DOI: http://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20193240

**Original Research Article**

# Uniqueness of Dr. JRK's 777 oil and how rancidity distorts *Wrightia tinctoria*

**Aruna Vadivel[1]*, Amruthavalli Gatla Venkata[1], Soundharya Ravi[2], Gayathri Rajagopal[3]**

[1]Department of Research and Development, [2]Department of Quality Control, Dr. JRK's Research and Pharmaceuticals Pvt. Ltd., Chennai, Tamil Nadu, India
[3]Dr. JRK's Research and Pharmaceuticals Pvt. Ltd., Chennai, Tamil Nadu, India

**Received:** 23 March 2019
**Revised:** 20 May 2019
**Accepted:** 04 June 2019

***Correspondence:**
Dr. Aruna Vadivel,
E-mail: aruna_v@jrkresearch.com



## ABSTRACT

**Background:** Several oil based AYUSH formulations are available for the treatment of psoriasis. In the recent time the importance given to *Wrightia tinctoria* has totally side-lined the quality of oil used in such preparation. Therefore, the importance of oil used in the formulation with reference to rancidity and how that would affect the therapeutic effect of *Wrightia tinctoria* needs to be studied.
**Methods:** In the present study we have adopted the following methods such as acid value, DPPH assay, nitric oxide inhibition, anti-glycation, CAM assay and fibroblast toxicity. The base oil used was studied for the above then the oil that showed least acid value was incorporated with *Wrightia tinctoria* extract and then studied by the above methods.
**Results:** The above study clearly show that the activity of *Wrightia tinctoria* decrease significantly when it was formulated in oil with high rancidity. Whereas the activity of *Wrightia tinctoria* was well preserved in oil with least rancidity. The contribution of acid value of oil in decreasing the activity of *Wrightia tinctoria* was much higher than the effect of acid value in increasing DPPH activity, Nitric oxide production, inducing glycation event and angiogenesis and causing fibroblast toxicity.
**Conclusions:** The present study clearly concludes that the acid value of base oil would not only worsen psoriasis but also can inactivate and convert the herbal metabolites to toxic elements. Due diligence is necessary in preparing AYUSH drugs where not only the quality of herbs should be considered but also the oil base as well.

**Keywords:** Rancid oil, *Wrightia tinctoria*, 777 oil, Anti-oxidant, Coconut oil

## INTRODUCTION

Psoriasis is an incurable auto-immune disorder of the skin that manifest as hyper proliferation of the epidermal cells with impaired skin barrier effect. The inflammatory changes do occur at different time intervals. Although several predisposing factors are hypothesized but the disease remain incurable and at best psoriasis can be managed and the remission phase can be prolonged.[1-3]

In modern medicine several drugs such as anti-mitotic preparations, topical/oral immune suppressive agents etc., are used however such therapy has definite side effects as well. UV therapy is also used and similarly monoclonal antibody therapy is also available. However, all such therapies are quite expensive.[4]

For the palliative therapy, large number of psoriatic patients in India often flocks around alternative stream of

healing practices. Several herbal preparations are vastly used for the treatment of psoriasis and as a result the business opportunity available for several manufacturers in India to introduce several single to poly herbal oils for psoriasis have increased significantly and such market is growing exponentially and unabated.[5]

Among various herbal oils from the alternative stream of healing practice for the treatment of psoriasis are available the oil prepared with the leaves of *Wrightia tinctoria* has occupied an exalted position in Indian market. Since the licensing system for the products from alternative stream of healing in India is not harmonized and is currently under the purview of respective state licensing authority. Further the quality norms for such products are not rigorous like the allopathic drugs. This has resulted in the entry of several herbal oils with *Wrightia tinctoria* as one of the ingredients for the treatment of psoriasis in Indian market.[6]

In our earlier study we have established that the base oil such as coconut oil or gingelly oil used for preparing *Wrightia tinctoria* based preparation by different manufacturers showed high acid value due to rancidity. The rancid oils are bound to worsen the problem of psoriasis and would further trigger the inflammatory response. The *Wrightia tinctoria* oil with high acid value is bound to harm psoriasis than act as drug. However, such side effects are seldom noticed or reported may be due to the acceptance of the fact that psoriasis is incurable and herbal drugs are always safe.[7-9]

The frequency of inflammatory episode vis-à-vis use of oils with high acid value for the treatment of psoriasis is not known due to lack of any focused clinical trial.

