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Research Article

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A Review of Biological and Pharmacological activities from the aerial part of *Tamarisk. G*

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ABSTRACT

The genus Tamarisk belongs to the Tamaricaceae family, and is employed in the traditional medicine beneficent agent. The Tamarisk is found to be rich in polyphenolic compounds such as flavonoids, phenolic acids, tannins, saponins, terpenes and coumarins.
This paper reviews the important pharmaceutical and biological activities of secondary metabolites represented from Tamarisk gallica, which reported over many years. These include anti-cancer related activity, as well as anti-inflammatory and anti-analgesic, anti-oxidant, anti-bacterial activities and inhabitation of crystallization of calcium oxalate.

Keywords: *Tamarisk gallica*, pharmaceutical and biological activities, phytochemical screening, therapy, medicinal plants, secondary metabolites.

INTRODUCTION

The use of medicinal plants as a source of remedy to treat themselves or prevent diseases is originating in the millennia until the recent Chinese civilization, Indian and the Middle East. It is become certainly an act. The modern pharmaceuticals industry itself is still supports widely on the diversity of plant secondary metabolites to find new molecules to biological properties unpublished. This source seems inexhaustible since only a small part of the 400000 known plant species have been investigated on plans phytochemical and pharmacological, and that each species may contain up to several thousands of different constituents. [1]

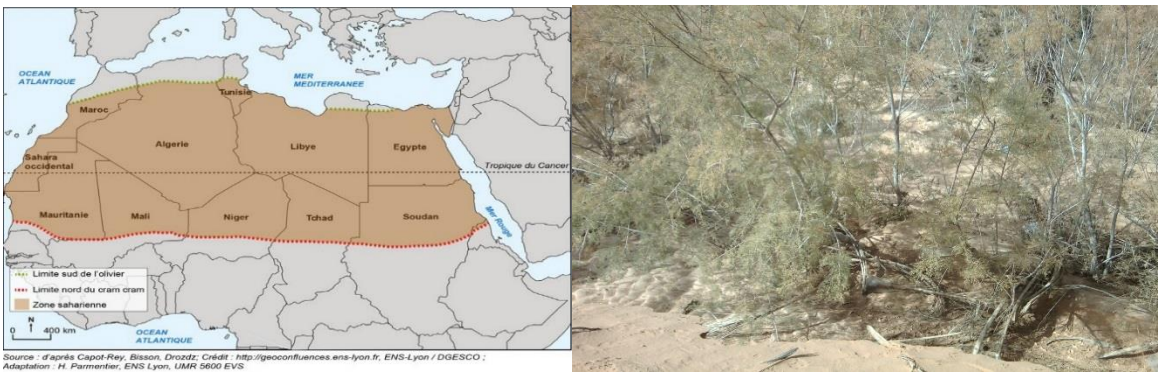

Fig 1. The geographical distribution of the genus Tamarisk in the area of northern Sahara.

The Tamarisk are trees or shrubs, frequent in salted soils, characterized by small leaves scaly, often nested, and giving the twigs the appearance of those of some junipers. The leaves are often punctuated by tiny holes corresponding to funnels at the bottom of which are placed the stomata and by where exudes a mucus containing salt and limestone. The roots are in general very developed; their wood contains vessels to large gauge. [2][3][4]

The flowers are grouped in cylindrical kittens that among some species of genus Tamarisk. We know sixty species of Tamarisk especially in the Mediterranean countries and the South Asia, in dry regions in particular. This kind plays an important role in North Africa and the northern Sahara, where it has about a dozen species of which two are particularly prevalent: T. articulata and *Tamarisk gallica*, designated in Arabic respectively under the names of "Thlaia" (more. "Ethel") and "Fersig" (more. "The aâriche") [5].

This species is characterized by:

-Elongated leaves, less nested, to punctuation little visible; dense kitten, position variable, the stamens in number equal to that of the petals.

- Small flowers, forming kittens of 3 to 4mm in diameter; spicules anthers, expanded bracts at the base.

Threads of the stamens inserted on the lobes of the disk; bracts completed by a long point exceeding the flowers, these are roses (sometimes white) [6] [7] [8].

