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Unveiling the Anticancer Potential of Curcuma Caesia Roxb. Against Ovarian Cancer: An Indigenous Plant of Assam

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ABSTRACT

Background: *Curcuma caesia* Roxb., (Black turmeric), a distinct variety of medicinal herb belonging to Zingiberaceae family is an underexplored plant of Assam, India whose essential oil composition and historic uses set it apart from other members of its family. This study aims to explore the anticancer potential of the phytoconstituents of this herb against ovarian cancer cell line.

Method: In-silico Molecular docking was carried out using PyRx for assessing the effectiveness of active constituents (Isoborneol, Alloaromadendrene, (+)-2-Bornanone, α- Santalol, Ar-tumerone, Megastigma-3,7(E),9-triene, 5,8,11,14,17-Eicosapentaenoic acid methyl ester, Retinal,9-cis, Androstenediol, Camphor, Benzene-1-(1,5-dimethyl-4-hexenyl)-4-methyl, 1,8-Cineole, β-elemene, Bornyl acetate & α-terpineol) against ovarian cancer protein (PDB ID:2NS2) and the potential 2D interaction of the active constituents with binding site of receptor was visualized using Biovia Discovery Studio software. For anticancer study, the rhizomes of *Curcuma caesia* Roxb., were extracted successively using Soxhlet extraction by hexane, ethyl acetate and methanol extracts respectively and the methanol extract (MECC) containing Androstenediol fraction was assessed for cytotoxicity against PA-1 ovarian cancer cell line by employing the MTT assay.

Result: The outcome of the in-silico analysis demonstrated the highest binding affinity of Androstenediol with a binding score of -7.1 kcal/mol and the 2D interaction revealed that the favorable interacting residues are GLN A:210, ALA A;132, PRO A:135 and VAL A:211 respectively. In cytotoxicity analysis, it was found that there was a dose-dependent reduction in cell viability with an IC_{50} value of 610 µg/ml. Apoptosis analysis through flow cytometry further revealed that MECC induced significant levels of early apoptosis (23.09%) and late apoptosis (3.50%) with negligible necrosis indicating that the extract primarily triggers programmed cell death.

Conclusion: This study suggests that the presence of bioactive compounds in Curcuma caesia Roxb. holds promising anticancer properties which provides a foundation for the development of novel plant-based anticancer therapies.

Keywords: *Molecular Docking, 2D interaction, Apoptosis, Cytometry, Zingiberaceae*

1. INTRODUCTION

Ovarian cancer menaces a global treat worldwide affecting thousands of women every year which possesses significant ramifications for both the medical profession and public health. Over 239,000 women worldwide are diagnosed with ovarian cancer each year and around 152,000 women population succumb to the illness. According to estimation, the annual incidence of ovarian cancer in the United States is 11.7 per 100,000 women with a 7.4 per 100,000 women fatality rate [1]. There are numerous subtypes and genetic anomalies of ovarian cancer which makes it a complicated and diverse illness. The most frequent and dangerous type of ovarian cancer is epithelial cancer which makes up around 90% of all cases. Most patients are detected at an advanced stage due to carelessness and a lack of early screening and diagnostic measures which contributes to dismal 5-year survival rate of around 10%. Ovarian cancer has a complex etiology that includes both environmental and genetic components [2,3]. Genetic anomalies have been closely linked to a higher risk of ovarian cancer including mutations in the BRCA1 and BRCA2 genes. Hormonal factors have also been linked to the development of ovarian cancer including the timing of reproductive events and the use of fertility medications. India sees an estimated 36,000 new ovarian cancer cases annually with a high mortality rate due to late diagnosis [4].

