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Ganoderma lucidum ethanolic extract for the treatment of androgenic alopecia in rats with testosterone-induced Baldness.

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Abstract

Background: Alopecia is a widespread hair loss condition affecting numerous individuals worldwide. This study aims to investigate the potential hair growth-promoting properties of a preparation containing an ethanolic extract of Ganoderma lucidum and its primary components in a model of testosterone-induced alopecia.

Aim: The objective of this research is to assess the effects of Ganoderma lucidum extract on hair regeneration using a testosterone-induced alopecia model.

Materials and Methods: The study was conducted using five groups of rats: negative control group (n = 6), positive control group (n = 6), testosterone plus minoxidil 2% (n = 6), testosterone and ethanolic extract of Ganoderma lucidum extract (10 mg/kg) (n = 6), and testosterone and ethanolic extract of Ganoderma lucidum extract (15 mg/kg). To induce alopecia, subcutaneous testosterone (1 mg/kg SC) was administered daily to all groups except the negative control group for 21 consecutive days. After 21 days, Anagen/telogen (A/T) ratio and the number of follicles were measured and recorded.

Results: The standard group, treated with minoxidil 2%, showed higher anagen/telogen (A/T) ratio, follicular density, and hair length compared to the positive control group. The groups treated with ethanolic extract of Ganoderma lucidum at 10mg/kg and 15mg/kg displayed increased anagen/telogen ratio and improved hair follicle quantity and morphology compared to the positive control group. Furthermore, the group treated with 15mg/kg of the extract showed more favorable outcomes compared to the 10mg/kg group. These results were comparable to the effects of the commonly prescribed drug, Minoxidil 2%, used to promote hair growth in individuals with androgenetic alopecia.

Conclusion: The preparation containing an ethanolic extract of Ganoderma lucidum, especially at a concentration of 15mg/kg, demonstrated significant hair growth-promoting effects in the testosterone-induced alopecia model. Our findings suggest the potential of Ganoderma lucidum extract as a possible treatment for alopecia and merit further exploration for therapeutic applications.

Keywords: absorption, bioavailability, Nanocarriers, peptide, permeability, protein drug delivery.

INTRODUCTION

Hair, along with sweat glands, sebaceous glands, and nails, is one of the vital body parts that emerges from the skin's ectoderm. Hair is classified as an accessory structure of the integument and serves as protective appendages on the body. It's been known for more than 2000 years that hair loss is a dermatological issue that affects 0.2% to 2% of people globally [1].

Hair receives a lot of aggression, thus it's possible that different diseases are interfering with its normal health. Alopecia is a dermatological condition that has been known for over a thousand years and is a frequent issue in both cosmetics and primary healthcare settings [2] .

The phrases "androgen" and "genes" are combined to form the term "androgenetic alopecia" (AGA). It is widely used to describe the systematically occurring loss of scalp hair in both men and women who are genetically prone. The most typical kind of hair loss is this one. The development of AGA is influenced by a variety of hereditary and environmental variables. Genetically vulnerable terminal hairs in the affected area shrivel into vellus hairs in androgenetic alopecia, which is predominantly caused by androgen [3]. Androgens are hypothesised to speed up the telogen-to-anagen transition by causing more hair follicles to enter the catagen and telogen phases and shortening the anagen phase. The terminal follicles turn into vellus-like follicles as a result of the follicular miniaturisation brought on by androgens, creating thinner and shorter hair [4].

In this circumstance, levels of 5"-dihydrotestosterone (5-"-DHT) and 5"-reductase activity are both elevated. Along with a number of inherited and environmental factors, androgens are one of the most common causes of alopecia. The hormone androgen eventually causes the normally large scalp hair follicles to shrink. There are numerous strategies for regulating the growth of androgen-dependent hairs. By reducing androgen production, preventing testosterone (T) from converting into 5-"-DHT, or by inhibiting androgen receptors [5,6].

When 5-"-DHT attaches to the androgen receptor in scalp hair follicles that are sensitive, the hormone-receptor complex activates the genes that cause the terminal hair follicles to gradually shrink in size[7]. As hair cycles progress, the duration of the anagen phase shortens and hair follicles miniaturise, resulting in shorter, finer hairs that inadequately cover the scalp11. The distinctive feature of AGA is these tiny hairs in a range of lengths and diameters [8]. Only two drugs, oral drugs like Finasteride and topical drugs like Minoxidil, are now USFDA-approved to treat AGA in men (exclusively for central/vertex hair loss) and women (only for female pattern hair loss). There are now treatment and management options for androgenetic and biological response modifiers. However, due to their low success rate and related side effects, they have restricted therapeutic usage.[9] Natural ingredients are being used more frequently in cosmetics, and several plant extracts have been investigated for their ability to encourage hair development. In the traditional Indian medical system, a variety of plants and herbal remedies have been asserted to promote hair development and enhance the condition of hair, but their use is constrained due to a dearth of trustworthy scientific proof [10].