Fig 2. The morphology of *Tamarisk gallica.*

Table 1. Classification

Parameters	
Kingdom	**Plantae – Plants**
Subkingdom	**Tracheobionta – Vascular plants**
Superdivision	**Spermatophyta – Seed plants**
Division	**Magnoliophyta – Flowering plants**
Class	**Magnoliopsida – Dicotyledons**
Subclass	**Dilleniidae**
Order	**Violales**
Family	**Tamaricaceae – Tamarix family**
Genus	**Genus Tamarix L. – tamarisk**

1. Phytochemical screening

In chemical valorization of the shrub *Tamarisk gallica*, the phytochemical study of this species has shown their richness in saponosides, steroids, terpenes, and unsaturated sterols revealed the strong presence of polyphenols especially in flavonoids and tannins. These last are widely present in the plant kingdom and their therapeutic activities [9] [10].

Table 2. The phytochemical screening of *Tamarisk gallica*

Plant material part	**The leaves**	**The barks**
The secondary metabolites		
Tannins and condensed tannins	+++	+++
Flavonoids	+++	-
Glycosides flavonoids	++	-
Heterosidiques flavonoids	++	-
Saponins	+	++
Cardinolides	-	-
Sterols and unsatured terpenes	+	+
Steroids and unsatured sterols	-	+
Alkaloids	-	-

'+' Presence, '++' Medium Presence, '+++' Strong Presence, '-'Absent

N.B: the rate of the presence depends on the speed of precipitation

Fig 3. The different organs of Tamarisk gallica (shoots, leaves and flowers) [06/02/2015 in the valley of Djenain Difallah, Bechar, Algeria]

1. Anti-inflammatory activity

Anti-inflammatory activity of *Tamarisk gallica* was tested by using Carrageenan induced rat paw edema and histamine induced rat paw edema:

1.1. Carrageenan induced rat paw edema

By using carrageenan induced rat paw edema model (Winter et al., 1962; Ahmed et al., 2004). Rats were randomly divided into four groups, each consisting of six animals. Group I was kept as control giving 1% (v/v) tween 80 solution in water, group II was kept as "positive control" and was given the standard drug Aspirin at a dose of 150

mg/kg of body weight; group III and IV were test groups, treated with extracts at the doses of 200 and 400 mg/kg of body weight respectively. Control vehicle, standard drop and the extracts were given orally 1 hour prior to the injection of 0.1 ml of 1% freshly prepared suspension of carrageenan.

The paw volume was measured by using a plethysmometer just before and 1, 2, 3, 4 and 5 h after the carrageenan injection. The percentage inhibition of the inflammation was calculated from the formula:

% inhibition = D_0-D_t/D_0 × 100 Where:
- D_0 Average inflammation (hind paw edema) of the control group of rats at a given time.

- D_t Average inflammation of the drug treated (i.e. extracts or reference diclofenac) rats at the same time [11] [12].

1.2. **Histamine induced rat paw edema**

The rats were divided into five groups of four per group. 0.1 ml of 1.0% histamine sulphate in normal saline (0.9% w/v NaCl) was injected to the sub plantar region of right hind paw15.Each group received one of the following treatments. Group I (control) received 10ml/kg, of normal saline (0.9% w/v, 0.1ml), Group II received standard drug diclofenac sodium 10 mg/kg, p.o, Group III received *Tamarisk gallica* 100 mg/kg, p.o, Group IV received Tamarisk gallica 200 mg/kg, p.o and Group V received *Tamarisk gallica* 300 mg/kg. The *Tamarisk gallica* was administered to the rat's 30 min before histamine injection. The percentage inflammation was calculated from the formula:

Where, D_t Average inflammation (hind paw edema) of the control group of rats at a given time, and D_0 Average inflammation of the drug treated (i.e. extracts or reference diclofenac) rats at the same time. Measured initially and at 1, 2, 3 and 4 hr. after histamine injection, using Plethysmograph) [13] [14] [15] [16].

1.2 Anti-analgesic activity

The anti-analgesic activity was provided by two different ways:

1.3 Central analgesic activity

The central analgesic activity was determined by radiant heat tail flick model in mice using analgesiometer (Inco, India).Experimental animals of either sex were randomly selected and divided into five groups designated as group-I, group-II, group-III, group-IV and group-V consisting of six mice in each group. Each group received a particular treatment i.e. control (Normal Saline 0.9%w/v, 10 ml/kg), positive control (Aspirin100 mg/kg, p.o) and the test sample (methanolic extract of 100mg/kg, & 200 mg/kg, 300mg/kg respectively). The tail flick latency was obtained thrice before drug administration and mean was used as pre drug latency. A cut off time of 10 sec was observed to prevent any tissue damage to the animal. The animal, which failed to withdraw its tail in 3-5 sec, was rejected from the study. The instrument's nichrome wire was heated to the required temperature and maintained by means of heat regulators. The strength of the current passing through the naked nichrome wire was kept constant at 4 Amps. The mice were kept in a holder with only the tail portion protruding. The tail was placed on the platform in such a way that the middle portion of the tail remained just above the hot wire but without touching it. The latency period (reaction time) was noted when the animal responded with a sudden and characteristic flick or tail lifting.

