Evaluation Of Anti-Proliferative and Anxiolytic Action of *Mandukaparni Sadhita Ghrita* in Imiquimod-Induced Psoriasis in Albino Wistar Rats – An In-Vivo Experimental Study

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***Abstract*—Background: Psoriasis is a chronic immune-mediated inflammatory skin disorder characterized by keratinocyte hyperproliferation and psychological stress. [1,2]In *Ayurveda,* psoriasis resembles *Eka Kushtha,* a *Vata-Kapha* predominant disorder. [3]*Mandukaparni (Centella asiatica)* is described as *Medhya Rasayana* and *Kushthaghna* drug. [4]*Ghrita* acts as an ideal vehicle enhancing bioavailability.[5]**

Objective: To evaluate the anti-proliferative and anxiolytic action of *Mandukaparni Sadhita Ghrita* in imiquimod-induced psoriasis in Albino Wistar rats.

Methods: Psoriasis was induced using 5% imiquimod cream. Forty-two rats were divided into seven groups (n=6): induced control, oral test drug, topical test drug, combined oral + topical test drug, oral standard (methotrexate), topical standard (hydrocortisone), and combined standard group. Anti-psoriatic activity was assessed using PASI score and histopathological parameters. Anxiolytic activity was evaluated using Light and Dark Box test. Statistical analysis was performed using one-way ANOVA followed by Dunnett's test.

Results: *Mandukaparni Sadhita Ghrita* significantly reduced PASI score and histopathological severity compared to induced control. Combined oral and topical administration showed better improvement than individual routes. Behavioral tests revealed increased time spent in light chamber and increased transitions indicating anxiolytic activity. Standard drugs showed maximum effect, while test drug groups showed moderate but significant improvement.

Conclusion: *Mandukaparni Sadhita Ghrita* possesses anti-proliferative and anxiolytic activity in experimental psoriasis and may serve as a safe *Ayurvedic* therapeutic option for managing psoriasis with psychological comorbidity.

Index Terms*—Psoriasis, *Eka Kushtha, Mandukaparni Sadhita Ghrita*, Imiquimod model, Anti-proliferative, Anxiolytic activity,*Ayurveda

I. INTRODUCTION:

Psoriasis is a chronic, immune-mediated inflammatory disorder affecting approximately 2–3% of the world population. It is characterized by erythematous plaques with silvery scales, epidermal hyperplasia, and infiltration of inflammatory cells. Psychological stress and anxiety commonly accompany psoriasis, classifying it as a psychodermatological disorder.

Modern management includes topical corticosteroids, methotrexate, retinoids, phototherapy, and biologics. These therapies provide symptomatic relief but are associated with adverse effects such as hepatotoxicity, nephrotoxicity, immunosuppression, and recurrence after discontinuation.

In *Ayurveda*, all skin disorders are described under *Kushtha Roga. Eka Kushtha*, a subtype of *Kshudra Kushtha*, is characterized by *Aswedana*, *Mahavastu* and *Matsyashakalopama Twacha* and is predominantly a *Vata-Kapha* disorder. *Mandukaparni (Centella asiatica)* is described as *Medhya Rasayana* and is indicated in *Kushtha Chikitsa. Ghrita* enhances drug penetration and acts as a *Yogavahi.*

Hence, the present study was undertaken to scientifically evaluate the anti-proliferative and anxiolytic effects of *Mandukaparni Sadhita Ghrita* in an established imiquimod-induced psoriasis animal model.[6]

II. MATERIALAS AND METHODS:

Study Design

An experimental randomized, controlled, in-vivo study.

Animals

Albino Wistar rats (150–200 g) were used. Animals were housed under standard laboratory conditions with free access to food and water. Ethical clearance was obtained from Institutional Animal Ethics Committee.

Induction of Psoriasis

Psoriasis-like lesions were induced by topical application of 5% imiquimod cream on shaved dorsal skin for 7–10 days.