In the present paper we have studied the rancidity status Dr. JRK's 777 oil and the importance of rancidity in worsening the psoriatic condition which was assessed by a battery of in vitro tests such as DPPH assay, nitric oxide inhibition anti-glycation, and angiogenesis by CAM assay and fibroblast toxicity. Details are presented in the paper.

## METHODS

### *Details of the products studied*

Dr. JRK's 777 oil is a registered, licensed proprietary Siddha product of Dr. JRK's Research and Pharmaceuticals. The oil is prepared with the leaf extract of *Wrightia tinctoria*. The product has been studied for the complete toxicity profile in order to establish its safety by following OECD guidelines. The tests performed for Dr. JRK's 777 oil were

- Skin sensitization in guinea pig;
- Effect on mucus membrane;
- In vitro mammalian cell gene mutation test- Chinese Hamster ovary K1 cell line;
- Bacterial reverse mutation test using *S. typhimurium* and *E. coli* WP2 uvr A;
- Mammalian erythrocyte micronucleus testing Swiss Albino mice;
- Acute lethal dose;
- Reproduction developmental toxicity screening.

*Coconut oil*

The above sample with acid value of 1.4 was used for the present study.

*Rancid coconut oil*

Coconut oil stored in our laboratory with acid value of 25 was used for the present study.

### *DPPH assay*

*Anti-oxidant (DPPH) or free radical scavenging assay*

The DPPH (2, 2 diphenyl-1- picryl hydrazyl) assay method is based on the reduction of DPPH, a stable free radical. The free radical DPPH with an odd electron gives maximum absorption at 517 nm (purple colour). When antioxidants react with DPPH, the molecule is reduced to DPPHH and as consequence, the absorbance decreases. The reduction of DPPH to DPPH-H results in more or less decolorization (yellow colour) with respect to the number of electrons captured. More the decolorization, more is the reducing ability of the test material. This test has been the most accepted model for evaluating the free radical scavenging activity of many chemical compounds.[10]

When a solution of DPPH is mixed with the in-study substance, it can donate a hydrogen atom giving rise to the reduced form of di phenyl picryl hydrazine; a non-radical with the loss of violet colour (although there would be a residual pale yellow colour from the picryl group if present).

Different concentrations (1, 2, 3 mg/ml) of the samples were added to toluene solution of DPPH radical. The final concentration was adjusted to 0.1 mM. The mixture was shaken vigorously for 1 minute by vortexing and left to stand at room temperature in dark condition for 30 minutes. The absorbance of the samples was measured using UV 160 spectrophotometer at 517 nm against ethanol blank. A negative control was taken after adding DPPH solution to 0.2 ml of water. The percent of DPPH discoloration of the sample was calculated according to the equation.

$$\% \text{ discolouration} = (1 - (\text{sample}/\text{control})) \times 100.$$

***Inhibition of nitric oxide (NO) production***

Nitric oxide (NO) is a diatomic free radical produced from L-arginine by constitutive and inducible nitric oxide synthase (cNOS and iNOS) in numerous mammalian cells and tissues. Nitric oxide (NO), superoxide (O2-) and their reaction product peroxynitrite (ONOO-) may be generated in excess during the host response against viral and antibacterial infections and contribute to some pathogenesis by promoting oxidative stress, tissue injury and even cancer.