1.2. Analgesic effect by hot plate method

The hot plate test in mice was performed by Eddy's hot Plate method. Animals were individually placed on a hot plate maintained at a constant temperature (55°C) The animals were positioned on Eddy's hot Plate kept at a temperature of 55±0.5°C) and the reaction of animals, such as paw licking or jump response was taken at the end point. Experimental animals of either sex were randomly selected and divided into five groups designated as group-I, group-II, group III, group-IV and group-V consisting of six mice in each group for control, positive control and test samples. Each group received a particular treatment i.e. control, positive control. (Aspirin 100 mg/kg) and the test sample (methanolic extract of 100mg/kg, & 200 mg/kg, 300mg/kg, respectively). The animals were positioned on Eddy's hot Plate kept at a temperature of 55±0.5 °C. A cut off period of 15 s was observed to avoid damage to

the paw. The reaction time in control and treated animals was recorded at 0, 30, 60 min after the treatment [17] [18] [19] [20].

Fig 4. The hot plate test

4.1Anti-oxidant activity

4.2 Evaluation of total antioxidant capacity

Evaluation of total antioxidant capacity the assay is based on the reduction of Mo (VI) to Mo (V) by the extract and subsequent formation of a green phosphate/Mo (V) complex at acid pH (Prieto et al., 1999). An aliquot (0.1 ml) of plant extract was combined to 1 ml of reagent solution (0.6 M sulfuric acid, 0.028 M sodium phosphate and 0.004 M ammonium molybdate). The tubes were incubated in a thermal block at 95 °C for 90 min. After, the mixture had cooled to room temperature; the absorbance of each solution was measured at 695 nm (Anthelie Advanced 2, SECOMAN) against a blank. The antioxidant capacity was expressed as mg gallic acid equivalent per gram dry weight (mg GAE/g DW). The calibration curve range was 0–500 µg/ml. All samples were analyzed in triplicate [21] [22] [23] [24].

4.2 Scavenging ability on DPPH radical

DPPH quenching ability of organ extracts was measured according to Hanato et al. (1988). One milliliter of the extract at known concentrations was added to 0.25 ml of a DPPH methanolic solution. The mixture was shaken vigorously and left standing at room temperature for 30 min in the dark. The absorbance of the resulting solution was then measured at 517 nm and corresponded to the ability of extracts to reduce the stable radical DPPH to the yellow-colored diphenylpicrylhydrazine. The antiradical activity was expressed as IC_{50} (µg/ml), the extract dose required to cause a 50% inhibition. A lower IC_{50} value corresponds to a higher antioxidant activity of plant extract. The ability to scavenge the DPPH radical was calculated using the following equation:

DPPH scavenging effect (%) = $[[A_0 – A_1)]/A_0] \times 100$

Where A_0 is the absorbance of the control at 30 min, and A1 is the absorbance of the sample at 30 min. All samples were analyzed in triplicate [25] [26] [27] [28].

4.3 ABTS assay

ABTS radical-scavenging activity of extracts was determined according to Re et al. (1999). The ABTS+ cation radical was produced by the reaction between 5 ml of 0.014 M ABTS solution and 5 ml of 0.049 M potassium persulfate ($K_2S_2O_8$) solution, stored in the dark at room temperature for 16 h. Before use, this solution was diluted with ethanol to get an absorbance of 0.700 ± 0.020 at 734 nm. In a final volume of 1 ml, the reaction mixture comprised 950 µl of ABTS+ solution and 50µl of the plant extract at various concentrations. The reaction mixture was homogenized and its absorbance was recorded at 734 nm. Ethanol blanks were run in each assay, and all measurements were done after at least 6 min. Similarly, the reaction mixture of standard group was obtained by mixing 950µl of ABTS+ solution and 50 µl of TROLOX or BHT. As for the antiradical activity, ABTS scavenging

ability was expressed as IC_{50} µg/ml). The inhibition percentage of ABTS radical was calculated using the following formula:

ABTS scavenging effect (%) = $[[A_0 – A_1)]/A_0] \times 100$

Where A_0 and A_1 have the same meaning as in equilibrium state [29] [30] [31] [32].