Ganoderma lucidum has been utilised in Far Eastern traditional medicine for thousands

of years. In China, Japan, and other Asian nations, the oriental fungus Ganoderma lucidum has a long history of use to extend life and improve health. Reishi or Lingzhi are two of its common names. On the outside, it has a huge, black, glossy, and woody mushroom. Because of the varnished appearance of the mushroom's surface, the Latin word lucidus, which meaning "shiny" or "bright," was coined [11].

" Ganoderma lucidum has exceptional therapeutic action thanks to its anticancer, antiallergenic, antiviral, hepatoprotective, antioxidant, immunomodulator, hypotensive, hypoglycaemic, anti-inflammatory, antithrombotic, and many other health effects. In addition, natural germanium (Ge), lectins, polysaccharides, triterpenoids, polysaccharide-peptide complexes, and -glucans are present. derived from the ganoderma plant's mycelia, which also contain a number of advantageous medicinal characteristics.[12] Currently, ganoderma lucidum is used as an alternative adjuvant in the treatment of diabetes, hepatitis, cancer, leukaemia, and cancer. Cultivation on solid substrates, stationary liquid medium, or by submerged cultivation has become a crucial component to meet the driving force towards the rising demands in the global market because the macrofungus is extremely rare in nature and not enough to be commercially exploited for life-saving therapeutic emergencies. [13]

Recently discovered triterpenoids from ganoderma spores and mycelium have caught the attention of chemists, among them Ganoderic acid.[14] Triterpenoid and Ganoderic Acid (Ganoderic Acid TR&B) has the ability to bind to the androgen receptor (AR) and has inhibitory action against the enzyme 5'-reductase, which allows it to block androgen. The steroid enzyme 5-reductase also changes the hormone testosterone into the androgen dihydrotestosterone, as we have already discussed. testosterone is converted into the more potent androgen dihydrotestosterone by the membrane-bound NADPH-dependent enzyme 5-reductase. Ganoderma lucidum (GL), a natural treatment for lowering elevated testosterone levels, has also been found to have potential for promoting hair growth.[14] The goal of the current study is to demonstrate GL's effectiveness in treating T-induced alopecia.[15]

Natural ingredients are being used more frequently in cosmetics, and several plant extracts have been investigated for their ability to encourage hair development. In the conventional Indian medical system, many herbs and herbal medicines have been said to promote hair development and enhance the condition of hair. These herbal treatments' purported mechanisms of action include improved scalp blood flow, DHT blockers, 5-Reductase blockers, and nutritional support. Employing natural treatments for the treatment of alopecia offers many advantages, including patient compliance, less side effects, easy accessibility, low cost, and different modalities of action.[16]

MATERIALS AND METHODS

Drugs & Chemicals

Test Drug: The Ganoderma lucidum extracted powder was obtained is collected from Natural Hub, Natural Ingredients Solution Provider, R-1/24A Mohan Garden, Near Gagan Bharti School, Uttam Nagar. New Delhi-110059.

Standard drug: 2% Minoxidil obtained from Angle Biopharma Ltd., Ahmedabad and all other chemicals used for experimental purpose were of analytical

arcade used as Standard drug.

Inducing Drug: Testovirone depot injection 100mg/ml from German Remedies Zydus Cedilla Healthcare. was used to induce Alopecia.

Other Chemicals: Fehling's solution A & B, Benedict's Solution, Ferric chloride, HCL, Sulphuric acid, conc. Nitric acid, Mayer's reagent, etc. from the drug store of vidyabharti college of pharamacy Amravati. For Preliminary phytochemical screening of treatment drug i.e. Ganoderma lucidum.

Experimental animal: The experiments were carried out with Wistar albino male rats of 150-200 g bred in the animal house of the Vidyabharti college of pharmacy Amravati. The animals were housed in polypropylene cages at a temp. Almost 24±2EC with a relative humidity of 40-6 0% and 12 h light-dark cycle, with free access to food and water ad libitum during the complete study. Animal were acclimatized to laboratory conditions before the experiment were started. Experiment would perform in accordance with the committee for the purpose and supervision of experimental animals (CPCSEA) guidelines after the approval of the experimental protocol by the Institutional Animal Ethical committee (IAEC).

Preliminary Phytochemical Screening

Qualitative Phytochemical Investigation: Chemical assays were run to identify the various phytoconstituents. Tests include the Molisch's Test, the Fehling's Test, the Keller-Killiani Test, the Salkowski Test, the Biuret Test, and the Soap Formation with Water Test. Due of their biological activity, the secondary metabolites were discovered utilising phytochemical testing.

Acute Dermal toxicity study

The day before the EEGL, all of the fur was removed from the dorsal areas of the rats. EEGL15 mg/kg and EEGL20 mg/kg were placed as uniformly as possible across the exposed area of skin over a 24-hour exposure period using a porous gauze bandage and no irritating tape. Beginning two to six hours after the commencement of the exposure session and continuing for the next 30 minutes, at least once every 30 minutes, the animals were observed everyday for the next 14 days. The first 24 hours saw frequent observations as well. The toxicity investigation found changes in the eyes, mucous membranes, skin, and fur. The 20mg/kg group experienced one death compared to the EEGL15mg/kg group, which got no therapy (OECD 402). The animal quickly noticed its regular activity and growth after 24 hours. There were no variations in any of the animals' overall appearances over the course of the observation period. This result indicates LD50>20mg/kg.[17,18].

