive model for nitrogen retention. Another bioassay for anabolic activity involves measurement of the increase in weight of the levator ani muscle in rats upon oral administration of an anabolic agent. This measure of myotrophic activity effect correlates well with the nitrogen retention bioassay and the two are usually performed for the determination of anabolic activity (Vogel, 2002, 2008). In the present investigation, the low dose of Fenu-FG (10 mg/kg) was ineffective as an anabolic agent, however, the higher dose of Fenu-FG (35 mg/kg) significantly increased the weight of the levator ani muscle without increasing the BUN (similar to exogenous testosterone (10 mg/kg) treatment) indicating anabolic activity.

Testosterone is a cholesterol-based steroid hormone (Griffin and Wilson, 2003). Apart from desire that is essential for the initiation of sex, penile tumescence and rigidity as well as the accessory muscles that help to provide additional penile rigidity and ejaculation are also dependent on androgen for normal sexual activity.

A physiologically low level of androgen, as seen in hypogonadism, is associated with decreased sexual desire and activity (Morales and Heaton, 2001; Rabkin et al., 1999). Testosterone treatment was reported to restore both sexual behaviour and penile erectile capacity in castrated rats (Aversa et al., 2000; Mills et al., 1996, 1992) and patients (Settel et al., 2004). Castration leads to a low androgenic status affecting structural, biochemical, pharmacological or any of these components of erectile physiology, which in turn cause a reduction in erectile function as observed from studies on various animal models (Hart et al., 1983).

A potential risk of testosterone replacement therapy is an increase in the incidence of prostate cancer. Furthermore, it is postulated that testosterone that is converted peripherally to dihydrotestosterone (DHT) by 5-alpha reductase is responsible for this risk (Petrov, 1986) DHT has a role in the growth of prostatic tissue and therefore can influence lower urinary tract symptoms. Testosterone is aromatized to form estradiol (E2) in fatty tissue and leads to increased aromatase activity, resulting in increased levels of estradiol (Hudak et al., 2006).

Endogenous testosterone in the circulation can be free (unbound), weakly bound to albumin, or tightly bound to sex hormone binding globulin (SHBG). The free and albumin-bound testosterone is available for use by the body. The largest percentage, however, is bound to SHBG and is unavailable for use in the body (Kaaks et al., 2003). Any condition that increases SHBG will decrease the amount of available testosterone. This includes conditions that elevate estrogens, elevated thyroid hormone and aging. Age-related low testosterone has features of both primary and secondary hypogonadism, and is associated with decreased total testosterone, i.e. free testosterone plus protein-bound testosterone (Nieschlag et al., 2000), decreased free testosterone associated with an increased sex-hormone binding globulin (SHBG) (Harman et al., 2001), decreased secretion of testosterone in response to human chorionic gonadotropin (Nieschlag et al., 1982). The results obtained in the present study showed that fenugreek furostanol glycoside possessed anabolic activity (increased muscle mass) without affecting total serum testosterone levels in castrated rats. The probable mechanism for this action may be due to increased availability of testosterone by dissociating it from the stored form, i.e. SHBG. This higher quantity of available testosterone may be utilized for androgenic and anabolic activity.

Furostanol glycosides are the major soluble saponins that are present in fenugreek. Saponins constitute a very heterogeneous group of substances which are preferentially found in plants. These glycosides consist of sterols or triterpene ring with attached sugars, which involve particular tensile active properties well known for complexing cholesterol in the cell membrane (Price et al., 1987). Subchronic oral treatment with purified sterol saponins extracted from fenugreek seeds increased the food intake in rats. Fenugreek seed extract fed to rats induced a clear cut hyperinsulinaemia, while steroidal saponin treatment did not modify plasma insulin and blood glucose levels in rats (Petit et al., 1993). The extract containing saponin has been reported to inhibit taurocholate and deoxycholate absorption in a dose dependent manner (Stark and Madar, 1993).

CONCLUSIONS

Furostanol glycosides isolated from fenugreek (Fenu-FG) appear to impart anabolic activity in male rats.

Acknowledgements

The authors would like to acknowledge Dr S. S. Kadam, Vice-Chancellor and Dr K. R. Mahadik, Principal, Poona College of Pharmacy, Bharati Vidyapeeth University, Pune, India for their support and encouragement and Sunil Bhaskaran, MD, Indus Biotech Private Limited, Pune for providing necessary facilities to carry out the study.

Conflict of Interest

The authors have declared that there is no conflict of interest.

REFERENCES


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