Grouping :(n = 6 per group)

1. Induced control (Tween 80)
2. Oral *Mandukaparni Sadhita Ghrita*
3. Topical *Mandukaparni Sadhita Ghrita*
4. Oral + topical *Mandukaparni Sadhita Ghrita*
5. Oral standard (Methotrexate)
6. Topical standard (Hydrocortisone)
7. Oral + topical standard

Assessment Parameters:

Anti-Psoriatic Parameters:

PASI score (erythema, scaling, thickness)[7]

Histopathology:

Polymorphonuclear leukocytes

Neutrophils

Fibroblasts

Neovascularization

Collagen score

Anxiolytic Parameters:

Light and Dark Box Test:[8]

Time spent in light chamber

Number of transitions

Statistical Analysis:[9],[10]

Data were analyzed using one-way ANOVA followed by Dunnett's multiple comparison test.

$p < 0.05$ was considered statistically significant.

Dose administration:

Table 4.1 dose administration of GROUP 1

Marking	Weight(gm)	Dose of 5% tween 80 solution(ml)
H	190	0.95
B	225	1.125
T	260	1.3
HB	230	1.15
BT	197	0.985
HT	190	0.95

Table 4.2 dose administration of Group 2

Marking	Weight(gm)	(Dose of test formulation) Dose in Mg	(Dose of test formulation) Dose in Ml
H	168	75.6	0.378
B	245	110.25	0.55125
T	210	94.5	0.4725
HB	212	95.4	0.477
BT	199	89.55	0.44775
HT	202	90.9	0.4545

Table 4.3 dose administration of Group 3

Marking	Weight(gm)	Dose of test formulation (ml)
H	202	0.2
B	174	0.2
T	175	0.2
HB	171	0.2
BT	180	0.2
HT	172	0.2

Table 4.4 dose administration of Group 4

Marking	Weight(gm)	(Dose of test formulation) Dose in Mg	(Dose of test formulation) Dose in ml	Dose of test formulation (ml)
H	196	88.2	0.441	0.2
B	198	89.1	0.4455	0.2
T	188	84.6	0.423	0.2
HB	186	83.7	0.4185	0.2
BT	192	86.4	0.432	0.2
HT	199	89.55	0.44775	0.2

Table 4.5 dose administration of Group 5

Marking	Weight(gm)	Dose of methotrexate (Dose in Mg)	Dose of methotrexate (Dose in ml)
H	198	0.198	0.198
B	199	0.199	0.199
T	245	0.245	0.245
HB	206	0.206	0.206
BT	276	0.276	0.276
HT	205	0.205	0.205

Table 4.6 dose administration of Group 6

Marking	Weight(gm)	Dose of hydrocortisone (Dose in ml)
H	229	0.2
B	224	0.2
T	201	0.2
HB	145	0.2
BT	207	0.2
HT	195	0.2

Table 4.7 dose administration of Group 7

Marking	Weight(gm)	Dose of methotrexate (Dose in Mg)	Dose of methotrexate (Dose in ml)	Dose of hydrocortisone (Dose in ml)
H	225	0.225	0.225	0.2
B	210	0.210	0.210	0.2
T	203	0.203	0.203	0.2
HB	214	0.214	0.214	0.2
BT	206	0.206	0.206	0.2
HT	214	0.214	0.214	0.2

4.2 OBSERVATION AND RESULTS:

4.2.1 OBSERVATION

1.Psoriasis Area Severity Index (PASI)

Table 4.8: PASI in group1,2 and 5

Group 1	Group 2	Group 5
4	2	0
3	2	0
4	3	1
3	2	1
2	4	0

Table 4.9: PASI in group1,3 and 6

Group 1	Group 3	Group 6
4	2	1
3	1	1
4	1	0
3	2	0
2	1	2

Table 4.10: PASI in group1,4 and 7

Group 1	Group 4	Group 7
4	1	0
3	1	1
4	2	0
3	3	0
2	1	1

Statistical analysis:

Table 4.11: Statistical analysis

Group 1 Mean ±SEM	Group 2 Mean ±SEM	Group 5 Mean ±SEM	Group 3 Mean ±SEM	Group 6 Mean ±SEM	Group 4 Mean ±SEM	Group 7 Mean ±SEM
3.2±0.374	2.6±0.400	0.4±0.245	1.4±0.245	0.8±0.374	1.6±0.400	0.4±0.245

Psoriasis area severity index (PASI)

