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ANTICANCER POTENTIAL OF *SOLANUM JASMINOIDES*

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Keywords:

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ABSTRACT: Objective: To investigate the anticancer potential of the extracts and the alkaloidal and nonalkaloidal fractions of *Solanum jasminoides* Paxt using *in-vitro* and *in-vivo* models. **Method:** The petroleum ether, methanol, and water extracts of the whole plant were prepared by successive soxhlet method. The methanol extract was treated with solvents to separate alkaloidal and non-alkaloidal fractions. All the samples were subjected to *in-vitro* anticancer studies using Vero, BRL-3, HEp-2, HeLa, MDCK, and A-549 cell lines. Based on the *in-vitro* results, the alkaloidal fraction was selected and subjected to *in-vivo* anticancer activity against DLA and EAC induced ascities in mice. **Results:** The results reveal that the extracts and the fractions show moderate to good *in-vitro* anticancer activity, and the alkaloidal fraction shows superior activity. In the *in-vivo* study, the alkaloidal fraction shows a dose dependent effect on changes in the body weight, mortality, and clinical symptoms induced by cancer cells. **Conclusion:** The results of the present study indicate that the alkaloidal fraction of the plant possesses good, both *in-vitro* and *in-vivo*, anticancer potential.

INTRODUCTION: Considerable efforts are being made to identify naturally occurring anticancer agents who would prevent, slow and reverse cancer induction and its subsequent development [1]. Plants contain abundant quantities of these compounds, and several plant-derived compounds are currently employed successfully in treating cancer. A major group of these compounds is the powerful antioxidants. And the rest include reactive groups that confer the protective properties.

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Anticancer agents from plants currently in clinical use can be categorized into four main classes, namely *Vinca* alkaloids, epipodophyllotoxins, taxanes and camptothecin [2]. The plant *Solanum jasminoides* Paxt (Family: Solanaceae) is a native of South America, introduced and found in the roadsides and waste places in the tempest regions of the Nilgiris, Tamil Nadu, India, at 1500-2500 m altitude regions. The leaves and fruits contain glycoalkaloids [3]. The present study was undertaken to investigate the anticancer potential of successive extracts and the isolated fractions of the plant.

MATERIALS AND METHODS:

Plant Material: The whole plant was collected from in and around the Nilgiris forests, Tamilnadu, India. In June 2004, and authenticated by the Survey of Medicinal Plants and Collection Unit,

Govt. of India, Ootacamund, India. A voucher specimen (TIFAC 03) has been deposited for future reference at the college herbarium.

Chemicals: 3-(4,5 Dimethyl thiazole-2-yl)-5-diphenyl tetrazolium bromide (MTT), and sulphorhodamine B (SRB) were obtained from Sigma–Aldrich Co., St. Louis, USA. 5-Fluorouracil (purity≥99%) was obtained from Ranbaxy Ltd., Gurgaon, India. All other chemicals used in the study were of analytical grade and purchased from E-Merck, Ltd., Mumbai, India.

Animals: Healthy adult Swiss albino mice weighing (25-30 g) were obtained from the animal house, J.S.S. College of Pharmacy, Ootacamund, India, and maintained under standard environmental conditions (22–28 ºC, 60–70% relative humidity, 12-h dark: 12-h light cycle). They were fed with standard rat feed (M/S Hindustan Lever Ltd., Bangalore, India) and water *ad libitum*. The animals were housed in large cages.

The experiments were conducted with prior approval of the Institutional Animal Ethics Committee (approval no. JSSCP/IAEC/Ph.D/PH. Chemistry/01/2005–2006).

Extraction Procedure and Phytochemical Analysis: The whole plant was chopped to small pieces and dried in the shade. The dried sample (100 g) was extracted successively with 600 mL each of petroleum ether (60–80 ºC), methanol and water in a Soxhlet extractor for 18–20 h. The successive petroleum ether, methanol, and water extracts will henceforth be called SJP, SJM, SJW, respectively. All the extracts were subjected to qualitative phytochemical analysis using standard tests [4].