4.4 Superoxide anion radical-scavenging activity

The measurement of O_2 scavenging capacity was assessed using the method of Duh et al. (1999). The reaction mixture contained 200 µl of phosphate buffer (pH 7.4), 200 µl of plant extract, 200 µl of PMS solution, 200 µl of NADH, and 200 µl of NBT. After incubation at ambient temperature, the absorbance was read at 560 nm. Assay of the antioxidant activity in plant extract was based on IC_{50} expressed as µg/ml. The inhibition percentage of superoxide anion generation was calculated using the following formula:

Superoxide quenching (%) = $[[A_0 – A_1)]/A_0] \times 100$

Where A_0 and A_1 have the same meaning as in equilibrium state [33] [34] [35] [36].

4.5 Chelating effect on ferrous ions

The ferrous ion chelating activity of *Tamarisk gallica* extracts was assessed as described by Zhao et al. (2006). Different concentrations of organ extracts were added to 0.05 ml of $FeCl_{24}H_2O$ solution (0.002 M) and left for incubation at room temperature for 5 min. Then, the reaction was initiated by adding 0.1 ml of ferrozine (0.005 M), and the mixture was adjusted to 3 ml with deionized water, shaken vigorously, and left standing at room temperature for 10 min. Absorbance of the solution was then measured spectrophotometrically at 562 nm. The percentage of inhibition of ferrozine–Fe2+ complex formation was calculated using the formula given bellow:

Metal chelating effect (%) = $[[A_0 – A_1)]/A_0] \times 100$

Where A_0 and A_1 have the same meaning as in Equilibrium state. Results were expressed as EC_{50}: efficient concentration corresponding to 50% ferrous iron chelating [37] [38] [39].

4.6 Iron reducing power

The capacity of plant extracts to reduce Fe3+ was assessed by the method of Oyaizu (1986). Methanol extract (1 ml) was mixed with 2.5 ml of sodium phosphate buffer (0.2 M, pH 6.6) and 2.5 ml of 1% potassium ferricyanide, and the mixture was incubated at 50 °C for 20 min. After that, 2.5 ml of 10% trichloroacetic acid were added, and the mixture was centrifuged at 650g for 10 min. The upper layer fraction (2.5 ml) was mixed with 2.5 ml of deionized water and 0.5 ml of ferric chloride and thoroughly mixed. The absorbance was measured at 700 nm and ascorbic acid was used as a positive control. A higher absorbance indicates a higher reducing power. EC_{50} value (mg/ml) is the effective concentration giving an absorbance of 0.5 for reducing power and was obtained from linear regression analysis.

4.7 β-Carotene bleaching test (BCBT)

A modification of the method described by Koleva et al. (2002) was employed. β-Carotene (2 mg) was dissolved in 20 ml chloroform and to 4 ml of this solution, linoleic acid (40 mg) and Tween 40 (400 mg) were added. Chloroform was evaporated under vacuum at 40 °C and 100 ml of oxygenated ultra-pure water was added, then the emulsion was vigorously shaken. Sample extract and reference compounds (BHT and BHA) were prepared in ethanol. An aliquot (150 µl) of the b-carotene: linoleic acid emulsion was distributed in each of the wells of 96-well

microtiter plates and ethanolic solutions of the test samples (10 µl) were added. Three replicates were prepared for each of the samples. The microtiter plates were incubated at 50 °C for 120 min, and the absorbance was measured using a model EAR 400 microtiter reader (Lab systems Multiskan MS) at 470 nm. Readings of all samples were performed immediately (t = 0 min) and after 120 min of incubation. The antioxidant activity (AA) of the extracts was evaluated in term of b-carotene blanching using the following formula:

$AA\ (\%) = [[A_0 – A_1)]/A_0] \times 100$

Where A0 and A1 have the same meaning as in Equilibrium state. The results are expressed as IC_{50} values (µg/ml) [40].