Table 4.12: Statistical analysis

Dunn's multiple comparisons test	Mean rank diff.	Significant ?	Summary	Adjusted p value
Group 1 vs. Group 2	3.100	No	ns	>0.9999
Group 1 vs. Group 5	22.20	Yes	**	0.0024
Group 1 vs. Group 3	12.20	No	Ns	0.3089
Group 1 vs. Group 6	18.20	Yes	*	0.0220
Group 1 vs. Group 4	11.00	No	Ns	0.4747
Group 1 vs. Group 7	22.20	Yes	**	0.0024

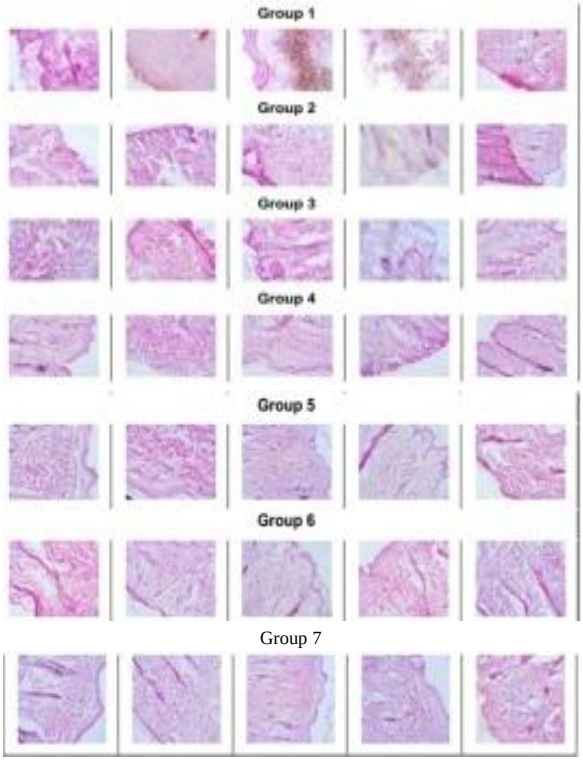

Fig.4.1: Histopathology

Table 4.13:Polymorphonuclear Leukocyte

Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
4	2	2	2	1	1	2
3	2	2	0	1	1	2
3	3	1	3	0	0	0
3	3	1	1	2	0	0
2	2	2	0	0	2	1

Table 4.14: Fibroblast

Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
3	2	3	2	0	1	2
3	3	2	1	3	2	2
2	3	1	2	2	0	0
3	3	2	2	1	1	0
4	2	1	1	1	2	1

Table 4.15: Neovascularization

Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
2	2	2	4	3	4	4
2	2	2	2	3	2	3
4	3	3	2	2	3	3
2	4	4	3	4	2	3
3	2	4	4	4	2	4

Table 4.16: Collagen Score

Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
2	2	4	2	2	2	2
3	2	2	3	3	3	3
2	2	3	4	3	3	4
2	3	2	4	4	4	2
3	2	2	2	2	2	2

Table 4.17: Neutrophils

Group 1	Group 2	Group 3	Group 4	Group 5	Group 6	Group 7
4	3	3	3	1	2	1
3	3	3	2	2	1	2
3	2	2	2	1	1	1
2	2	1	3	2	2	1
3	1	1	1	1	1	1