Isolation of Total Alkaloids from SJM: The *Solanaceae* family is known to possess glycoalkaloids [3]. The total alkaloidal fraction was, therefore, isolated from SJM. The SJM was treated with hydrochloric acid (3%, HCl) and kept overnight. It was then filtered through a Whatman filter paper, and the next day the residue was washed several times with 3% HCl (100 ml). The filtrate was extracted with chloroform (300 ml, 4 times), and the chloroform extracts were combined and dried (non alkaloid fraction). The acid layer was treated with ammonium hydroxide solution (50%) to make it alkaline (>pH 10). The precipitated alkaloids were further extracted in chloroform (250 ml each, 5 times). The chloroform extracts were combined**,** filtered, and concentrated to dryness under reduced pressure and controlled temperature (40–50 ºC). A pale yellow powder (0.5312 g, 0.53 %) was obtained (alkaloid fraction). Henceforth, the total alkaloidal and non-alkaloidal fraction of SJM will be called as SJM-A and SJM-NA, respectively.

Cell Lines and Culture Medium: Vero (normal fibroblast-like African green monkey cell), BRL-3 (normal fibroblast-like rat liver cell) and HEp-2(human laryngeal epithelial carcinoma) cell cultures were procured from National Centre for Cell Sciences (NCCS), Pune, India. HeLa (Epithelial human cervix cancer) and MDCK (dog kidney) cell lines of both normal and cancerous cell lines were used for the comparison purposes, are procured from V.P. Chest Institute, New Delhi, India. A-549 (human non-small cell lung carcinoma) cells were obtained from Christian Medical College, Vellore, India. Stock cells of all cell lines were cultured in DMEM supplemented with 10% inactivated FBS/NBCS, penicillin (100 IU/mL), streptomycin (100µg/mL) and amphotericin B (5µg/mL) in a humidified atmosphere of 5% CO_2 at 37 ºC until confluent.

The cells were dissociated with TPVG solution (0.2% trypsin, 0.02% EDTA, 0.05% glucose in PBS). The stock cultures were grown in 25 cm^2 tissue culture flasks and all the experiments were carried out in 96 well microtitre plates. DLA and EAC cells were procured from Amla Cancer Research center, Thrissur, Kerala, India. The cells were maintained *in-vivo* in Swiss albino mice by intraperitoneal transplantation. EAC cells aspirated from the peritoneal cavity of mice were washed with saline and given intraperitoneally to develop an ascitic tumor.

Preparation of Suspension and Solutions: For *in-vitro* studies each extract (10mg) was separately dissolved in 1 mL of dimethyl sulphoxide (DMSO), and the volume was made up to 10 mL with maintenance medium to obtain a stock solution of 1 mg/mL concentration and sterilized by filtration. Further dilutions were made from the stock and used for short and long term cytotoxicity studies.

For *in-vivo* studies SJP, SJM, SJW, SJM-A, SJM-NA, and standard 5-FU were suspended in sodium carboxymethyl-cellulose (CMC, 0.3%) and administered orally to the animals with the help of an intragastric catheter.

Cytotoxicity Studies: The cytotoxicity assay was carried out using 0.1 mL of cell suspension, containing 10,000 cells seeded in each well of a 96 well microtitre plate (Tarson India Pvt., Ltd., Kolkata). Fresh medium containing different concentrations of the test sample was added after 24 h of seeding. Control cells were incubated without the test sample. The microtitre plate was incubated at 37 ºC in a humidified incubator with 5% CO_2 for a period of 72 h. Sixteen wells were used for each concentration of the test sample. The CTC_{50} was determined by the standard MTT [5] and SRB assays [6].

Short Term Cytotoxicity Studies: The short term toxicity studies were carried out against DLA and EAC cells using standard procedures [7]. The long term toxicity study was carried out by the method of Freshney [8]. A series of cultures were prepared in 25 cm^2 tissue culture flasks, three for each concentration and three control of seeding 2×10^5 cells in 4 mL of growth medium and incubating at 37 ºC for 48 h. Long term survival studies against normal (Vero) and cancer (HEp-2) cell cultures were carried out, and the regeneration capacity of the cells was estimated by standard clonogenic assay. Three nontoxic concentrations of each extract were tested for their reduction in regenerative capacity, in comparison with untreated cell control. Among all the tested samples, SJM-A showed well *in-vitro* cytotoxicity against all cancer cells *in-vitro*. SJM-A was, therefore, selected to study it's *in-vivo* anticancer activity in different tumor models.

