International Journal for Multidisciplinary Research (IJFMR)



E-ISSN: 2582-2160 • Website: <u>www.ijfmr.com</u> •

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Anticancer Activity of Alangium Salvifolium on Human Breast Cancer Cell Lines MCF-7, MDA-MB-468

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Abstract and Figures

Alangium salvifolium is a plant that grows in the alangium family. The study's plant species were chosen. The present study was implemented for revealing the anticancer effect of the leaves of methanolic extract of Alangium salvifolium. Phytochemical Screening of A.Salvifolium leaf extract was done which showed the presence of Alkaloids, Flavonoids, Terpenoids.

Human Breast Cancer cell lines MCF-7, MDA-MB-468 cells were grown in RPMI media and the 5-10methylenetetrahydrofolate reductase (MTHFR) Enzyme inhibitory activity was investigated.

MTHFR is a one-carbon metabolism enzyme that redirects the pool of folate from DNA synthesis and repair to methylation. The enzyme is vital for cellular homeostasis due to its key functions in the one carbon cycle, which include methionine and protein, DNA and RNA synthesis.

Cancer cell lines MCF-7 and MDA-MB-468 treated with Alangium Salvifolium compounds AS1-Deoxytubulosine and AS2 - carboline Hermaline.

EFFECT OF A. SALVIIFOLIUM COMPOUND AS1 AND AS2 ON INHIBITION OF MTHFR ACTIVITY OF HUMAN BREAST CANCER CELL MCF-7 AND MDA-MB-468

The MTHFR Enzyme inhibitory activity in MCF-7 and MDA-MB-468 cells treated with Alangium Salvifolium showed that at 320M concentration, the Enzyme activity was completely inhibited. In treated cells, about 80% inhibition activity was determined (Table 1.1, Graph 1.1). The current study was in line with a previous study on a plant alkaloid called -carboline benzoquinolidine, which inhibited DTHFR enzyme activity.

salvitonum compounds										
ANOVA				-						
		Sum o Squares	ofdf	Mean Square	F	Sig.				
	Between Groups	12.373	3	4.124	10530.3 55	.000				
AS1MCF7	Within Groups	3.133E-03	8	3.917E-04						

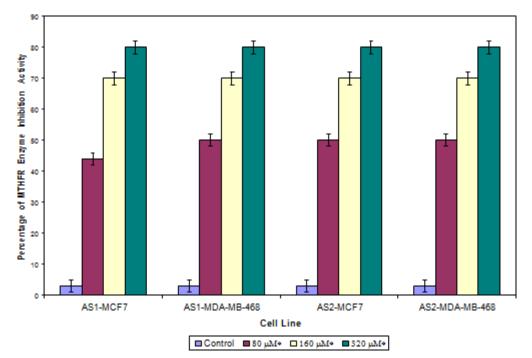
Table 1.1: Analysis of variance for methylene tetra hydro folate reductase inhibition activity of A. salvifolium compounds



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	Total	12.376	11			
AS1MDA MB	Between	7.302	3	2.434	1085.76	.000
	Groups				5	
	Within	1.793E-02	8	2.242E-03		
	Groups					
	Total	7.320	11			
AS2MCF7	Between	4.205	3	1.402	797.224	.000
	Groups					
	Within	1.407E-02	8	1.758E-03		
	Groups					
	Total	4.219	11			
AS2MDA MB	Between	2.625	3	.875	946.042	.000
	Groups					
	Within	7.400E-03	8	9.250E-04		
	Groups					
	Total	2.633	11			



Graph 1.1: MTHFR enzyme inhibitory activity of *A. salvifolium* compounds treated MCF-7 and MDA-MB-468 cells

The single (*) indicates a very significant difference from the Control (P 0.05), one way ANOVA Dunnett C Test. MTHFR Enzyme Inhibitory Activity of A.Salvifolium compound treated cells compared to that of control. The results are the averages plus standard deviations of three independent experiments that were carried out in triplicate



The goal is to investigate anticancer properties, identify and characterize selected chemical compounds, and test and evaluate the anticancer compounds' effects on cancer cells using cell proliferation inhibition assays and the possibility of developing a novel anticancer drug.

biochemical analysis of *A. salviifolium* compounds AS1 and AS2 MCF7 and MDA- MB-468 has inhibition activity on methylene tetra hydro folate reductatase enzymes. As a result, *A. salviifolium* compounds AS1- Deoxytubulosine and AS2 - carboline Hermaline have the potential to control Breast Cancer Cells.