Oxidative damage, caused by action of free radicals, may initiate and promote the progression of diseases that indicates inflammation. The mechanism of inflammation injury is attributed, in part to the release of reactive oxygen species (ROS) from the activated neutrophils and macrophages. ROS propagate inflammation by stimulating the release of mediators such as NO and cytokines.

Macrophages were seeded at the density of $5 \times 10^3$ cells per well in a micro titer plate. The plate was incubated for 12 h at 37˚ C with 5% $CO_2$. Then media of each well was then aspirated, added and fresh FBS-free DMEM media was replaced. Different concentrations (5, 10 and 15 mg/ml) of the samples oils were used for testing.[10]

After 1 h treatment, the cells were stimulated with 1 µg/ml of LPS for 24 h. The presence of nitrite was determined in cell culture media using commercial NO detection kit. After 10 minute incubation, the absorbance was measured at 540 nm using microplate reader. The amount of nitrite in the media was calculated from sodium nitrite ($NaNO_2$) standard curve

***Anti-glycation assay***

Glycation is the key molecular basis of several diabetic complications like diabetes retinopathy, nephropathy, neuropathy and some cardiovascular diseases. This is a non-enzymatic reaction between amino groups of proteins and carbonyl groups of reducing sugars forming a fluorescent, insoluble advanced glycation end product that accumulate as long living protein thus compromising the physiological function. Glycation is a non-enzymatic condensation reaction between reducing sugars and amino groups of proteins that undergo rearrangements to stable ketoamines, leading to the formation of advanced glycation end products (AGEs).

Antiglycation activity was determined using the bovine serum albumin assay with slight modification. In the experiment, the final reaction volume was 1.0 ml and carried out in 1.5 ml Eppendorf tubes. Bovine Serum Albumin 500 µl (1 mg/ml concentration) was incubated with glucose 400 µl (500 mM final concentration) and different concentrations of the samples (50, 40, 30, 20, 10 mg/ml) in 100 µl of phosphate buffer saline was used and 100 µl Arbutin was used as standard. A negative control was carried out at the same time with BSA 500 µl (1 mg/ml concentration), 400 µl phosphate buffer saline and the different concentrations of the sample (as described above) incubated under the same conditions.[10]

The reaction was allowed to proceed at 60 °C for 24 hours and the reaction was terminated by adding 10 µl of 100% (W/V) trichloroacetic acid (TCA). The TCA added mixture was kept at 4°C for 10 minutes and then centrifuged for 4 minutes at 13000 rpm. The precipitate was re-dissolved in alkaline phosphate buffer saline (pH 10) and was quantified for the relative amount of glycated BSA based on fluoresce intensity by fluorescent microplate reader. The excitation and emission wavelength that used were 370 nm and 440 nm respectively. Each concentration of the sample was analyzed thrice. Percentage of inhibition was calculated and the sample concentration required for the 50% of inhibition was calculated using the formula

% of inhibition = OD blank – (OD sample – OD sample negative)/OD blank x 100

***Angiogenesis- CAM assay***

Chick chorio allontoic membrane is an extra embryonic membrane which is rich in blood vessels and is widely used in assessment of angiogenic and anti-angiogenic products. In the present study we have used 9 days old fertilized eggs. The eggs were surface sterilized and then incubated at $37^0$C for one day. Then the eggs were observed under egg candling box to visualize the presence of blood vessels. A small hole was drilled in the air space to drain out the air. Further a small square shaped window was drilled out on the egg shell without damaging the membrane beneath. 10 eggs were used for each experiment.[11]

Different concentrations of the samples (1, 2 mg/ml) were used for the study. The sample was loaded in sterilized methyl cellulose disc which was then carefully placed in the window made in the egg. The eggs were then incubated for further 3 more days at $37^0$C. After incubation the egg was opened and the extent of angiogenesis was observed. Saline was used as negative control and SLS was used as positive control

***Fibroblast toxicity assay***

*MTT assay- principle*

The cell proliferation occurs as a result of the mitochondrial succinate-terazolium reductase system that convert 3-(4, 5- dimethylthiazol-2-yl)-2, 5-diphenyltetrazolium bromide (MTT) to a blue colored formazan. The test denotes the survival cells after exposure to the test samples.