The dysregulation of signaling pathway promotes tumor progression and metastasis eventually leading to resistance in therapy. The various signaling pathways associated with ovarian cancer includes PI3K/AKT/mTOR pathway, RAS/RAF/MEK/ERK (MAPK) pathway, Notch signaling, Hedgehog (Hh) pathway, WNT/β-Catenin pathway, TP53 pathway, NF-κB pathway, hormonal signaling, angiogenesis (VEGF/VEGFR), DNA Repair pathways, Immune checkpoints, TGF-β pathway, JAK/STAT pathway and Apoptosis pathways. Components which promote the progression of ovarian cancer includes PI3K, AKT, mTOR, RAS, RAF, MEK, ERK, Notch receptors (NOTCH1-4), ligands (Jagged, Delta-like), SHH, PTCH1, SMO, GLI, WNT ligands, Frizzled, β-catenin, Oestrogen (ER), Progesterone (PR), and Androgen receptors (AR), BRCA1/2, homologous recombination (HR) genes, PD-1/PD-L1, CTLA-4 and BCL-2 family [5-9]. The dysregulation of this pathway leads to various abnormalities including mutations in PIK3CA (activation) or PTEN (loss), KRAS/BRAF mutations in low-grade serous carcinoma (LGSC), mutations in CTNNB1 (β-catenin) in endometrioid, chemoresistance, loss of cell cycle control/apoptosis; genomic instability, HR deficiency in HGSOC, STAT3 activation linked to proliferation/immune evasion and anti-apoptotic protein overexpression (BCL-2) [10,11]. Currently several marketed drugs are available for treatment of ovarian cancer which includes Alpelisib (PI3K), MK-2206 (AKT), Everolimus (mTOR), Trametinib (MEK inhibitor) for LGSC, Demcizumab (anti-Notch), gamma-secretase inhibitors, Vismodegib (SMO inhibitor), Porcupine inhibitors (LGK974), Proteasome inhibitors (bortezomib) or IKK inhibitors, Tamoxifen (ER antagonist), AR inhibitors (Enzalutamide), Bevacizumab (anti-VEGF), PARP inhibitors (Olaparib, Niraparib), Pembrolizumab (PD-1 inhibitor), Galunisertib (TGF-βR inhibitor), Ruxolitinib (JAK inhibitor), Venetoclax (BCL-2 inhibitor) etc [12-16]. Even though these drugs treat ovarian cancer but many patients fail to respond initially due to tumor heterogeneity or pre-existing mutations (e.g., BRCA wild-type tumors resistant to PARP inhibitors). Even initially effective drugs (e.g., PARP inhibitors, platinum chemotherapy) lose efficacy over time due to restoration of homologous recombination repair (e.g., BRCA reversion mutations), activation of bypass signaling pathways (e.g., PI3K/AKT compensating for MEK inhibition) and upregulation of drug efflux pumps (e.g., ABC transporters expelling chemotherapies) [17, 18]. PARP inhibitors (e.g., Olaparib, Niraparib), hematologic toxicity (anaemia, thrombocytopenia), fatigue, nausea, are rare but serious risks of myelodysplastic syndrome (MDS). Anti-angiogenics (e.g., bevacizumab) causes hypertension, proteinuria, and gastrointestinal perforation whereas targeted therapies (e.g., PI3K/AKT/mTOR inhibitors) cause hyperglycaemia, rash, and metabolic disturbances and immunotherapies (e.g., PD-1 inhibitors) causes immune-related adverse events (e.g., colitis, pneumonitis). Moreover, there is a limited efficacy in subtypes for example: PARP inhibitors are only effective in homologous recombination-deficient (HRD) tumours (~50% of HGSOC), MEK inhibitors (e.g., trametinib) benefit low-grade serous carcinoma (LGSC) but not high-grade subtypes, hormonal therapies (e.g., tamoxifen) is limited to ER/PR-positive tumours (minority of cases) and immunotherapies causes low response rates (~10–15%) due to ovarian cancer's immunosuppressive microenvironment [19-22]. Many drugs (e.g., anti-Notch, Hedgehog inhibitors) lack validated biomarkers to identify responsive patients, leading to trial failures. Drugs like PEGylated liposomal doxorubicin (PLD) or gemcitabine have modest survival benefits (3-6 months). Due to stromal interactions cancer-associated fibroblasts (CAFs) and immunosuppressive cells (TAMs) shield tumors from drugs**.** The expensive targeted therapies such as PARP inhibitors and biologics (e.g., bevacizumab) cost >$10,000/month, limits its access in low-resource settings and the biomarker HRD or BRCA testing is not universally available which further restricts its personalized therapy [23-25]. Thus, in order to overcome the aforesaid limitations, an attempt has been made in this study to promote the use of naturally available *Curcuma caesia* Roxb., plant as an alternative therapy for ovarian cancer [26]. This study explores the anticancer potential of *Curcuma caesia* Roxb. Extracts specifically focusing on their cytotoxic effects and apoptosis-inducing capabilities in human ovarian cancer cell lines (PA-1). With the identification of the bioactive components responsible for these effects, this research shall contribute to the growing body of evidence supporting the use of natural compounds in cancer therapy. This is the first ever investigation reported for evaluation of anticancer property of *Curcuma caesia* Roxb. which paves a way for future studies.