Effect of SJM-A on Mice Bearing DLA Cells Swiss Albino mice were divided into five groups with six animals in each group. All the animals were injected with DLA cells (1×10^6) intraperitoneally except the normal group. This was taken as day zero. Group, I served as normal control and group II as tumor control. These two groups received sodium CMC suspension (0.3%) administered orally. Group III served as a positive control and was treated with standard 5-Fluorouracil (5-FU) at 20 mg/kg per oral route. Group IV and V were treated with SJM-A at a dose of 25 and 100 mg/kg per oral route, respectively. All these treatments were given 24 h after the DLA cells were inoculated, once daily for 10 days. The clinical signs, mortality, and body weight changes were recorded throughout the study period.

Effect of SJM-A on Mice Bearing EAC Cells: Swiss Albino mice were divided into five groups, with six animals in each group. All the animals were injected with EAC cells (2×10^6) intraperitoneally except the normal group. Group, I served as normal control and group II as tumor control. These two groups received sodium CMC suspension (0.3%), orally. Group III served as a positive control and was treated with standard, 5-Fluorouracil (5-FU) at 20 mg/kg p.o. Group IV and V were treated with SJM-A at a dose of 25 and 100 mg/kg p.o., respectively. All these treatments were given 24 h after the tumor inoculation, once daily for 10 days. The clinical signs, mortality, and body weight changes were recorded throughout the study period.

Statistical Analysis: The *in-vivo* data was analyzed by One-Way Analysis of Variance (ANOVA) followed by Turkey-Kramer multiple comparison tests, and $p<0.05$ was considered significant.

RESULTS: Qualitative phytochemical analysis revealed that SJP contains steroids, tannins, and fatty acid. SJM contains alkaloids, carbohydrates, flavonoids, glycosides, tannins, and triterpenoids. SJW contains carbohydrates, glycosides, and tannins. The results of the *in-vitro* cytotoxicity studies are shown in **Table 1.**

The results reveal that among the samples tested SJM-A shows significant cytotoxicity with CTC_{50} value 64.84, 71.28, 78.16, 111.29, 116.62, 132.84 µg/mL towards A-549, HEp-2, BRL-3A, HeLa, Vero, and MDCK, respectively. The results of the *in-vitro* short term toxicity studies are shown in **Table 2**.

The data reveal that SJM-A has moderate toxicity against DLA and EAC cells, whereas others do not show any significant toxicity even at higher concentrations. The results of the long term survival studies are shown in **Table 3**. The data reveal that in Vero cell culture, treatment with 100

µg/ml of SJM-A for 72 h, the total number of colonies reduces to 14 ± 0.11 when compared to cell control (62 ± 3.25), but treatment with 100 µg/mL of SJM-NA the total number of colonies reduces to only 21 ± 1.83. In HEp-2 cell culture. After 72 h of treatment with 50 µg/mL of SJM-A, the total number of colonies reduces to 16 ± 0.11 when compared to cell control (63 ± 2.60), but treatment with SJM-NA the total number of colonies reduces to only 19 ± 1.83. SJM-A thus shows good cytotoxicity against all cancer cells cultures. SJM-A also shows moderate activity in long term toxicity studies by clonogenic assay. SJM-A was, therefore, selected for *in-vivo* anticancer activity in different tumor models.