*Cell culture study*

Fibroblast cell line was used for the present study. The cells were maintained and sub cultured in 25mm2 tissue

culture flasks using 5 ml of minimum essential medium (MEM) supplemented with 10% fetal bovine serum, 3% L-glutamine, penicillin (100 IU/ml), streptomycin (100 µg/ml), Amphotericin B (20 µg/ml), phenol red. The pH of the medium was adjusted to 7.2-7.4 with 7.5% sodium bicarbonate and all flasks were incubated at 37°C in a humidified 5% $CO_2$ /95% $O_2$ incubator.

Cells were grown in an appropriate growth media and seeded at the concentration of 6×104 cells per ml of media in a 24 well plate. Cells were allowed to adhere to the bottom for 12 hours followed by treatment with different concentrations of the oils and incubated for 48 hours at 37°C with 5% $CO_2$. After treatment of 48 hours, cells were treated with 10% MTT in media for 4 hours in 37°C and 5% $CO_2$. Media was then aspirated and the adherent cells were treated for formazon product that was dissolved in DMSO, centrifuged at an RPM of 5000 for 15 minutes to remove debris and the spectrophotometric absorbance of the sample was measured using a microplate reader. The wavelength used for measuring the absorbance of formazan product was 570 nm and the percentage of reduction in cell proliferation was determined.

## RESULTS

### *Rancidity assay*

The acid value of Dr. JRK's 777 oil was 1.2 whereas the coconut oil used in the Dr. JRK's 777 oil was 0.85. The acid value of the rancid coconut oil was 25.2 (Table 1).

**Table 1: Acid value of Dr. JRK's 777 oil.**

| S. no | Product name | Acid value |
|---|---|---|
| **1** | Dr. JRK's 777 oil | 1.2 |
| **2** | Coconut oil | 0.85 |
| **3** | Rancid coconut oil | 25.2 |

### *DPPH assay*

Dr. JRK's 777 oil showed high anti-oxidant effect in a concentration dependent manner. Similarly, the coconut oil with less acid value also showed good anti-oxidant effect. On the contrary, the rancid coconut oil exhibited poor anti-oxidant effect (Table 2).

**Table 2: Free radical scavenging assay of various anti-psoriatic oil.**

| Sample | % inhibition/concentration in mg/ml | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| **Dr. JRK's 777 oil** | 78.0 | 82.0 | 83.5 |
| **Coconut oil** | 85.0 | 88.3 | 88 |
| **Rancid coconut oil** | 40.5 | 38.5 | 35.5 |

### *Inhibition of nitric oxide (NO) production*

The effect Dr. JRK's 777 oil on NO production inhibition is presented in the Table 3.

In order to reconfirm the antioxidant effect of Dr. JRK's 777 oil which was tested by DPPH assay, nitric oxide inhibition assay was also performed. LPS was used as a positive control to induce NO production. The values of NO production after treatment were subtracted from the NO value after treatment with LPS was used to arrives the percentage inhibition of NO.

Dr. JRK's 777 oil showed high activity in inhibiting NO production with 84%. Whereas the rancid coconut oil showed least effect in inhibiting NO production (Table 3).

**Table 3: Inhibition of nitric oxide.**

| Sample details | Concentration of sample in mg/ml and % of NO production (Values in the parenthesis denote% inhibition) | | |
|---|---|---|---|
| | **5** | **10** | **15** |
| **Only LPS (Lipo poly saccharide positive control) (83% production of NO)** | | | |
| Dr. JRK's 777 oil | 43 (48) | 21 (75) | 13 (84) |
| Coconut oil | 20 (76) | 44 (47) | 59 (29) |
| Rancid coconut oil | 29 (65) | 32 (61) | 48 (42) |

### *Anti-glycation assay*

Anti-glycation effect of Dr. JRK's 777 oil, i.e., IC50 concentration was evaluated. IC50 concentration of Arbutin the positive control was 0.8 mg/ml. Dr. JRK's 777 oil exhibited activity at 10 mg/ml. Interestingly the coconut oil with acid value of 0.85 also showed IC50 activity at 10 mg/ml. on the contrary the rancid coconut oil was least effective in inhibiting the glycation event (Table 4).