4.8 Inhibition of lipid oxidation

The ferric thiocyanate (FTC) method of Sanchez-Moreno et al. (1999) used a linoleic acid model to evaluate inhibition of lipid oxidation. Leaf and flower extracts (0.5 ml) at different concentrations were mixed with an emulsion of 2.5% linoleic acid in absolute ethanol (0.5 ml), 0.05 M phosphate buffer pH 7 (1 ml) and distilled water (0.5 ml), shaken and incubated (in an oven) at 40 °C in the dark. The same mixture without sample extract was used as negative control. At two time intervals (0–5 days), 0.1 ml of each tube was transferred to other tube containing 9.7 ml of 75% ethanol and 0.1 ml of 30% ammonium thiocyanate followed by the addition of 0.1 ml of 0.02 M ferrous chloride (prepared in 3.5% hydrochloric acid). After 3 min, the absorbance was measured at 500 nm. Every assay was done in triplicate [41].

5. Antibacterial activity

Table 3 shows the antibacterial activities of leaves and flowers measured by the agar diffusion method against selected pathogenic bacteria. The mean inhibition zone for all bacteria treated with *Tamarisk gallica* extracts increased from 0 to 6.5 mm when the concentration increased from 2 to 100 mg/l. Flower extracts were more active against bacteria as compared to leaf ones. Indeed, mean inhibition zone for all bacteria was 4 and 2.7 mm, respectively, for flowers and leaves. In the two organs evaluated for their effect on bacterial growth, the inhibition zones around bacteria increased with the extract concentration, except for P. aeruginosa. The strongest activity of *Tamarisk gallica* was recorded against M. luteus and the lowest activity was observed against E. coli. Concerning antifungal tests, both organ extracts failed to show any activity against all Candida excepting the highest extract concentration (100 mg/ml) which showed weak to moderate activity against the tested Candida. These results suggest that methanolic extracts of *Tamarisk gallica* were more efficient to inhibit bacterial growth than fungal one, probably in relation to their active molecules. Several studies attributed the inhibitory effect of plant extracts against bacterial pathogens to their phenolic composition (Baydar et al., 2004; Rodriguez Vaquero et al., 2007) [42].

Table 3. Antibacterial activity of *Tamarisk gallica* leaf and flower extracts at different concentrations (mg/ml). Inhibition zone calculated in diameter around the disc (mm ± SD).

Bacterial strains	mg/ml	Leaves	Flowers	Control (+) gentamycin (10UI)	Mean of inhibition zone
S. aureus, ATCC25923	100 4 2	8.66 ± 0.33 7.66 ± 0.33 7.00 ± 0.00	10.00 ± 0.00 10.66 ± 0.330 9.66 ± 0.33	22 22 22	3.5 3.5 2.75
S. epidermidis, CIP106510	100 4 2	9.33 ± 0.66 7.33 ± 0.66 7.00 ± 0.00	11.00 ± 0.00 10.33 ± 0.66 8.33 ± 0.66	30 30 30	4 3.5 2
Micrococcus luteus, NCIMB 8166	100 4 2	9.33 ± 0.66 7.66 ± 0.33 7.00 ± 0.00	15.00 ± 0.00 13.00 ± 0.00 12.33 ± 0.66	26 26 26	6.5 4.5 4
E. coli, ATCC 35218	100 4 2	8.00 ± 0.00 6.00 ± 0.00 6.00 ± 0.00	8.00 ± 0.00 6.00 ± 0.00 6.00 ± 0.00	27 27 27	2 0 0
P. aeruginosa, ATCC 27853	100 4 2	8.00 ± 0.00 12.33 ± 0.66 10.00 ± 0.00	10.33 ± 0.66 10.00 ± 0.00 10.00 ± 0.00	16 16 16	2.5 5.5 4.5
Mean of inhibition zone		2.72	4.025		

No antimicrobial activity (), inhibition zone <1 mm. Weak antimicrobial activity (w), inhibition zone = 1 mm. Slight antimicrobial activity (+), inhibition zone 2–3 mm. Moderate antimicrobial activity (++), inhibition zone 4–5 mm. High antimicrobial activity (+++), inhibition zone 6–9 mm. Strong antimicrobial activity (++++), inhibition zone >9 mm. Standard deviation ± 0.5 mm. For all bacteria, the inhibition zone of the control (+) gentamycin (10 UI) was higher than 9 mm (++++). The diameter of disc was 6 mm. SD: standard deviation.