2. MATERIALS AND METHODS

2.1 Molecular Docking and 2D Interaction

Chemsketch software was used to prepare the structure of the herb's active constituents which include Isoborneol, Alloaromadendrene, (+)-2-Bornanone, α-Santalol, Ar-tumerone, Megastigma-3,7(E),9-triene, 5,8,11,14,17-Eicosapentaenoic acid methyl ester, Benzene-1-(1,5-dimethyl-4-hexenyl)-4-methyl, Retinal, 9-cis, Androstenediol, Camphor, 1,8-Cineole, β-elemene, Bornyl acetate and α-terpineol. The energy of the compounds was minimized using Chem 3D software in order to obtain the most stable lowest energy conformation. The RCSB Protein Data Bank provided the necessary ovarian cancer protein with PDB ID:2NS2. To find the desired binding affinity with receptor, Molecular Docking research was conducted using PyRx software and Biovia Discovery Studio software was used to visualise the possible two-dimensional (2D) interaction of the active components with the receptor's binding site.

2.2 Plant Collection and Authentication

The rhizomes of *Curcuma caesia* Roxb. were collected from the Sivasagar district in Assam, India and authenticated by the Botanical Survey of Shillong, India (Reference number: USTM/GR/02/2023) with a BSI reference number of BSI/ERC/Tech/2023-24/1546. This authentication process was critical for ensuring the accurate identification of the plant species.

2.3 Extraction Process

Freshly collected rhizomes were washed, dried and pulverized. Successive Soxhlet extraction was carried out using three solvents: hexane, ethyl acetate and methanol to extract non-polar, mildly polar and polar compounds respectively. The hexane extraction lasted for 48 hours while ethyl acetate and methanol extractions were performed for 24 hours each. The methanolic extract containing Androstenediol fraction was separated out and the extracts were concentrated through lyophilization and stored at 4°C for further analysis.

2.4 Cell Culture

Human ovarian cancer cell lines (PA-1) were obtained from the National Centre for Cell Science (NCCS), Pune, India. The cells were cultured in Minimum Essential Medium (MEM) supplemented with 10% fetal bovine serum (FBS), 1% sodium bicarbonate, sodium pyruvate and non-essential amino acids. The culture was maintained at 37°C in a humidified atmosphere with 5% CO_2, following established protocols for cancer cell line maintenance.

2.5 Cytotoxicity Analysis (MTT Assay)

The cytotoxic effect of the methanol extract (MECC) was assessed using the MTT assay which is a colorimetric assay that measures cell viability based on the reduction of tetrazolium salt to formazan by metabolically active cells. PA-1 cells were seeded in 96-well plates (2×10^2 cells per well) and treated with varying concentrations of MECC (100-1000 µg/ml). After 24 hours, MTT solution was added to the wells and the cells were incubated for an additional 4 hours [16].

2.6 Apoptosis Analysis

Apoptosis, or programmed cell death is a critical mechanism by which cancer therapies exert their effects. In this study, apoptosis was assessed using the Annexin V-FITC Apoptosis Detection Kit followed by flow cytometry. PA-1 cells were treated with the IC_{50} concentration of MECC for 24 hours [17].