**Table 4: Anti-glycation effect of various anti-psoriatic oils.**

| Sample details | Inhibition of glycation event - IC 50 concentration in mg/ml |
|---|---|
| **Dr. JRK's 777 oil** | 10 |
| **Coconut oil** | 10 |
| **Rancid coconut oil** | 20 |

### *CAM assay*

Dr. JRK's 777 oil did not induce angiogenesis. Neither secondary nor tertiary blood vessels had increased due to the treatment of Dr. JRK's 777 oil. The *Wrightia* tinctoria extract did not induce angiogenesis however when the

extract was treated in rancid oil showed significant impact in inducing angiogenesis in chick embryo CAM assay. Interestingly the rancid oil induced angiogenesis, however the extent of angiogenesis induced by rancid oil was lower than that of *Wrightia* extract treated in rancid oil.

**Table 5: Effect of various anti-psoriatic oil in inducing angiogenesis (CAM assay).**

| Sample | Concentration (mg/ml) | Number of blood vessels/ hour after treatment | | | | | |
|---|---|---|---|---|---|---|---|
| | | Secondary | | | Tertiary | | |
| | | 48 h | 72 h | 96 h | 48 h | 72 h | 96 h |
| **Control (normal saline)** | - | 16 | 17 | 16 | 172 | 172 | 169 |
| **Dr. JRK's 777 oil** | 1 | 18 | 18 | 19 | 160 | 165 | 162 |
| | 2 | 17 | 18 | 18 | 162 | 167 | 165 |
| ***Wrightia tinctoria* extract** | 1 | 15 | 15 | 16 | 169 | 170 | 172 |
| | 2 | 16 | 17 | 16 | 172 | 170 | 170 |
| ***Wrightia tinctoria* extract in rancid coconut oil** | 1 | 33 | 32 | 34 | 190 | 200 | 211 |
| | 2 | 32 | 34 | 35 | 199 | 211 | 213 |
| **Coconut oil** | 1 | 16 | 16 | 16 | 171 | 170 | 172 |
| | 2 | 15 | 15 | 17 | 172 | 171 | 170 |
| **Rancid coconut oil** | 1 | 21 | 22 | 23 | 180 | 184 | 187 |
| | 2 | 22 | 24 | 25 | 190 | 186 | 189 |

***Fibroblast toxicity assay***

Dr. JRK's 777 oil did not show any cytotoxic effect in fibroblast, whereas rancid coconut oil produced cytotoxicity. The fibroblast toxicity was directly proportional to acid value (Table 6).

**Table 6: Fibroblast toxicity assay.**

| Sample | IC90 concentration in µg/ml | Toxicity ranking |
|---|---|---|
| **Dr. JRK's 777 oil** | 2000 | Nil |
| **Coconut oil** | 2000 | Nil |
| **Rancid coconut oil** | 100 | High |

***Free radical scavenging assay of Wrightia tinctoria extracts***

TheWrightia tinctoria extract that was used in the formulation of Dr. JRK's 777 oil was tested for anti-oxidant effect. Extract of the above plant showed high free radical scavenging activity in a concentration dependent manner (Table 7).

**Table 7: Free radical scavenging assay of various herbal extracts.**

| Sample | % inhibition/ concentration in mg/ml | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| ***Wrightia tinctoria* extract** | 85 | 88 | 92 |

***Free radical scavenging assay of Wrightia tinctoria extract treated in rancid coconut oil***

To understand the impact of rancidity upon the anti-oxidant effect of *Wrightia tinctoria* the present study was under taken.