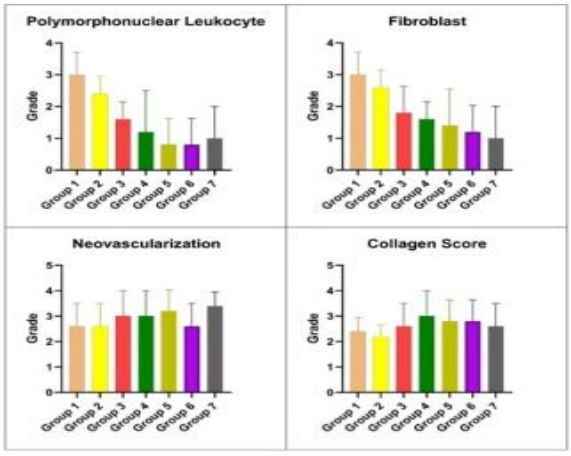

Fig.4.4: graphs

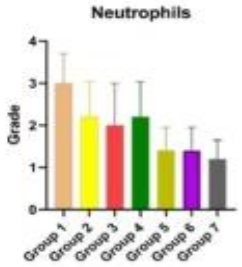

Fig.4.5: graph

Table 4.19:One way ANOVA

Dunn's multiple comparisions test	Mean rank diff.	Significant ?	Summary	Adjusted p value
Polymorphonuclear leukocyte				
Group 1 vs Group 2	4.300	No	ns	>0.9999
Group 1 vs Group 3	11.90	No	ns	0.3426
Group 1 vs Group 4	15.30	No	ns	0.0866
Group 1 vs Group 5	19.10	Yes	*	0.0136
Group 1 vs Group 6	19.10	Yes	*	0.0136
Group 1 vs Group 7	17.10	Yes	*	0.0376
Fibroblast				
Group 1 vs Group 2	3.000	No	ns	>0.9999
Group 1 vs Group 3	11.60	No	ns	0.3713
Group 1 vs Group 4	13.70	No	ns	0.1647
Group 1 vs Group 5	15.10	No	ns	0.0905
Group 1 vs Group 6	17.20	Yes	*	0.0338
Group 1 vs Group 7	18.50	Yes	*	0.0174
Neovascularization				
Group 1 vs Group 2	0.000	No	ns	>0.9999
Group 1 vs Group 3	-4.500	No	ns	>0.9999
Group 1 vs Group 4	-4.500	No	ns	>0.9999
Group 1 vs Group 5	-6.900	No	ns	>0.9999
Group 1 vs Group 6	0.000	No	ns	>0.9999
Group 1 vs Group 7	-9.300	No	ns	0.7595
Collagen score				
Group 1 vs Group 2	2.900	No	ns	>0.9999
Group 1 vs Group 3	-1.600	No	ns	>0.9999
Group 1 vs Group 4	-6.100	No	ns	>0.9999
Group 1 vs Group 5	-4.500	No	ns	>0.9999
Group 1 vs Group 6	-4.500	No	ns	>0.9999
Group 1 vs Group 7	-1.600	No	ns	>0.9999
Neutrophils				
Group 1 vs Group 2	7.500	No	ns	>0.9999
Group 1 vs Group 3	10.00	No	ns	0.6100
Group 1 vs Group 4	7.500	No	ns	>0.9999
Group 1 vs Group 5	16.50	Yes	*	0.0415
Group 1 vs Group 6	16.50	Yes	*	0.0415
Group 1 vs Group 7	19.00	Yes	*	0.0112

2. Light and Dark Evaluation

Table 4.20: Light and Dark Evaluation

No. of enter in light chamber			Time spent in light chamber (min)		
Group 1	Group 2	Group 5	Group 1	Group 2	Group 5
3	4	5	56.65	121.36	85.36
4	5	6	48.85	141.23	39.56
3	7	7	68.65	34.36	75.35
5	8	5	49.36	61.59	74.16
5	5	8	95.65	67.42	62.58

Table 4.21: Light and Dark Evaluation

No. of enter in light chamber			Time spent in light chamber (min)		
Group 1	Group 3	Group 6	Group 1	Group 3	Group 6
3	6	5	56.35	56.19	49.15
4	4	6	48.15	74.45	94.36
3	2	2	68.25	59.25	36.38
5	3	2	49.36	35.32	57.36
5	8	3	95.45	74.15	41.36

Table 4.22: Light and Dark Evaluation

No. of enter in light chamber			Time spent in light chamber (min)		
Group 1	Group 4	Group 7	Group 1	Group 4	Group 7
3	4	5	56.15	46.25	49.55
4	5	9	48.45	58.45	85.55
3	6	4	68.25	84.25	74.23
5	8	7	49.36	82.32	41.16
5	2	2	95.35	45.23	84.25