3. RESULT

3.1 Molecular Docking and 2D Interaction Analysis

The molecular docking of active constituents Isoborneol, Alloaromadendrene, (+)-2-Bornanone, α-Santalol, Ar-tumerone, Benzene-1-(1,5-dimethyl-4-hexenyl)-4-methyl, Megastigma-3,7(E),9-triene, 5,8,11,14,17-Eicosapentaenoic acid methyl ester, Retinal,9-cis, Androstenediol, Camphor, 1,8-Cineole, β-elemene, Bornyl acetate & α-terpineol was analysed using PyRx by taking the protein with PDB ID:2NS2 which is tabulated in Table 1. It was observed that out of all the constituents, Androstenediol had displayed the highest binding affinity with a binding score of -7.1 kcal/mol. The 2D interaction of the most potent constituent Androstenediol with the active site of protein with PDB ID: 2NS2 along with the favorable interacting residues; GLN A:210, ALA A;132, PRO A:135 and VAL A:211 respectively is given in Figure 1.

Table 1: Binding Affinity Score of Various Active Constituents for Ovarian Cancer

Active Constituents	Docking score (Kcal/mol)	PDB ID
Isoborneol	-5.4	2NS2
Alloaromadendrene	-6.7	2NS2

(+)-2-Bornanone	-5.5	2NS2
Benzene-1-(1,5-dimethyl-4-hexenyl)-4-methyl	-6.1	2NS2
α-Santalol	-6.3	2NS2
Ar-tumerone	-6.3	2NS2
Megastigma-3,7(E),9-triene	-6.3	2NS2
Retinal,9-cis	-6.8	2NS2
Androstenediol	**-7.1**	2NS2
Camphor	-5.5	2NS2
1,8-Cineole	-5.8	2NS2
β-elemene	-6.5	2NS2
Bornyl acetate	-5.7	2NS2
α-terpineol	-6.1	2NS2
5,8,11,14,17-Eicosapentaenoic acid methyl ester	-5.5	2NS2

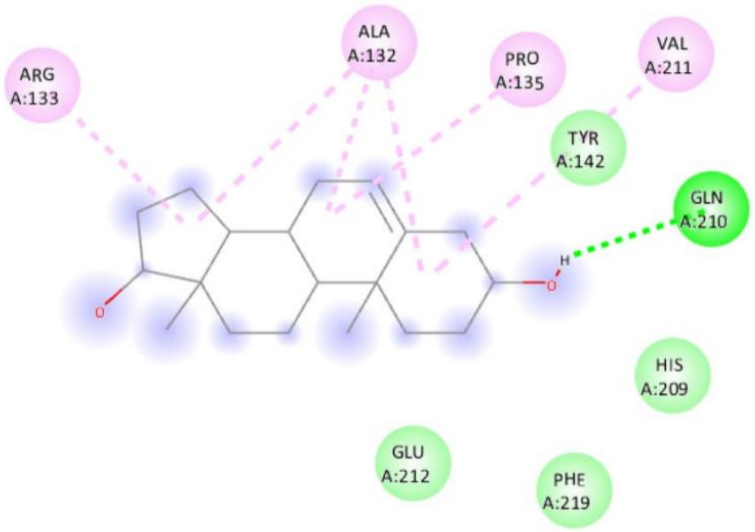

Figure 1: 2D Interaction of Androstenediol with Target Receptor Having PDB ID: 2NS2

3.2 Cytotoxicity Analysis (MTT Assay)

For cytotoxicity analysis (MTT Assay) the absorbance was measured at 570 nm using a microplate reader to determine cell viability. The IC_{50} value was found to be 610 µg/ml and is shown in Table 2 with graphical representation in Figure 2.