The activity of *Wrightia tinctoria* showed great dip when treated in rancid oil (Table 8).

**Table 8: Free radical scavenging assay of various herbal extracts treated in rancid coconut oil.**

| Sample | % inhibition/ concentration in mg/ml | | |
|---|---|---|---|
| | 1 | 2 | 3 |
| ***Wrightia tinctoria*** | 55 | 52 | 50 |
| **Coconut oil** | 85.0 | 88.3 | 88 |
| **Rancid coconut oil** | 40.5 | 38.5 | 35.5 |

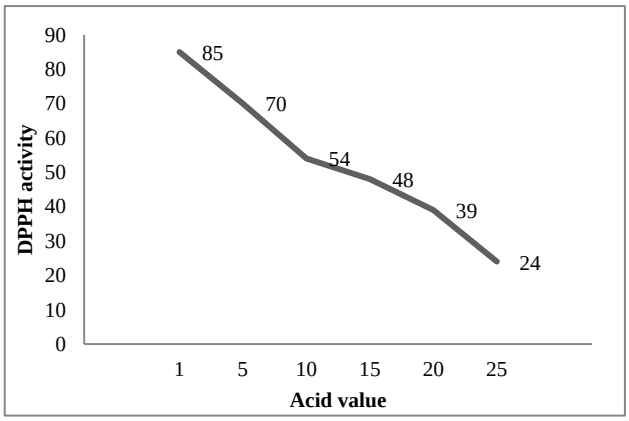


**Figure 1: DPPH activity of *Wrightia tinctoria* vs. acid value.**

***Graph showing effect of acid value on DPPH activity of Wrightia tinctoria***

In order to understand the effect of the acid value on DPPH activity of *Wrightia tinctoria* we have taken up the present study. 3 mg/ml concentration of *Wrightia tinctoria* was incorporated in different sets of coconut oil

with varying acid values and then DPPH activity was measured. The findings show that acid value is directly proportional to decreased DPPH activity of *Wrightia tinctoria* (Figure 1).

***Time kinesis– effect of rancidity on DPPH activity of Wrightia tinctoria***

After establishing the definite role of rancidity in reducing the anti-oxidant effect of *Wrightia tinctoria* a study was planned to understand the impact of time of rancid oil in attenuating the anti-oxidant effect. Like the definite role of rancidity in retarding the anti-oxidant activity of *Wrightia tinctoria*, the duration of treatment of rancid oil was also found to have strong negative impact on the anti-oxidant effect (Table 9).

**Table 9: Time kinesis of rancidity on DPPH activity of extracts.**

| Sample | % inhibition/concentration in 3 mg/ml duration of treatment in days | | |
|---|---|---|---|
| | 5 | 10 | 15 |
| ***Wrightia tinctoria*** | 49 | 40 | 36 |

## DISCUSSION

The present study has unraveled several mysterious facets of the acid value – herbal interaction. Due to the globally accepted limitation in treating psoriasis, the patients often go panic and would run from pillar to post in search of newer and newer therapies/treatment products. As a result of the above scenario, several manufacturers of various Siddha and Ayurveda products have come up with many single or poly herbal oil preparations for the management of psoriasis.

Among the various herbs used in the preparation of anti-psoriatic oils, the leaves of plant *Wrightia tinctoria* enjoys great patronage and credibility. Several 'touch and go' experiments have shown that *Wrightia tinctoria* is likely to possess some anti-psoriatic effect.[4,5,12] However various nuances and nitty-gritty of the oil base and its quality that might either boost or impair the therapeutic excitement for the treatment of psoriasis is not clearly understood.