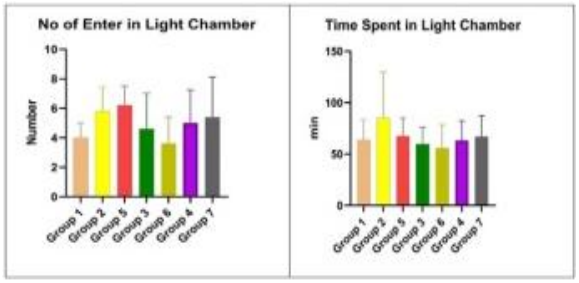

Fig.4.6: graphs

Table 4.23: Light and Dark Evaluation

Groups	No.of enter in light chamber	Time spent in light chamber(Min.)
Group 1	4 ±0.447	63.83±8.721
Group 2	5.8 ±0.735	85.19±19.881
Group 3	6.2 ±0.583	67.40±7.841
Group 4	4.6 ±1.077	59.87±7.187
Group 5	3.6 ± 0.812	55.72±10.294
Group 6	5.00 ± 1.000	63.30±8.489
Group 7	5.40 ±1.208	66.95±9.127

Table 4.24: Light And Dark Evaluation- No. of Enter in Light Chamber

Dunnett's multiple comparisons test	Mean Diff.	95.00% CI of diff.	Significant?	Summary	Adjusted P Value
Group 1 vs. Group 2	-1.800	-5.180 to 1.580	No	ns	0.5120
Group 1 vs. Group 5	-2.200	-5.580 to 1.180	No	ns	0.3189
Group 1 vs. Group 3	-0.6000	-3.980 to 2.780	No	ns	0.9925
Group 1 vs. Group 6	0.4000	-2.980 to 3.780	No	ns	0.9983
Group 1 vs. Group 4	-1.000	-4.380 to 2.380	No	ns	0.9173
Group 1 vs. Group 7	-1.400	-4.780 to 1.980	No	ns	0.7354

Table 4.25: Light And Dark Evaluation Time spent in Light Chamber(Sec)

Dunnett's multiple comparisons test	Mean Diff.	95.00% CI of diff.	Significant?	Summary	Adjusted P Value
Group 1 vs. Group 2	-21.36	-63.82 to 21.10	No	ns	0.5669
Group 1 vs. Group 5	-3.570	-46.03 to 38.89	No	ns	0.9997
Group 1 vs. Group 3	3.960	-38.50 to 46.42	No	ns	0.9996
Group 1 vs. Group 6	8.110	-34.35 to 50.57	No	ns	0.9889
Group 1 vs. Group 4	0.5320	-41.93 to 42.99	No	ns	>0.9999
Group 1 vs. Group 7	-3.116	-45.58 to 39.34	No	ns	0.9997

Fig.4.7: Experimental study in drug discovery and development unit (animal hous), NIA,Jaipur

III. RESULTS:

This study entitled generated marked findings in dermatological (anti-proliferative) and neurobehavioral (anxiolytic) parameters.

1. Psoriasis Area Severity Index (PASI) Results

Outcome measured: Reduction in PASI score (more reduction = better improvement)
Reference comparison: All groups compared with Group 1 (Normal / No treatment)

- Group 1 vs Group 2 (*Ayurveda* Oral)-Only moderate reduction compared to normal,Not significant — improvement exists but not statistically strong
- Group 1 vs Group 5 (Allopath Oral)-Very high reduction vs normal,Highly Significant ($p = 0.0024$) → Strong clinical improvement
- Group 1 vs Group 3 (Ayurveda Local)-Better than Group 1 but not strong difference,Not significant
- Group 1 vs Group 6 (Allopath Local)-Good improvement,Significant ($p = 0.0220$)
- Group 1 vs Group 4 (Ayurveda Oral + Local combined)-Improvement is present but variation large,Not significant
- Group 1 vs Group 7 (Allopath Oral + Local combined)-Maximum improvement,Highly Significant ($p = 0.0024$) → Best treatment outcome

TABLE: RESULTS

Treatment approach	Effectiveness in PASI Reduction	Statistical Strength
Allopathic Oral + Local (Group 7)	Best improvement	Highly significant
Allopathic Oral alone (Group 5)	Very effective	Highly significant
Allopathic Local alone (Group 6)	Effective	Significant

Ayurvedic therapies (Groups 2, 3, 4) Improvement present not statistically significant.
Needs larger sample size or stronger regimen.
Allopathic treatments, especially combined oral + local therapy, showed the highest reduction in PASI scores with strong statistical significance.