Preparation of Doses

Sample preparation: During each study protocol drugs were freshly prepared The extracted powder of Ganoderma lucidum were incorporated into Solution was made with ethanol base and adding propylene glycol in proportion of 90:10.

Testosterone test solution: Testosterone solution was prepared in the vehicle ethanol/propylenglycol (90:10).

Minoxidil solution: the standard of Minoxidil (2%) was diluted with ethanol and propylene glycol were applied topically.[19]

Experimental design

Animals divided into 5 groups of 6 rats each. The following treatment given to animals of different groups:

Table1. Different groups of animals and their treatment.

Sr.no	Groups	Treatment
1	I Negative control	Distilled Water (po)
2	II Positive control	Testosterone (1mg/kg sc)
3	III Standard	Testosterone (1mg/kg sc)+ 2%Minoxidil(Topical)
4	IV EEGL10mg/kg	Testosterone (1mg/kg sc)+EEGL(10mg/kg Topical)
5	V EEGL15mg/kg	Testosterone (1mg/kg sc)+EEGL (15mg/kg Topical)

Experimental procedure

1) The animals were taken over from the Vidyabharti College of Pharmacy in Amravati and were acclimated for a week.

2) Five groups of six rats each were formed from the rats. cm2 section of the dorsal portion of the skin had all of the rat's hairs removed using cream. The negative control group was given 21 days to recover naturally.

3)(Alopecia's induction and therapy) All groups, with the exception of the negative control group, received daily inducing agent testosterone (1 mg/kg sc) by subcutaneous injection for 21 straight days. Before 1 hour, apply topically 2% minoxidil to the control group, and administer EEGL preparations of 10mg/kg and 15mg/kg to treatment groups 1 and 2, respectively..

4)The difference in growth of hair in each group was evaluated by visual observation and was recorded by photograph after 21 days.

5) After shaving off the lengthy hair, the dorsal portion of each animal's skin was dissected and preserved in 10% formalin. A vertical piece of skin was prepared after fixing. Haematoxylin and eosin was used to stain the sections. The portion was then examined for various indicators of hair development. A follicular density (number of follicles/mm) measurement was taken of the number of follicles in a 2 mm area. In addition, the number of follicles in the anagen phase (the active growth phase) and those in the telogen phase (the resting phase) were counted in order to calculate the anagen/telogen ratio.

A. Method and Confirmation of Euthanasia: Regardless of the method of euthanasia used,
death Must be confirmed before disposal of the animal.

Methods: Euthanasia by following method

a. Chemicals

b. Destruction of the brain

c. Dislocation of the neck (cervical dislocation).

The following should be used to evaluate consciousness or confirm death.

- Lack of a heartbeat
- Lack of respiration
- Lack of corneal reflex
- Presence of rigor mortis [20].

B. Skin Biopsy: A skin biopsy is a procedure to remove cells from the surface of your body so that they can be tested in a lab. A skin biopsy is used most
Types of skin biopsy
1.Shave biopsy. A tool like a razor is used to scrape the surface of your skin. It gathers a cell sample from the top layers of the skin. These layers are called the epidermis and the dermis.

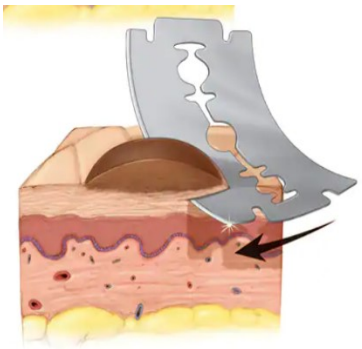

Figure.1 Shave skin biopsy [21]
2. Punch biopsy: A round-tipped cutting tool is used to remove a small core of skin, including deeper layers. The sample might include tissue from layers called the epidermis, the dermis and the top layer of fat under the skin [22].

Evaluation parameters

Skin irritation test: The hair of the Wistar albino male rats was removed, and a test was conducted to determine how much the formulations (EEGL 15 mg/kg and 10 mg/kg) irritated the rats' unharmed skin. Male Wistar albino rats had their dorsal region hairs plucked; this area was then cleaned with spirit, and the rats were administered medicine topically. The sites were observed for erythema and oedema for 48 hours following treatment.