Our earlier study has shown that *Wrightia tinctoria* possess activity against keratinocyte proliferation and the activity was found to be non–cytotoxic in nature. After the recognition of *Wrightia tinctoria* for the treatment of psoriasis, several manufactures in India started to exploit the plant and have brought out several preparations. Oils with *Wrightia tinctoria* alone and or in combination with several other herbal extracts are available in the market today. Despite the availability of large number of *Wrightia tinctoria* based oils in India still the incidence and prevalence of psoriasis is on increase. Therefore naturally the question would emerge on the real therapeutic benefit of *Wrightia tinctoria.*

We have established earlier the acid value of the oil that was used for preparing products for psoriasis has a definite role in worsening the problem than offering any benefit. However, such broad hypothesis was arrived purely from the per see of acid value without including and considering the multi-various therapeutic benefits of various herbs.[13,14]

In our previous study we have established that several herbs can act as metal chelators to nullify the metal toxicity.[15] Similarly, whether some herbs can nullify the effect of acid value of the base oil and offer therapeutic benefit that remains unclear. To answer the above question we have undertaken the present study.

Dr. JRK's 777 oil showed very high anti-oxidant activity. However the activity of the *Wrightia tinctoria* extract showed significant decrease when treated with rancid oil. This led us to presume that the acid value alone may not be contributing negatively towards the DPPH activity but the acid value may be definitely modify the herbal constituents thereby decrease its activity which is not clearly known to the scientific fraternity until our present study.

To affirm the above postulate through an experimental design we have further treated the *Wrightia tinctoria* extract in rancid oil and tested for anti-oxidant activity at different time points. With increase in time, decreased anti-oxidant activity of *Wrightia tinctoria* was observed. Similarly the role of different acid value numbers on the antioxidant effect of the *Wrightia tinctoria* was also studied. Both the duration of treatment as well as the acid value number was contributing equally to diminish the anti-oxidant activity of *Wrightia tinctoria*. On the contrary the concentration of the extract was seems to have less resisting power against acid value. This suggests that however high the concentration of herbal extract (s) may be in the formulation has less effect if the base oil is rancid.

To further establish the co-relation between the rancidity of the base oil and herbal interaction versus therapeutic benefits we studied Dr. JRK's 777 oil for Nitric Oxide (NO) inhibition. The findings clearly show that Dr. JRK's 777 oil showed greater effect in inhibiting NO production. However, *Wrightia tinctoria* treated in rancid oil showed poor activity.

We further intrigue the above co-relation to unearth the possible science and hence performed CAM assay. Dr. JRK's 777 oil did not induced angiogenesis in secondary or tertiary blood vessels in chick embryo up to a period of 96 hours. *Wrightia tinctoria* extract and coconut oil with low acid value also did not induce angiogenesis. Interestingly *Wrightia tinctoria* treated in rancid oil induced angiogenesis significantly when compared to rancid coconut oil.

CAM assay being a semi-in vivo method, the result obtained by us assumes great significance. The possible explanation could be the end product of herbal- rancid oil interaction which we presume may be extremely toxic than acid value.

The fibroblast toxicity assay gives further support to our postulate that rancidity is directly linked to toxicity and may be indirectly linked to modifying the bio-active molecules of various herbs. However, this postulates requires further study.

We are not surprised with the superior quality and the safety bio-data of Dr. JRK's 777 oil.

Dr. JRK's 777 oil may be the first and the only Siddha product having studied thoroughly and elaborately for various safety parameters as per OECD guidelines.

Psoriasis being an auto-immune disorder the etio-pathalogy of the disease also includes free radical generation and glycation end products. Therefore, use of anti-oxidants also as one of the medicaments for the management of psoriasis is well known.[2,3]

## CONCLUSION

In the present context the strong anti-oxidant effect of Dr. JRK's 777 oil showed superior therapeutic value for psoriasis. The anti-angiogenesis effect further reaffirms the importance of Dr. JRK's 777 oil in the management of inflammatory stage of psoriasis. Neo-genesis of blood vessel and further ramification are considered as one of the precursors that prelude inflammatory responses. Angiogenesis is the process where the formations of secondary and tertiary blood vessels are subdued. Dr. JRK's 777 oil did not possess fibroblast toxicity however it showed strong keratinocyte proliferation inhibition effect. The above findings suggest that the therapeutic effect of Dr. JRK's 777 oil is not based on producing cytotoxicity upon the keratinocytes but may be targeting early or mid-events of cell multiplication at nuclear level.