Pure *Ayurvedic* groups show improvement, but results did not reach statistical significance compared to normal controls due to either:

- smaller change in scores
- small sample size
- Allopathy demonstrates superior short-term PASI reduction
- Ayurveda shows positive trends but needs further strengthened protocol or longer duration
- Combined *Ayurvedic* therapy (Group 4) better than single-mode, but still not statistically significant

"According to Dunn's multiple comparison test, Group 5 (Allopathic oral) and Group 7 (Allopathic combined therapy) demonstrated statistically highly significant improvement in PASI scores ($p < 0.01$) compared to Group 1 (Normal). *Ayurvedic* interventions showed clinical improvement; however, the differences were not statistically significant."

2. Histopathological Results

The table summarizes the histopathological changes observed in across Groups 1–7. The parameters evaluated were polymorphonuclear leukocytes, fibroblasts, neovascularization, collagen deposition score, and neutrophils. The findings reflect the level of inflammation, granulation tissue formation, angiogenesis, and collagen maturation.

1) Polymorphonuclear Leukocytes (Acute Inflammation)

Group 1 showed the highest score (3.0 ± 0.316) indicating strong inflammation.
Scores progressively reduced across treatment groups.
Group 5 (0.8 ± 0.374) and Group 6 (0.8 ± 0.374) showed the least inflammatory cells, suggesting better resolution of acute inflammation.
This indicates that these groups experienced faster transition from inflammatory phase to proliferative phase

3. Fibroblasts (Granulation Tissue Formation)

Fibroblast count decreased from Group 1 (3 ± 0.316) to Group 7 (1 ± 0.447).
Higher fibroblast levels show active granulation tissue; lower values indicate more maturity of tissue.
Group 1 and Group 2 had higher fibroblast activity → early healing stage.
Group 6 and Group 7 showed the lowest fibroblast numbers → more advanced tissue maturation.

4. Neovascularization (New Blood Vessel Formation)

Group 1 had moderate neovascularization (2.6 ± 0.400).

Enhanced neovascularization noted in groups 3, 4, 5, and 7, ranging from 3.0–3.4.

Group 7 (3.4 ± 0.245) showed the highest angiogenesis, indicating improved nutrient supply and accelerated healing.

Group 6 showed moderate values, similar to control.

5. Collagen Score (Collagen Deposition / Maturation)

Group 1 had a collagen score of 2.4 ± 0.245.

Collagen increased across groups, with highest in Group 4 (3.0 ± 0.447) and Group 6 (2.8 ± 0.374).

Indicates strong collagen deposition and better tensile strength in these groups.

Group 3 and 7 also showed improvement compared to control.

6. Neutrophils (Inflammatory Response)

Group 1 showed highest neutrophils (3 ± 0.316), meaning active inflammation.

Values reduced in all treatment groups.

Group 5 (1.4 ± 0.245) and Group 7 (1.2 ± 0.200) showed the lowest neutrophil infiltration, suggesting effective inflammation control and better healing.

1). Histopathological analysis showed that treatment groups demonstrated reduced inflammation, enhanced neovascularization, increased collagen deposition, and more advanced tissue maturation compared to control (Group Groups 5, 6, and 7 consistently showed better inflammatory control and superior tissue repair parameters, indicating enhanced healing potential in these groups.

Dunn's post-hoc test was applied to identify specific group differences in histopathological parameters. The comparison was performed between Group 1 (control) and all treatment groups (Groups 2–7). The outcomes are summarized below.

1. Polymorphonuclear Leukocytes (PMNL)

PMNL infiltration reflects acute inflammation.

No significant difference was observed between Group 1 and Groups 2, 3, or 4 ($p > 0.05$).

Significant reductions in PMNL were seen in Group 5, Group 6, and Group 7 ($p = 0.0136$, 0.0136, and 0.0376 respectively).

These groups showed a marked decrease in inflammatory cells, indicating effective suppression of acute inflammation.

Groups 5, 6, and 7 exhibited significantly better anti-inflammatory activity compared to control.