Morphological evaluation: The difference in growth of hair in each group was evaluated by visual observations and was recorded by photographs after 21 days.
Measurement hair length: On the twenty-first day of therapy, sterile forceps were used to randomly remove hair from the depilated area. Scales were used to measure hair length, and the results were calculated as mean length SEM of 25 hairs and two parameters: (a) hair growth initiation time, which is the quickest period of time needed to begin observable hair growth, and (b) hair growth completion time, which is the quickest period of time needed to completely cover the denuded skin region with new hair. The interval between the beginning and end of hair formation was recorded for each group of animals.
Histopathological studies: After the hair was removed from each animal's dorsal region, the skin tissue was dissected. The 10% formalin solution was kept in a glass jar with the skins within. To determine the Anagen Telogen Ratio (A/T ratio) and Hair Follicle Density, the samples were sent to Histopathology Nagpur.
Statistical analysis: Statistical analysis. The data were expressed as mean ± SEM. Results were analysed statically Two-way ANOVA followed by Bonferroni multiple comparisons $p<0.0001$ compared to Positive control. For measurement of hair length. And by One way ANOVA followed by Dunnett's Multiple Comparison Test $p<0.05$. for Follicular density anagen telogen ratio.

RESULTS

Observation of Phytochemical Test

The information in the table 2 shows that the ethanolic extract of Ganoderma lucidum contained terpenoid alkaloids, flavonoids, carbohydrates, glycosides, phenolic compounds, proteins, and amino acids.
1.Skin irritation test: Rats did not show any erythema or oedema, as shown in Fig. 1, indicating that the EEGL 10 mg/kg and EEGL 15 mg/kg solution did not irritate the skin of rats. A skin

irritancy test revealed that the EEGL solution is safe to apply topically(figure1). Rats accepted the generated solution well, and it had no adverse effects on their skin, demonstrating its safety for application

Table 2. Phytochemical Test of Ethanolic extract of Ganoderma lucidum.

Sr. No.	Natural Product	Test Performed	Inference
1	Alkaloid	Wagner's reagent	+
2	Steroids	Sakowaski test	+
3	Phenolic compounds	Ferric chloride Test	+
4	Terpenoied	Salkowiski Test	+
5	Carbohydrate	Molisch's Test	+
		Fehling's Test	+
7	Saponin	Soap Formation with water	-
8	Glycoside	Keller-killiani test	+
9	Protein and Free Amino Acids	Biuret Test	+

Indicates absence + Indicates presence

Table 3. Observation of erythema and oedema in skin irritation test.

Solution	**Visual observation**	
	Erythema	**Oedema**
1.EEGL 15mg/kg solution With ethanol and propylene glycol	No	No
2. EEGL 10mg/kg solution With ethanol and propylene glycol.	No	No

Figure. 2: Observation of skin irritation test

2.Morphologic observation (visual observation): Group I animals exhibited normal hair, whereas group II animals showed a patch of dispersed hair loss. The dorsal region of the rats saw a discernible loss of hair after 20 days of testosterone administration. Testosterone is given to group III (standard) animals at the same time as groups IV (EEGL 10 mg/kg) and V (EEGL 15 mg/kg). The absence of the alopecic condition in this group of animals suggests that their activity was suppressed by the testosterone-blocking effects of EEGL10 mg/kg and EEGL15 mg/kg as well as Minoxidil 2%. (figure3)