The inhibitory effect of these phenolic could be explained by adsorption to cell membranes, interaction with enzymes, substrate and metal ion deprivation (Scalbert, 1991). These results suggest that the antibacterial capacity needed, as compared to antioxidant activity, which has a good efficiency with crude extracts of *Tamarisk gallica*, more concentration and even purification of phenolic compounds. Purified components may be used as natural antimicrobials in food systems, as well as to prevent the growth of food borne bacteria resulting in extension of the shelf life of processed foods [44] [45].

6. Anti-Cancer activity on human colon cancer cells

Colorectal cancer is one of the major causes of cancer related mortality in the populations of developed countries (Mao et al. 2011). Recently, research has been focusing on the cell-cycle arrest analysis as a major approach for the cancer eradication (Cardenas et al. 2008; Liu et al. 2008). A number of Cdk/cyclin protein family, which characterize the checkpoint controls of the cell division (Murray 2004; Wu et al. 2006), regulates the cell-cycle progression. *Tamarisk gallica* extracts prevent the progression of liver cancer by restoring the level of antioxidant enzymes in rat liver (Sehrawat and Sultana 2006). Until now, there is no available information regarding the anticancer effects of *Tamarisk gallica* on human colon cancer.

6.1 Cell cycle and cell viability analysis

Caco-2 cells (2 9 104 cells/ml) were exposed to 100 µg/ml of *Tamarisk gallica* extracts for 72 h and both suspended and attached cells were collected. The cells were washed with PBS, fixed with 70 % ice-cold ethanol and stored at -

20 C until analysis. Before measurement, ethanol was removed; the cells were suspended in 500 µl cell cycle reagent (Guava technologies, Hayward, CA, USA) and incubated in the dark at room temperature for 30 min. Cell cycle
distribution and cell viability analysis were determined by Guava flow cytometry (Guava Technologies) [46].

6.2 DAPI staining

After 72 h treatment with extracts of *Tamarisk gallica* shoots, flowers and leaves (100 and 200 µg/ml), Caco-2 cells at the concentration of 2 $\times 10^4$ cells/ml were washed two times with PBS. Then, the cells were fixed with 3.7 % formaldehyde in PBS for 10 min at room temperature. After washing two times with PBS and staining with DAPI (40,6-diamidino-2-phenylindole)
solution, cells were analyzed using fluorescence microscopy.

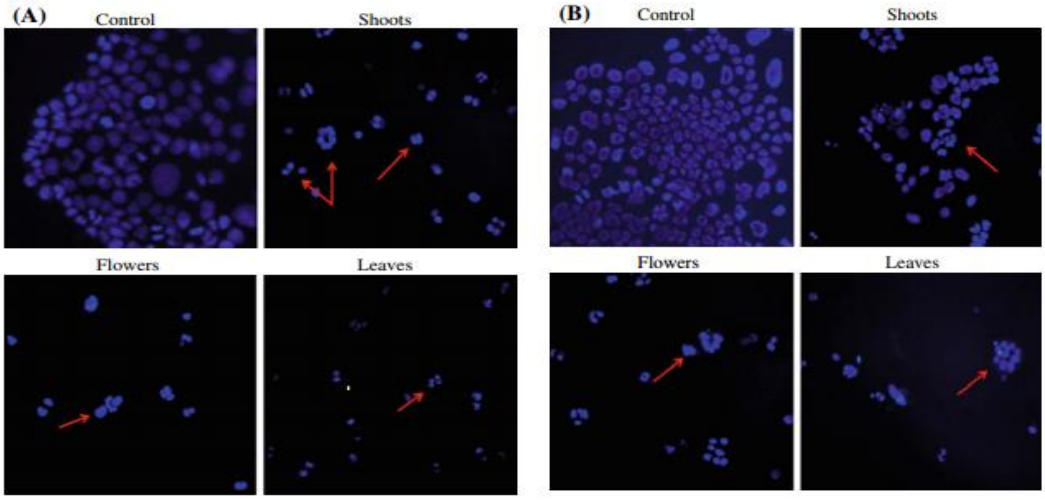

Fig 5. DAPI staining of Caco-2 cancer cells treated with *Tamarisk gallica*. Cells were incubated for 72 h with *T. gallica* plant extracts (shoots, flowers and leaves) at a concentration of 100 (a) and 200 µg/ml (b). After incubation, the nuclear morphologies of the cells were examined using a fluorescent DNA-binding agent, DAPI. DNA was analyzed using fluorescence microscopy. Arrows indicate mitotic cells with condensed chromosomes and no nuclear membrane. All photographs were taken at 2009 magnification.