TABLE 1: CYTOTOXIC ACTIVITY OF SJP, SJM, SJW, SJM-A, AND SJM-NA AGAINST NORMAL AND CANCER CELL CULTURES

Cell Cultures	SJP			SJM			SJW			SJM-A			SJM-NA		
	MTT	SRB	Avg.	MTT	SRB	Avg.	MTT	SRB	Avg.	MTT	SRB	Avg.	MTT	SRB	Avg.
Vero	656.58 ±19.29	595.60 ±13.50	626.09	325.34 ±14.38	345.01 ±20.45	335.18	332.85 ±16.34	349.46 ±17.76	341.16	123.12 ±5.37	110.11 ± 4.93	116.62	183.32 ±6.26	191.63 ± 7.27	187.48
BRL-3A	589.32 ±12.64	565.00 ±7.56	577.16	297.92 ±13.17	304.8 ±10.87	301.37	165.35 ±7.36	173.46 ± 8.76	169.41	81.56 ±3.16	74.75 ±2.27	78.16	182.23 ±5.14	176.22 ± 5.24	179.23
MDCK	472.00 ±3.56	501.85 ±18.65	486.92	245.91 ±10.14	271.82 ±14.78	258.86	172.28 ±8.42	181.91 ± 7.21	177.10	124.35 ±4.37	141.32 ±5.95	132.84	166.49 ±4.32	166.19 ± 7.44	166.34
HeLa	512.85 ±6.89	494.76 ±12.50	503.80	132.83 ±4.39	123.86 ±5.72	126.89	165.23 ±7.13	156.93 ± 6.74	161.08	113.33 ±5.36	109.26 ± 4.57	111.29	148.16 ±6.37	151.20 ± 7.27	149.68
HEp-2	456.80 ±11.58	479.2 ±6.72	468.02	133.49 ±4.28	125.40 ±5.26	129.46	153.36 ±7.23	162.23 ±6.86	157.79	76.29 ±3.42	66.27 ±3.45	71.28	90.93 ±3.92	102.89 ± 4.11	96.91
A-549	549.50 ±4.02	515.32 ±1.52	532.41	312.43 ±4.36	321.82 ±15.22	317.13	134.92 ±5.88	144.99 ± 6.27	146.99	59.25 ±3.11	70.46 ±3.91	64.86	88.35 ±4.63	82.13 ± 3.74	85.24

Values are mean ± SEM, n=6

TABLE 2: SHORT TERM *IN VITRO* CYTOTOXICITY OF SJP, SJM, SJW, SJM-A, AND SJM-NA ON DLA AND EAC CELLS

DLA			EAC		
Extract & Fraction	Conc. used (µg/mL)	CTC_{50}	Extract & Fraction	Conc. used (µg/mL)	CTC_{50}
	1000			1000	
SJP	500	>500	SJP	500	>500
	250			250	
	500			500	
SJM	250	>500	SJM	250	>500
	125			125	
	500			500	
SJW	250	>500	SJW	250	>500
	125			125	
	500			500	
SJM-A	250	175.15 ± 7.83	SJM-A	250	265.15 ± 5.09
	125			125	
SJM-NA	500		SJM-NA	500	
	250	>500		250	>500
	125			125	

Values are mean ± SEM, n=6

TABLE 3: CYTOTOXICITY OF SJM-A AND SJM-NA BY LONG TERM SURVIVAL STUDY ON NORMAL VERO CELL CULTURES AND CANCER HEP-2 CELL CULTURES

Normal Vero cell cultures				Cancer HEp-2 cells cultures			
Isolated Fraction	Conc. Used (µg/mL)	Avg. No. of Colonies ± S.E.M.*	CTC_{50}	Isolated Fraction	Conc. Used (µg/mL)	Avg. No. of Colonies ± S.E.M.*	CTC_{50}
	100	14 ± 0.11			50	16 ± 0.11	
SJM-A	50	27 ± 1.42	116.62	SJM-A	25	34 ± 1.42	71.28
	25	55 ± 2.29			12.5	55 ± 2.29	
	200	15 ± 0.04			100	17 ± 0.04	
SJM-NA	100	21 ± 1.83	187.48	SJM-NA	50	19 ± 1.83	96.91
	50	43 ± 2.37			25	37 ± 2.37	
Cell control		62 ± 3.25		Cell control		63 ± 2.60	

*Values are mean ± SEM, n=6

The effects of SJM-A on the average life span of DLA bearing mice are shown in **Fig. 1**.

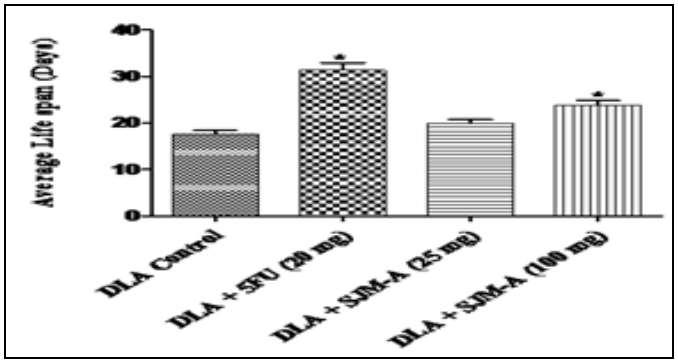

FIG. 1: EFFECT OF SJM-A ON AVERAGE LIFE SPAN OF DLA TUMOR BEARING MICE. Values are mean ±SEM, * p<0.05 when compared to DLA control, n=6