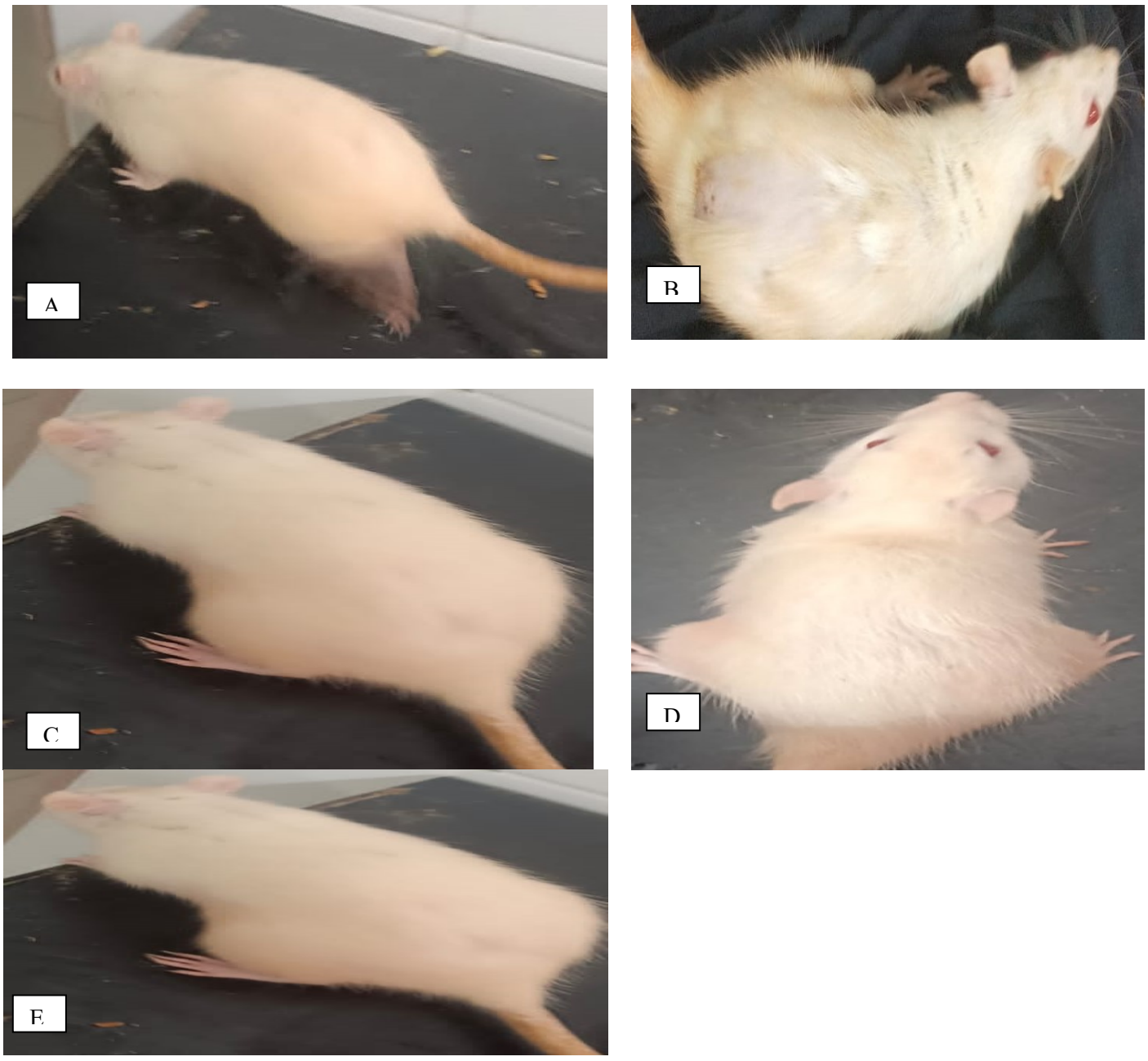

Figure3. Comparison of baldness pattern in each group. (A) Normal animal without any drug (B)Animal treated with testosterone showing diffuse alopecia (C)Animal treated with testosterone and 2%Minoxidil showing more hair growth. (D) Animal treated with testosterone and EEDL10mg/kg showing less hair growth (E) Animal treated with testosterone and EEDL15mg/kg showing more hair growth.

Table 4: Observation of hair growth

Groups	Time taken for Hair growth in (11 days/mm)	Time taken for hair growth completion (22 days/mm)
I (Negative control) Normal	2.320 ± 0.062	4.250±0.226
II (Possitive control) Inducing	0.783±0.047	2.433±0.049
III (Standard)	3.433±0.066	10.27±0.146
IV (EEGL10mg/kg)	2.667±0.033*	6.233±0.049*
V (EEGL 15mg/kg)	3.183±0.070	9.417±0.104*

3.Measurement hair length: Up until the end of the treatment cycle, hair length started to grow. When compared to 2% minoxidil solution (standard), the ethanolic extract of Ganoderma lucidum (15 mg/kg) produced a virtually same effect on hair length, with the other group of animal hair being 9 mm at the conclusion of the course (22 days). In comparison to Ganoderma lucidum 10mg/kg extract, Ganoderma lucidum 15mg/kg extract had a larger impact on hair length. This can be because the follicles switched around too soon. This indicates that the minoxidil 2% and ganoderma lucidum 15 mg/kg extracts include a higher number of hair follicles that are in the anagen phase of the hair development cycle (Table 4). Both of them have important effects.

Value expressed as mean ± SEM (n=6), Two-way ANOVA followed by Bonferroni multiple comparisons *p< 0.0001 compared to Positive control.