Table 2: % Cell Viability of *C. caesia* Methanolic Extract (MECC) Containing Androstenediol Fraction

Concentration (µg/ml)	**Average**	**% Cell Viability**	**IC_{50} value**
Control	0.5713	100	
100	0.5262	92	
200	0.4959	86	

300	0.4269	74	610 µg/ml
400	0.3557	62	
500	0.3149	55	
600	0.2917	51	
700	0.2436	42	
800	0.2025	35	
900	0.1878	32	
1000	0.1744	30	

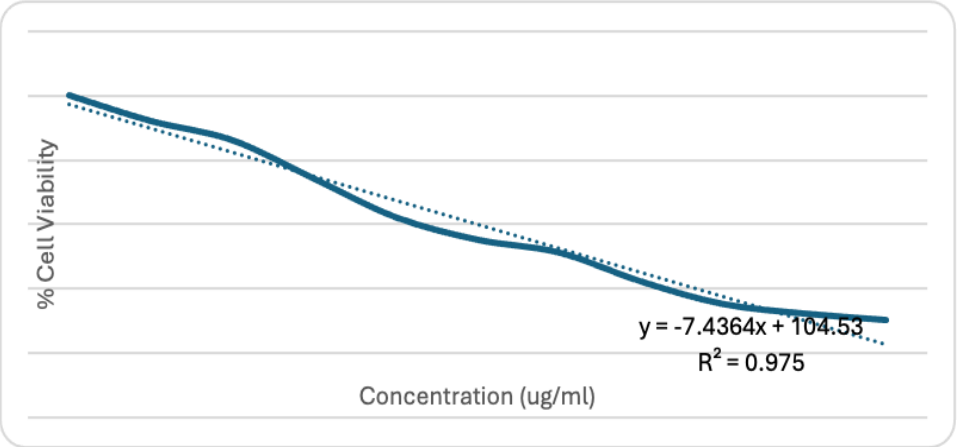

Figure 2: % Cell Viability of the Androstenediol Fraction at Various Concentrations Ranging From 100-1000 µg/Ml

3.3 Apoptosis Analysis

Flow cytometry analysis revealed that MECC containing Androstenediol fraction induced significant early apoptosis (23.09%) and late apoptosis (3.50%) with minimal necrosis (0.01%), indicating that MECC containing Androstenediol fraction promotes apoptosis rather than necrosis in ovarian cancer cells (Figure 3).

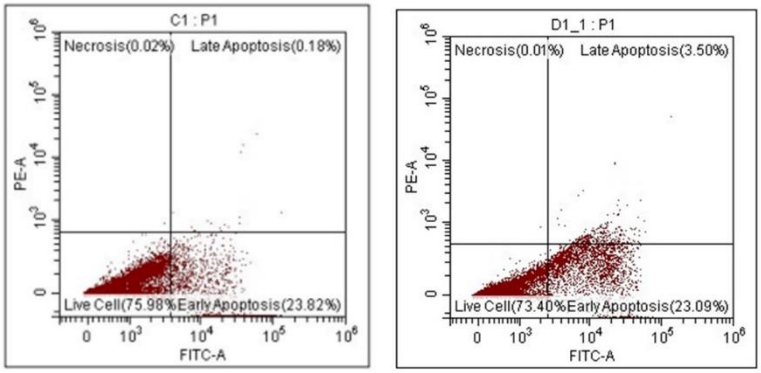

Figure 3: Flow Cytometry Analysis of Apoptosis in Ovarian Cancer Cell Line Induced by *Curcuma caesia* Roxb in MECC Containing Androstenediol Fraction

4. DISCUSSION

Here with we report the anticancer potential of *Curcuma caesia* Roxb. against ovarian cancer cells. A Molecular docking study was carried out which revealed that Androstenediol had exhibited the highest binding score with the target receptor of ovarian cancer among all the active constituents present in the methanolic extract. The 2D interaction demonstrated that the

favorable interacting residues are GLN A:210, ALA A;132, PRO A:135 and VAL A:211. The experimental findings indicate that MECC containing Androstenediol fraction exhibits significant cytotoxicity and induces apoptosis in a dose-dependent manner with an IC_{50} value of 610 µg/ml. Apoptosis is a key mechanism by which MECC exerts its anticancer effects as demonstrated by the substantial induction of both early and late apoptotic markers. The results of this study align with previous research on curcuminoids, which have been shown to inhibit cancer cell proliferation and induce apoptosis in various cancer types, including ovarian cancer. These findings suggest that the bioactive compounds in *Curcuma caesia*, particularly the methanolic extract containing Androstenediol holds promising therapeutic efficacy for treatment of ovarian cancer. Future studies will be needed in the direction of focusing on isolating and characterizing the specific compounds responsible for this observed cytotoxic effects. This research lays the groundwork for the development of plant-based cancer therapies that offer fewer side effects than conventional treatments.