2.Fibroblast Count

Fibroblast activity reflects granulation tissue formation.

Groups 2–5 did not show significant differences from Group 1 ($p > 0.05$).

Group 6 ($p = 0.0338$) and Group 7 ($p = 0.0174$) showed significant differences, indicating more advanced wound maturation compared to control.

Groups 6 and 7 promote more efficient fibroblast regulation and healing progression.

3.Neovascularization

Represents development of new blood vessels.

None of the groups (2–7) showed significant changes compared with Group 1.

All p-values were > 0.05.

No significant effect of treatments on angiogenesis compared to control.

4.Collagen Score

Reflects collagen deposition and maturation.

No group showed significant differences from control ($p > 0.05$).

Although Groups 4 and 6 showed numerically higher collagen scores, these changes were not statistically significant.

Collagen deposition improved qualitatively in some groups, but statistical significance was not achieved.

5.Neutrophils

Indicates inflammation and immune response.

No significant difference between Group 1 and Groups 2, 3, or 4.

Group 5 ($p = 0.0415$), Group 6 ($p = 0.0415$), and Group 7 ($p = 0.0112$) showed significantly lower neutrophil infiltration.

This suggests advanced resolution of inflammation.

Groups 5, 6, and 7 demonstrated significant anti-inflammatory effects, reducing neutrophil count compared to control.

Dunn's multiple comparison test revealed that among the evaluated histopathological parameters,

significant improvements were primarily observed in Groups 5, 6, and 7, particularly in polymorphonuclear leukocytes, fibroblast activity, and neutrophil infiltration. These findings indicate that these groups exhibited superior anti-inflammatory response and more advanced wound healing compared with the control group. No significant differences were observed in neovascularization or collagen deposition, although some numerical trends favored the treated groups.

Psoriatic pathology in the control group showed:

- Dense polymorphonuclear leukocyte (PMN) infiltration
- High fibroblast proliferation
- Increased neovascularization
- Disturbed collagen and dermal architecture
- After treatment with Mandukaparni Sadhit Ghrita:
- PMN cells reduced from 3.0 ± 0.31 → 1.2 ± 0.58
- Fibroblasts reduced from 3.0 ± 0.31 → 1.6 ± 0.24
- Neovascularization normalized
- Collagen deposition improved

The formulation shows strong anti-inflammatory, dermal repair, and tissue-remodeling properties consistent with both *Ayurvedic Kushthaghna* action and modern anti-proliferative pharmacology.

7. Light and Dark Box Test (Anxiolytic Activity),

The table shows two behavioural parameters measured in different groups:

1. Number of Entries into Light Chamber
(How many times the animal entered the light compartment)

2. Time Spent in Light Chamber (minutes)
(How long the animal stayed in the light area)

More entries into the light chamber,More time spent in the light chamber indicate reduced anxiety (anxiolytic effect)Because normally, animals prefer dark areas when anxious.

Group-Wise Interpretation

Group 1
Entries: 4 ± 0.447
Time: 63.83 ± 8.721 ➞Baseline/Control behaviour: moderate anxiety.

Group 2
Entries: 5.8 ± 0.735
Time: 85.19 ± 19.881 ➞Highest entries & maximum time in light.
→ Strong anxiolytic (anti-anxiety) effect.

Group 5
Entries: 6.2 ± 0.583
Time: 67.40 ± 7.841 ➞High entries but moderate time.
→ Good anxiolytic activity, but less than Group 2.

Group 3
Entries: 4.6 ± 1.077
Time: 59.87 ± 7.187 ➞Slight improvement but not strong.

Group 6
Entries: 3.6 ± 0.812
Time: 55.72 ± 10.294 ➞Lowest entries & low time.
→ Higher anxiety / poor anxiolytic activity.

Group 4
Entries: 5.00 ± 1.000
Time: 63.30 ± 8.489 ➞Moderate anxiolytic effect.

Group 7
Entries: 5.40 ± 1.208
Time: 66.95 ± 9.127 ➞Good number of entries, moderate time.
→ Mild to moderate anxiolytic activity.