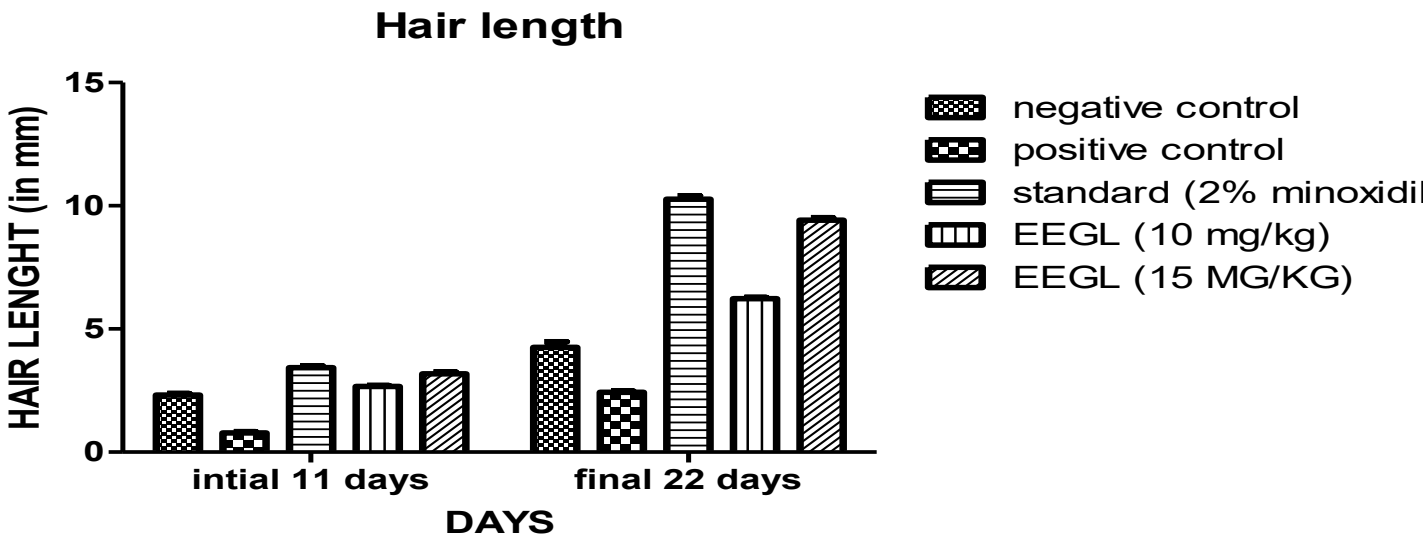

Figure.4 Observation of hair length

4.Histopathological observation:

Hair follicular density and the Anagen/Telogen ratio were the two evaluation criteria that were noted in the histopathology analysis. The gradual miniaturisation of hair follicles is a hallmark of AGA. The diameter of the hair shafts is noticeably altered when the biopsy specimen is sectioned transversely at the level of the sebaceous duct entrance into the hair follicle. In comparison to the little hair follicles, sebaceous glands appear to be enormous. The number of total follicles has significantly decreased, as shown by the horizontal sectioning of the scalp biopsy. A relative rise in telogen results from the gradual shortening of anagen (figure5).

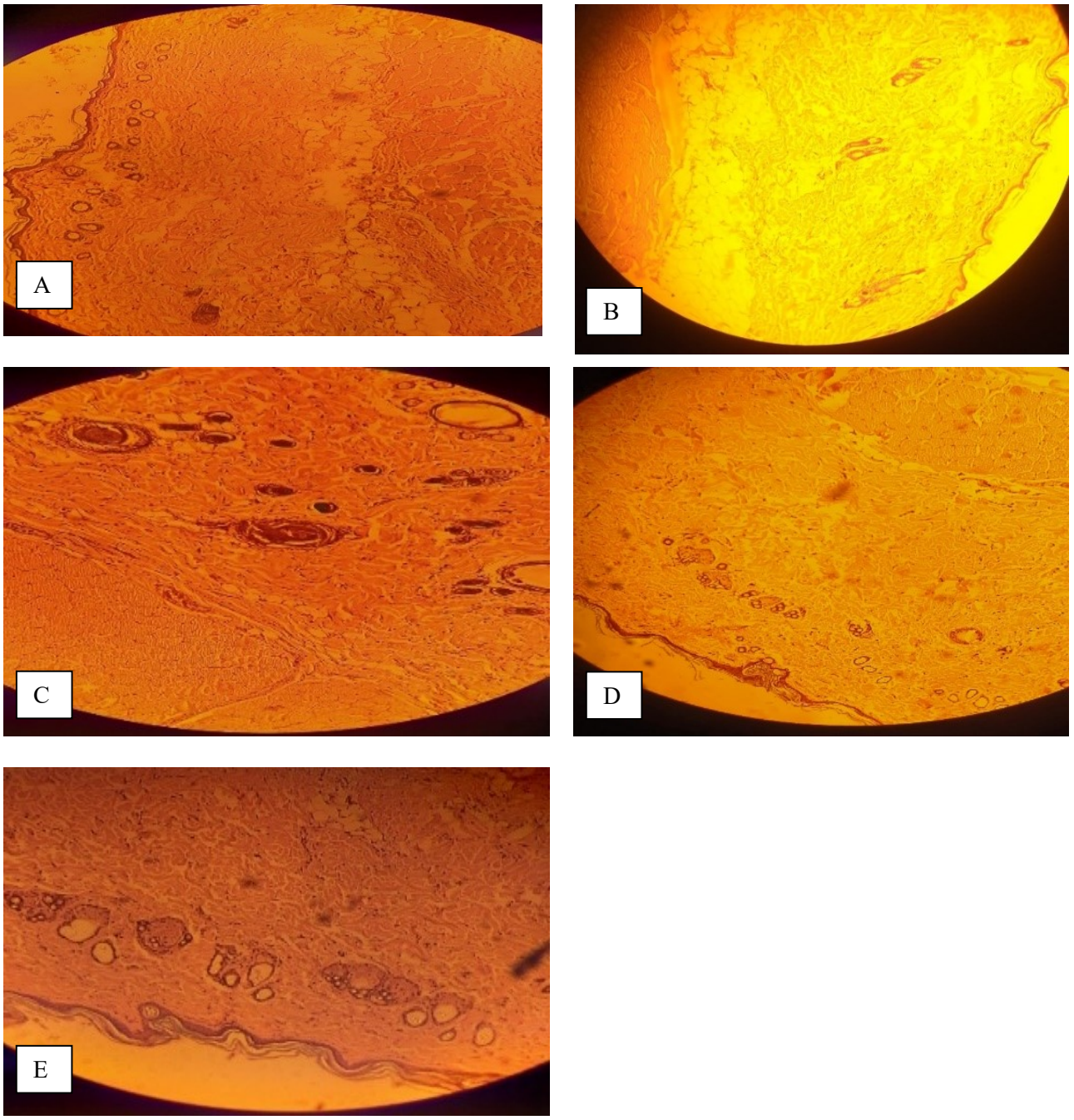

Figure 5: Photomicrograph of skin of animal in each group, (A) rat skin tissue of Negative control group, (B) Skin of animal treated with testosterone, (C) Skin of animal treated with testosterone and Minoxidil2% solution, (D) Skin of animal treated with testosterone and EEGL10 mg/kg), (E) Skin of animal treated with testosterone and EEGL15mg/kg.