Our study clearly establishes that therapeutically effective herbal preparations can countermand the treatment success if the base oil quality is not ascertained or followed. During processing whether the base oil turns rancid also needs to be understood. However sacred and sacrosanct the ancient wisdom may be, judicious integration of modern science is inevitable to offer superior therapeutic benefit and prevent side effects.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: The study was approved by the institutional ethics committee*

## REFERENCES

1. Smith CH. Psoriasis and its management. BMJ. 2006;333(7564):380–4.
2. Griffiths CE, Barker JN. Pathogenesis and clinical features of psoriasis. Lancet. 2007;370(9583):263–71.
3. Ashcroft DM, Li Wan PA, Williams HC, Griffiths CEM. Systematic review of comparative efficacy and tolerability of calcipotriol in treating chronic plaque psoriasis. BMJ. 2000;320:963-7.
4. Griffiths CE, Clark CM, Chalmers RJ, Li Wan Po A, Williams HC. A systematic review of treatments for severe psoriasis. Health Technol Assess. 2000;4:1-125.
5. Whan B. Kim, MD. Diagnosis and management of psoriasis. Can Fam Physician. 2017;63(4):278–85.
6. El-Gammal A, Di Nardo V, Daaboul F, Tchernev G, Wollina U, Lotti J, et al. Is There a Place for Local Natural Treatment of Psoriasis? Open Access Maced J Med Sci. 2018;6(5):839–42.
7. Wahedi HM, Jeong M, Chae JK, Do SG, Yoon H, Kim SY. Aloesin from Aloe vera accelerates skin wound healing by modulating MAPK/Rho and Smad signaling pathways in vitro and in vivo. Phytomedicine. 2017;28:19–26.
8. Srivastava R. A review on phytochemical, pharmacological, and pharmacognostical profile of Wrightia tinctoria: Adulterant of kurchi. Pharmacogn Rev. 2014;8(15):36–44.
9. Sundarrajan S, Lulu S, Arumugam M. Deciphering the Mechanism of Action of Wrightia tinctoria for Psoriasis Based on Systems Pharmacology Approach. J Alternative Complementary Med. 2017;23(11):1-13.
10. Aruna V, Amruthavalli Gv, Gayathri R. Use of cosmetic products for treating certain diseases-Know the science. J Cosmetic Dermatol. 2018;1-5.
11. Ullah HMA, Zaman S, Juhara F, Akter L, Tareq SM, Masum EH, et al. Evaluation of antinociceptive, in-vivo & in-vitro anti-inflammatory activity of ethanolic extract of Curcuma zedoaria rhizome. BMC Complement Altern Med. 2014;14:346.
12. Gunapadam Part 2 Thathu-Jeeva Vagupu. Indian Medicine, Homeopathic Dept., 2004: 65–66.
13. Aruna V, Rajagopal G. Rancid Oil- The Hidden Villain of Psoriasis. Res Rev: A J Unani, Siddha Homeopathy. 2015;2(2):22–5.
14. Aruna V, Rajagopal G, Abbas A. Evidence for the Superior Quality and Efficacy of Dr. JRK's 777 Oil. Res Rev: A J Drug Design Discovery. 2017: 19-21.
15. Aruna V, Rajagopal G. Secret behind the Safety of the Siddha Products: An Insight from Metal Chelation. Res Rev: A J Unani, Siddha Homeopathy. 2016;3(2):24–7.

**Cite this article as:** Vadivel A, Venkata AG, Ravi S, Rajagopal G. Uniqueness of Dr. JRK's 777 oil and how rancidity distorts Wrightia tinctoria. Int J Res Dermatol 2019;5:618-24.