6.1. Western blotting

Caco-2 cells (2 $\times 10^4$ cells/ml) were incubated with 100 µg/ml shoot, flower and leaf extracts of *Tamarisk gallica* during 72 h. Then, cells were washed in PBS and lysed by RIPA buffer (Sigma Aldrich Co) with protease inhibitor cocktail (Sigma Aldrich Co). The mixture was centrifuged at 12,000-×g for 20 min at 4 °C. The protein-containing supernatant was kept and the quantification of the proteins was performed using Plus 1 2D Quant kit (GE Healthcare, Piscataway, NJ, USA). Proteins (20 µg) were resolved on 12 % sodium dodecyl sulfate–polyacrylamide gel electrophoresis and transferred onto nitrocellulose membrane using blot dry blotting system (Invitrogen/Life Technologies, Carlsbad, CA, USA). After blocking with 5 % non-dry fat milk, membrane was incubated at 4 °C overnight under shaking with primary antibodies anti-p38, b-actin (Sigma Aldrich Co), phosphoErk1/2, Erk1/2, pp38, Cyclin B1, pChk1, Chk1, pChk2 and Chk2 (Santa Cruz Biotechnology, Santa Cruz, CA, USA). Horseradish peroxidase-conjugated secondary antibodies using enhanced chemiluminescence system ECL (Amersham Biosciences/GE Healthcare) [26] detected the bands [47].

Table 4. Effect of different organ extracts of *Tamarisk gallica* on the cell cycle arrest. Cells were treated with shoot, flower and leaf extracts of *T. gallica* at a concentration of 100 µg/ml in order.

	control	shoots	flowers	leaves
G0/G1	42.4 ± 7.07	37.8 ± 2.12	36.6 ± 2.83	37.25 ± 6.86
S	17.65 ± 2.05	13.8 ± 2.4	14.45 ± 2.33	14.05 ± 2.9
G2/M	38.95 ± 5.02	47.3 ± 4.38*	48.1 ± 4.95*	47.1 ± 8.91*
Sub-G0	1.05 ± 0.07	1.1 ± 0.14	0.85 ± 0.21	1.55 ± 0.92

The treatment time was 72 h. Data of three independent experiments are presented as mean ± SD. * Statistical significance (p\ 0.05) between treated and control cells.

7. Inhibition of crystallization of calcium oxalate

The crystals of calcium oxalate (CaOx) are the primary constituent of more than 60% of the majority of human kidney stones; they exist in the form of CaOx monohydrate (COM) and CaOx dehydrate (COD). After the preparation of the extract, we have followed three ways of study but in this work, we have used the model turbidimetric.

-Turbidimetric study

- Study without inhibitor

- Study with inhibitor

the crystallization studies of calcium oxalate (CaOx) have been an interest to the researchers and urologists for many years The use of some natural substances as inhibitors in the formation of nephrolithiasis allowed us to study the inhibitor effect of the acid fraction of the extract of *Tamarisk gallica* on the inhibition of COD crystal. The Precipitation of calcium oxalate at 37C and pH 6.5 has been studied by the measurement of turbidity at 620 nm. A spectrophotometer UV/Vis (Beckman Du series 520) was employed to measure the turbidity of the formation of calcium oxalate. Pure chemicals, including calcium chloride dehydrate CaCl2, 2H2O (Riedel–De Hae ¨n), sodium oxalate Na2C2O4 (Riedel–De Haenag) and sodium chloride NaCl (Fluka), are used for this study [48].

The maximum values of the variation of absorbance, and the turbidimetric slopes relating to the curves of crystallization without and with inhibitors (10, 50, and 100%) are gathered in Table 5.

Table 5. The maximum values of the variation of absorbance, and the turbidimetric slopes relating to the curves of crystallization without and with inhibitors

CI (%)	TS	I (%)	ΔD	R 2	Cv (%)
0	0.517	00.00	0.399	0.856	4.79
10	0.148	71.37	0.206	0.924	4.00
50	0.101	80.46	0.181	0.986	4.19
100	0.079	84.71	0.140	0.940	6.84

CI concentration of inhibitor, TS turbidimetric slope, R^2 linear regression of the quadruplicate data, Cv (%) coefficient of variation, ΔD variation of absorbance, I percentage of inhibition.