Hair follicular density

It refers to the quantity of hair follicles per millimetre of skin. Skin slices from group I animals' histology revealed normal follicles and skin. Hair follicles shrank in size as a result of testosterone treatment applied to the skin. Follicles in group II exhibited traits of telogen follicles, such as being shorter in length, hollow, necrotic, and having more damaged follicles. Follicle shrinkage also means that the diameter of the follicle reduces rather than going deeper. There were a number of hair follicles in the telogen phase. The conventional medication, 2% Minoxidil, EEGL 10 mg/kg, and EEGL 15 mg/kg, respectively, reduced the effect of testosterone on hair follicle miniaturisation in

group III, IV, and V animals. (Table 5)

Anagen telogen ratio (A/T ratio

Transverse slices can be used to identify anagen hairs since they have an inner root sheath that seems normal and don't have any individual cell necrosis. Loss of the inner root sheath makes it possible to identify telogen hairs under the level of the sebaceous duct. The keratotic components that make up the inner root sheath remains are arranged in an atypical stellate pattern. An early telogen hair bulb has a cornifying club with a serrated rim and an interdigitating outer root sheath. A fully cornified club is visible in a late telogen hair follicle. An irregular basaloid star-shaped island of cells with a peripheral nuclear palisade is the terminal stage of telogen, also known as the telogen germinal unit.

Follicles in groups III, IV, and V displayed characteristics of anagen follicles, such as longer hair, dense follicles (number increases as compared to group II), less cell necrosis, and deeper presentation. The number of follicles in anagen phase was significantly increased, and fewer follicles in telogen phase were observed. There are more follicles in groups III and V than in groups I and IV. The cells of the epidermis and dermis did not significantly change. Again, as treatment time progressed, more follicles entered the hair growth phase. Anagen to telogen ratio (A/T ratio) and hair follicular density were calculated.(Table 5)

Table 5. Hair follicular density and A/T ratio in sections of skin of different groups of animals.

Sr.No.	Group no.	Treatment	Hair follicular density (no./mm)	Anagen to Telogen
1.	I	Negative control group (Normal)	1.833±0.166	1.8:1.5
2.	II	Positive control group (Inducing)	0.833±0.166	0.6:3
3.	III	Standard group (Minoxidil 2%)	3.333±0.210	2.5:1
4.	IV	Treatment group1(EEGL10mg/kg)	2.333±0.210**	2:1.3**
5	V	Treatment group 2(EEGL15mg/kg)	3.167±0.166**	2.3:1.1**

Value expressed as mean ± SEM (n=6), One way ANOVA followed by Dunnett's Multiple Comparison Test **p < 0.05 compared to Positive control for follicular density.
**p<0.05 compared to positive control for A/T ratio.
(A/T ratio: Anagen/Telogen ratio).

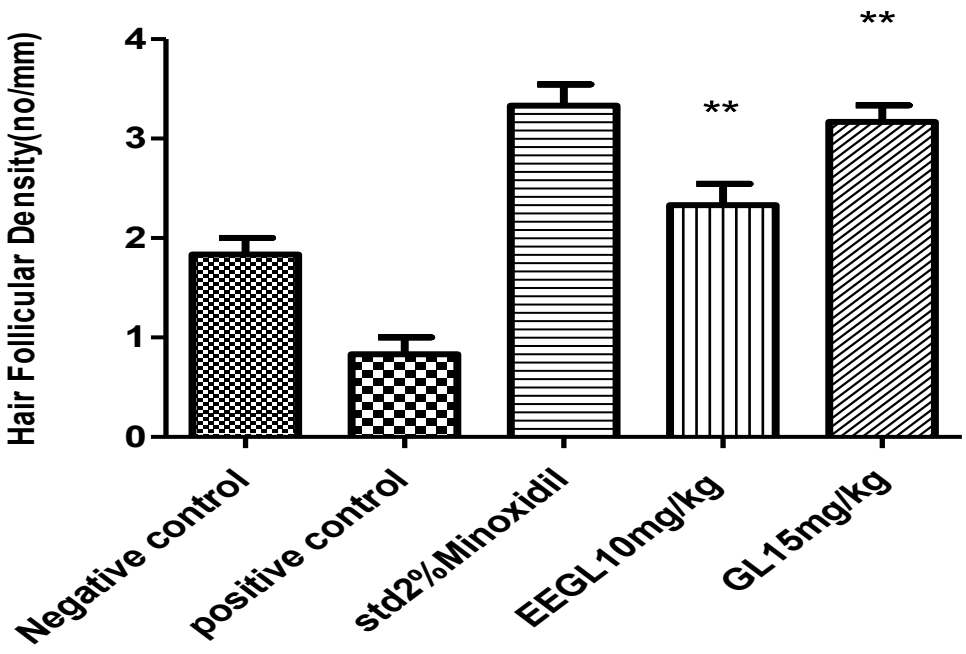

Figure 6. Hair follicular density in section of skin of different groups of animals