The data reveal that treatment with SJM-A at 100 mg/kg significantly ($p \leq 0.05$) increases the average life span (ALS) of DLA bearing mice from 17.59 ± 0.82 to 23.86 ± 0.99 days. The standard 5-FU at 20 mg/ kg significantly ($p \leq 0.001$) increases the life span to 31.36 ± 1.59 days. SJM-A at low doses does not show any significant increase in ALS. The effect of SJM-A on the % increase in body weight of DLA bearing mice is shown in **Fig. 2.**

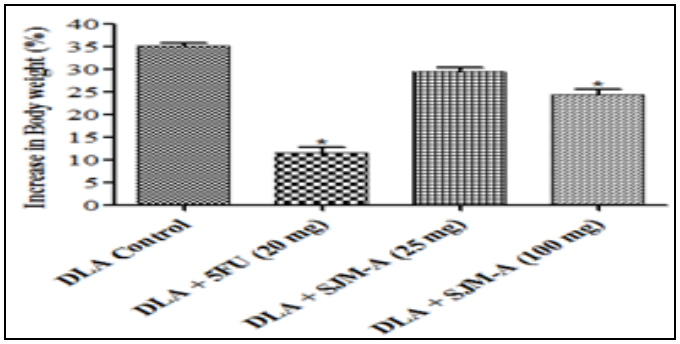

FIG. 2: EFFECT OF SJM-A ON % INCREASE BODY WEIGHT OF DLA TUMOR BEARING MICE. Values are mean ±SEM, * p<0.05 when compared to DLA control, n=6

The data reveal that treatment with SJM-A has a significant reduction in the percentage increase in body weight (p<0.001) when compared to DLA control. The standard 5-FU, however, is more potent in inhibiting the increase in body weight of tumor-bearing mice. The data reveal that the treatment with SJM-A at 100 mg/kg significantly ($p \leq 0.01$) increases the average life span (ALS) of EAC bearing mice when compared to EAC tumor control group. This is comparable to standard 5-FU at 20 mg/kg. SJM-A at 25 mg/kg also shows a moderate increase ($p \leq 0.01$ to $p \leq 0.05$) in ALS, namely, 20.55 ± 1.75.

The effect of SJM-A on the average life span (ALS) of EAC tumor-bearing mice is shown in **Fig. 3**.

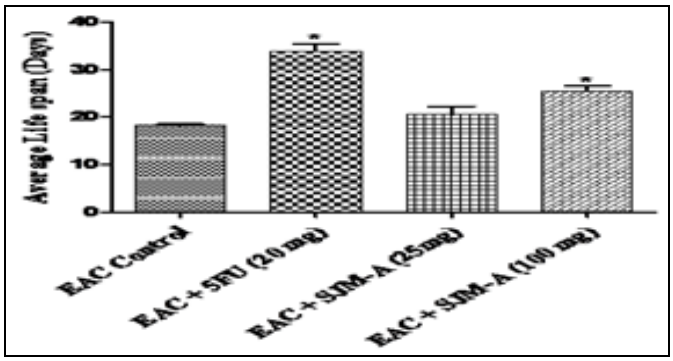

FIG. 3: EFFECT OF SJM-A ON AVERAGE LIFE SPAN OF EAC TUMOR BEARING MICE. Values are mean ±SEM, * p<0.05 when compared to DLA control, n=6

The effect of SJM-A on the percentage increase in body weight of EAC tumor-bearing mice is given in **Fig. 4.**