Group 2 shows the best anxiolytic effect, with maximum entries and longest time in the light chamber.
Group 5 also shows good improvement, especially in number of entries.
Group 6 shows poor performance, indicating higher anxiety.
Other groups show moderate anxiolytic activity.
This pattern helps identify which treatment or drug is most effective in reducing anxiety behaviour.
Compared Group 1 (control) with all other groups for two behavioural outcomes:
1. Number of Entries in Light Chamber
2. Time Spent in Light Chamber (seconds)

Using: One-Way ANOVA → Dunnett's post-hoc test (Dunnett compares each treatment group only against control)

MAIN FINDING (Very Important)
None of the treatment groups showed statistically significant difference from Group 1.
All comparisons show: "ns" (not significant).
All adjusted p-values are greater than 0.05.

This means: → No treatment produced a significant anxiolytic effect compared to the control group.
1. Number of Entries in Light Chamber
No treatment increased the entries enough to be statistically significant.
2. Time Spent in Light Chamber (Seconds)
Time spent in the light chamber is not significantly different between control and any treated groups.

One-way ANOVA followed by Dunnett's post-hoc test revealed that:
None of the treatment groups (Groups 2–7) showed a statistically significant change in either number of entries into light chamber, or time spent in the light chamber,when compared with Group 1 (control).All p-values were greater than 0.05, and confidence intervals crossed zero.Therefore, no treatment demonstrated significant anxiolytic (anti-anxiety) activity in this test.

IV. DISCUSSION:

Mandukaparni possesses *Tikta* and *Kashaya rasa*, *Laghu guna* and *Sheeta virya*, which pacify *Vata* and *Kapha dosha*. Its phytochemical constituents such as asiaticoside, flavonoids and triterpenoids exhibit anti-inflammatory, antioxidant and immunomodulatory actions.[11],[12]
Ghrita enhances drug penetration and nourishes skin tissues. The observed reduction in epidermal hyperplasia and inflammatory infiltration may be attributed to regulation of keratinocyte proliferation and immune response modulation.[13]
The anxiolytic effect is explained by *Mandukaparni's Medhya Rasayana* property, acting on CNS and neuro-immune pathways. [14]Combined administration yielded superior results due to both systemic and local action.
Limited histological regression compared to standard drugs may be due to shorter duration of study and experimental limitations.

V. CONCLUSION:

The present experimental study was undertaken to evaluate the antiproliferative and anxiolytic action of *Mandukaparni Sadhit Ghrita* using the Imiquimod-induced psoriasis model in Albino Wistar rats. Based on observations and the results obtained from PASI scoring, histopathological examination, and behavioral analysis, the following conclusions can be drawn:

1. Antiproliferative Effect
Mandukaparni Sadhit Ghrita produced a significant reduction in erythema, scaling, and skin thickening in IMQ-induced psoriasis. The decrease in PASI scores indicates a strong anti
proliferative and anti-inflammatory effect.
Histopathology further confirmed: reduction in polymorphonuclear infiltration, decreased fibroblast proliferation, normalized neovascularization, restoration of normal epidermal architecture.
These findings validate the *Kushtaghna*, *Vranaropaka*, and *Rasayana* actions of *Mandukaparni*, as described in *Ayurveda.*

2.Anxiolytic Effect
The Light–Dark Box test revealed that *Mandukaparni Sadhit Ghrita* significantly increased the time spent in the light chamber, indicating a clear anxiolytic effect.
This supports its classical *Medhya Rasayana* property.

3.Dual Therapeutic Action
The formulation demonstrated both dermatological and psychological benefits, addressing:
skin inflammation and hyperproliferation, and associated anxiety-like behavior.This dual action is especially valuable in psoriasis, a disease with strong psychosomatic involvement.
Such holistic efficacy aligns completely with the *Ayurvedic* approach of treating *Sharira* and *Manas* together.

4.Efficacy

When compared with standard treatments such as methotrexate and hydrocortisone:

Mandukaparni Sadhit Ghrita showed comparable importance.

VI. FUTURE SCOPE:

Long-term toxicity studies
Molecular marker evaluation (IL-17, TNF-α, IL-23)
Clinical trials in psoriasis patients
Standardization of formulation
Comparative studies with biologics

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