Discussion

All over the body, androgens act as mediators for the growth of terminal hair. Androgen-Androgenetic alopecia has a unique presentation that is reliant and heritable. Testosterone is necessary for androgenetic alopecia in addition to a genetic predisposition. Hair follicles are the focus of androgen-stimulated miniaturisation, which leads in the replacement of thin, pigmented hair with thick, coarse hair. Histological study reveals that androgen-responsive hair follicles in androgenetic alopecia (AGA), a condition triggered by the hormone dihydrotestosterone (DHT), continually contract and are accompanied by perifollicular fibrosis. In rats, testosterone treatment results in alopecia.[23] Dihydrotestosterone, a more potent androgen than testosterone that also causes the hair follicle to shrink and alters the cyclic phase of the hair development cycle, is the main androgen responsible for androgenic alopecia. DHT binds to androgen receptors in frail hair follicles, activating the genes that cause follicular miniaturisation in balding males. The current androgen action-based therapy targets the dermal papilla at the base of the follicle.[24,25] By binding to androgen receptors either directly or after being converted to dihydrotestosterone by the enzyme 5a reductase, testosterone impacts hair follicles. The demise of hair follicles may be the reason for testosterone hair loss as opposed to the androgen metabolic pathway. The goal of the ongoing research is to find alternatives to the steroid-based drugs now in use.[26]

To the best of our knowledge, this study is the first to describe the possibility that EEGL, at the two suggested dose concentrations of EEGL 10 mg/kg and EEGL 15 mg/kg, may lead to the formation of hair. Using the Rat as a model, the prepared biopsies were used to assess the hair development activity of this plant. The first method, biopsy, is still an effective instrument. A biopsy is the quickest and least painful technique to learn the most about the anatomy and histology of a hair follicle.

The rats in the Standard group received Minoxidil 2% at the same time as the Treatment group received topically applied EEGL 10 mg/kg and EEGL 15 mg/kg solution to prevent the alopecia caused by testosterone (1 mg/kg). The androgen affects hair follicles either directly or after being changed by the enzyme 5a reductase into dihydrotestosterone, a stronger androgen that attaches to androgen receptors in hair follicles. To encourage hair growth, a synthetic anti-androgenic drug known as minoxidil is available for purchase.

Animals fed testosterone and EEGL did not display any balding symptoms. Along with hair length, histological data (follicular density and anagen/telogen ratio) and visual evaluation also suggest that the doses of EEGL 10mg/kg and 15mg/kg may be possible inhibitors of androgenic activity.

Therefore, it is believed that EEGL15mg/kg is a more advantageous formulation for topical application in commercial herbal medications for androgenic alopecia and other disorders linked to androgens. According to phytochemical analysis of EEGL, only a few of the compounds discovered in this plant include triterpenes, flavonoids, sugars, and alkaloids. The terpenoids ganoderic acid TR and ganoderic acid B have an inhibitory action on 5a reductase, which allows EEGL to stop testosterone from turning into DHT.[27]

Consequently, it makes sense to assume that terpenoid play a part in this plant's ability to promote hair development. Topical application of Ganoderma lucidum ethanolic extract can safely treat testosterone-induced baldness. A hair growth promoter should have a high anagen to telogen ratio and follicular density. Thus, it follows that EEGL can be utilised to encourage hair development in those with androgenetic alopecia. These may work by preventing 5-a reductase from performing its job.

Conclusion and Future prospectives

. According to the findings of this study, an ethanolic extract of Ganoderma lucidum may be used to treat androgenic alopecia. The outcomes of this study will help the researcher better understand how EEGL10 mg/kg and EEGL15 mg/kg, which are drugs used to stimulate hair growth in androgenetic alopecia, a disorder brought on by an increase in androgen levels in the scalp, work. Additionally, it shown that while treating androgenic alopecia, EEGL 15mg/kg is superior to EEGL 10mg/kg. The current study will therefore help the researcher identify important novel therapeutic strategies for the treatment of androgenic alopecia.

Dihydrotestosterone also causes benign prostatic hyperplasia, acne, prostate cancer, and other androgen-dependent conditions. It has been discovered that the extract and its isoforms, ganoderic acid TR and ganoderic acid B, both of which include chemical compounds with 5a reductase activity, block this enzyme, making them potential candidates for further study in the treatment of these conditions.

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Ethical approval

Our research was done under ethical approval received from department of Pharmacology Department of Vidyabharti college of Pharmacy Amravati India

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