Keywords: MTHFR Enzyme, MCF-7, MDA-MB-468 cells, RPMI media, alkaloids

REFERENCE:

- 1. HUFF J. 1992. Chemical toxicity and chemical carcinogenesis. Is there a causal connection? A comparative morphological evaluation of 1500 experiments. IARC Sci Pub 116: 437–475.
- 2. WEISBURGER JH. 1999. Carcinogenicity and mutagenicity testing, then and now. Mutat Res 437: 105–112.
- 3. GOMES-CARNEIRO MR, RIBEIRO-PINTO LF AND PAUMGARTTEN FJ. 1997. Environmental risk factors for gastric cancer: the toxicologist's standpoint. Cad SaúdePública 13 (Suppl): 27–38.
- 4. HUFF J. 1999. Chemicals associated with tumours of the kidney, urinary bladder and thyroid gland in laboratory rodents from 2000 US National Toxicology Program / National Cancer Institute bioassays for carcinogenicity. IARC Sci Pub 147: 211–225.
- 5. BUTTERWORTH BE AND BOGDANFFY MS. 1999. A comprehensive approach for integration of toxicity and cancer risk assessments. RegulToxicolPharmacol 29: 23–36.
- 6. GUTIÉRREZ JB AND SALSAMENDI AL. 2001. Fundamientos de ciênciatoxicológica. Diaz de Santos, Madrid, p. 155–177.
- Khatun, M.; Habib, M.R.; Rabbi, M.A.; Amin, R.; Islam, M.F.; Nurujjaman, M.; Karim, M.R.; Rahman, M.H. Antioxidant, cytotoxic and antineoplastic effects of Carissa carandas Linn.leaves. Exp. Toxicol. Pathol., 2017, 69(7), 469-476.
- 8. Reya, T.; Morrison, S.J.; Clarke, M.F., Weissman, I.L. Stem cells, cancer, and cancer stem cells. Nature, 2001, 414(6859), 105-11.
- 9. Dean, M.; Fojo, T.; Bates, S. Tumour stem cells and drug resistance. Nat Rev Cancer, 2005, 5(4), 275-84.
- Colak, S.; Medema, J.P. Cancer stem cells--important players in tumor therapy resistance. FEBS. J., 2014, 281(21), 4779-91.
- J. A. Wessels, S. M. van der Kooij, S. le Cessie, W. Kievit, P. Barerra, C. F. Allaart, T. W. Huizinga, H. J. Guchelaar, A clinical pharmacogenetic model to predict the efficacy of methotrexate monotherapy in recent-onset rheumatoid arthritis: Arthritis Rheum. 2007, 56, 1765.
- M. N. Islam, M. Hossain, S. A. Haq, M. N. Alam, P. M. Ten Klooster, J. J. Rasker, Efficacy and safety of methotrexate in articular and cutaneous manifestations of systemic lupus erythematosus: Int. J. Rheum. Dis. 2012, 15, 62.
- 13. 13.B. C. Kieseier, D. R. Jeffery, Chemotherapeutics in the treatment of multiple sclerosis: Ther. Adv. Neurol. Disord. 2010, 3, 277.



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- W. B. Pratt, R. W. Ruddon, W. D. Ensminger, J. Maybaum, Anticancancer Drugs, Oxford University Press, New York, Oxford, 1994 15.Workman P and Al-Lazikani B. Drugging cancer genomes. Nat Rev Drug Discov 2013; 12: 889–90.
- 15. Jiang H, Wu J and Zhang L et al. Chemical biology in China takes on signal transduction. Nat Chem Biol 2008; 4: 515–8.
- Jiang H, Hsieh-Wilson L and Arruda P et al. Voices of chemical biology. Nat Chem Biol 2015; 11: 446–7.
- 17. Schwartz GK, Shah MA. Targeting the cell cycle: a new approach to cancer therapy. J Clin Oncol 2005; 23: 9408–9421.
- 18. Williams GH, Stoeber K. The cell cycle and cancer. J Pathol 2012; 226: 352–364.
- 19. Manchado E, Guillamot M, Malumbres M. Killing cells by targeting mitosis. Cell Death Differ 2012; 19: 369–377.
- Komlodi-Pasztor E, Sackett DL, Fojo AT. Inhibitors targeting mitosis: tales of how great drugs against a promising target were brought down by a flawed rationale. Clin Cancer Res 2012; 18: 51–63.