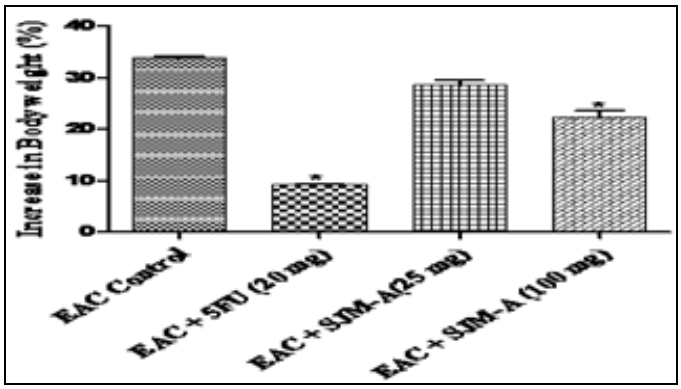

FIG. 4: EFFECT OF SJM-A ON % INCREASE BODY WEIGHT OF EAC TUMOR BEARING MICE. Values are mean ±SEM, * p<0.05 when compared to DLA control, n=6

The data reveal that treatment with SJM-A (at both dose 25, 100 mg/kg) has a significant reduction in percent increase in body weight ($p \leq 0.001$) when compared to EAC control. The standard 5-FU, however, is found to be more potent in inhibiting the percentage increase in body weight of tumor-bearing mice.

DISCUSSION: Alkaloids represent a very extensive group of secondary metabolites, with diverse structures, distributed in nature with important biological activities. The cyclic compound containing nitrogen in a negative oxidation state is distributed to a limited extent in living organisms [9]. However, strong physiological effects of some alkaloids present opportunities for

utilizing them in human medicine. Although a number of the polyhydroxylated alkaloids have been reported to show anticancer activity, recent research has concentrated on developing alkaloids as drug candidates for the management of human malignancies. There is considerable evidence that alkaloids enhance the natural antitumor defenses of the body [10, 11]. Merschjohann has analyzed the cytotoxic potential of 34 alkaloids of various structural types against *T. brucei* and *T. congolense* [12]. Many alkaloids, of which more than 21,000 structures have been found in plants, show a high degree of toxicity towards animals. Though many alkaloids are neurotoxins that interfere with neuroreceptors, ion channels or other parts of the neuronal signal chain, a substantial number of alkaloids are cytotoxic.

Cytotoxicity is the result of the molecular interactions of an alkaloid with one or several important targets present in a cell. The main targets include DNA, RNA, and the associated enzymes and related processes (*i.e.,* replication, repair, transcription, DNA polymerase, RNA polymerase, reverse transcriptase, repair enzymes, topoisomerase, telomerase), protein biosynthesis, protein conformation, biomembrane integrity, and membrane proteins [13, 14, 15, 16, 9]. Cytotoxicity is one of the chemotherapeutic targets of antitumor activity [17]. Most of the clinically used antitumor agents possess significant cytotoxic activity in cell culture systems [18.] Although the mechanism of tumor-inhibiting the activity of alkaloids is not clear, they are reported to decrease the high glycolytic activity of Ehrlich's ascites cells by inhibition of (Na-K)- ATPase in the plasma membrane [19]. This might be responsible for the inhibition of tumor growth.

The plants of the Solanaceae family are known to possess glycoalkaloids. The alkaloidal fraction of the methanolic extract of the plant, *Solanum jasminoides* (SJM-A) was, therefore, tested for its anticancer activity. The reliable criteria for judging the value of the anticancer potential of a sample is the prolongation of the life span of animals. In the present study, treatment with of the alkaloidal fraction SJM-A increases the life span of animals significantly with significant prevention of body weight changes when compared to tumor-bearing mice indicating its potent anticancer property.

Ascitic tumor implantation is known to induce per se local inflammatory reactions with an increase in vascular permeability, resulting in an intense edema formation, cellular migration, and a progressive ascetic fluid formation [20]. The ascitic fluid is essential for tumor growth since it constitutes the direct nutritional source for tumor cells [21]. The prevention of the body weight changes in the treated animals may be due to the reduction in ascitic fluid volume, which in turn may have restricted the tumor progression.

CONCLUSION: The results indicate the strong anticancer property of the alkaloidal fraction of the plant, *Solanum jasminoides*. Alkaloids are known to be widely distributed in the plant kingdom, and many of them have shown good anticancer activity [22]. It is not surprising; therefore, the alkaloidal fraction of the plant, *Solanum jasminoides* shows anticancer potential. Further studies are in progress on the isolation of the alkaloids, their structure elucidation, and biological evaluation.

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COMPETING INTEREST: The authors declare that they have no competing interest.

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