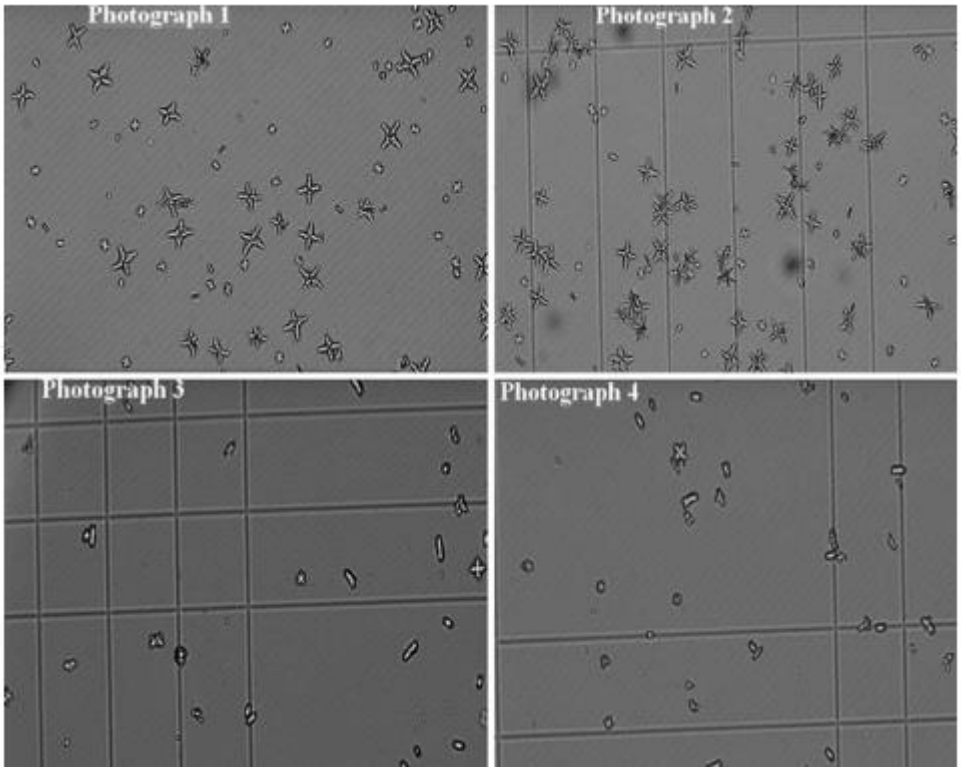

Fig 6. Photographs of crystallization for tries without and with inhibitor.

The various results obtained in our work (Turbidimetric slope, absorbance and percentage of inhibition) show that the acid fraction of *Tamarisk gallica* has an important inhibiting effect in vitro due to the effect of the functions of acid. On the other hand, the mechanism of action of the inhibitors being related to its chemical composition. The photographs obtained by microscope, show clearly that the acid compounds manifests at the stage of growth [49].

In the following table shows the actual therapeutic remedies of this plant

Table 6. Traditional Use and therapeutic indications of the species *Tamarisk gallica*

Uses	Usual Part	Administration	Process of treatment
Diarrhea	Powder of barks	oral	Grinding
Hemostasis	Powder of barks	External use	Grinding
Cold	leaves	oral	decoction
Diseases of the skin	Aerial part	A poultice	decoction
Abortion	leaves	oral	infusion
Sudorifique	Aerial part	oral	decoction
Tonsillitis	Aerial part	oral	decoction
Affection of the eyes	Aerial part	A poultice	decoction
Hemorrhoids	Powder of barks	A poultice	Grinding
Gingivitis	barks	In gargle	decoction
The Digestive Diseases	leaves	oral	Infusion/decoction

Bacterial Infection	Aerial part	Oral with the meat of goat	Grinding in powder

Conclusion

The current review indicates that Tamarisk gallica is a highly useful plant and have already found its application in wide array of medicines to cure several diseases, such as rheumatism, diarrhea and gingivitis …etc. to mention a few. It is however found that not all phytoconstituents present in the plant have been studied in depth and needs be explored more. In future study, the isolated bioactive substances principles from Tamarisk gallica needs to be evaluated in scientific manner using specific experimental animal models and clinical trials to understand further the medicinal activities of the plant.

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