5. CONCLUSION

This preliminary study is in the direction of evaluating the anticancer potential of *Curcuma caesia* Roxb. against ovarian cancer cells which demonstrate that MECC containing Androstenediol fraction induces dose-dependent cytotoxicity and apoptosis with an IC_{50} value of 610 µg/ml. These findings highlight the therapeutic promise of *Curcuma caesia* Roxb. as a source of bioactive compound for ovarian cancer treatment. This foundational research opens avenues for the development of plant-based anticancer therapies with potentially fewer side effects than conventional treatments.

REFERENCES

[1] Marigoudar JB, Sarkar D, Yuguda YM, Abutayeh RF, Kaur A, Pati A, Mitra D, Ghosh A, Banerjee D, Borah S, Barman K. Role of vitamin D in targeting cancer and cancer stem cell populations and its therapeutic implications. Medical Oncology. 2022 Oct 29;40(1):2.

[2] Siegel RL, Miller KD, Jemal A. Cancer statistics, 2018. CA: a cancer journal for clinicians. 2018 Jan;68(1):7-30.

[3] Cragg GM, Newman DJ. Plants as a source of anti-cancer agents. Journal of ethnopharmacology. 2005 Aug 22;100(1-2):72-9.

[4] Perrone C, Angioli R, Luvero D, Giannini A, Di Donato V, Cuccu I, Muzii L, Raspagliesi F, Bogani G. Targeting BRAF pathway in low-grade serous ovarian cancer. Journal of Gynecologic Oncology. 2024 May 14;35(4):e104.

[5] Karikas GA. Anticancer and chemopreventing natural products: some biochemical and therapeutic aspects. J buon. 2010 Dec;15(4):627-38.

[6] Jiang Y, Lim J, Wu KC, Xu W, Suen JY, Fairlie DP. PAR2 induces ovarian cancer cell motility by merging three signalling pathways to transactivate EGFR. British Journal of Pharmacology. 2021 Feb;178(4):913-32.

[7] Kumari A, Shonibare Z, Monavarian M, Arend RC, Lee NY, Inman GJ, Mythreye K. TGFβ signaling networks in ovarian cancer progression and plasticity. Clinical & Experimental Metastasis. 2021 Apr;38:139-61.

[8] Park SA, Kim LK, Park HM, Kim HJ, Heo TH. Inhibition of GP130/STAT3 and EMT by combined bazedoxifene and paclitaxel treatment in ovarian cancer. Oncology Reports. 2022 Mar;47(3):52.

[9] Ji Z, Tian W, Gao W, Zang R, Wang H, Yang G. Cancer-associated fibroblast-derived interleukin-8 promotes ovarian cancer cell stemness and malignancy through the Notch3-mediated signaling. Frontiers in cell and developmental biology. 2021 Jul 1;9:684505.

[10] Rinne N, Christie EL, Ardasheva A, Kwok CH, Demchenko N, Low C, Tralau-Stewart C, Fotopoulou C, Cunnea P. Targeting the PI3K/AKT/mTOR pathway in epithelial ovarian cancer, therapeutic treatment options for platinum-resistant ovarian cancer. Cancer Drug Resistance. 2021;4(3):573.

[11] Meng J, Peng J, Feng J, Maurer J, Li X, Li Y, Yao S, Chu R, Pan X, Li J, Zhang T. Niraparib exhibits a synergistic anti-tumor effect with PD-L1 blockade by inducing an immune response in ovarian cancer. Journal of Translational Medicine. 2021 Dec;19:1-6.

[12] Chugh NA, Koul A. Altered presence of extra cellular matrix components in murine skin cancer: Modulation by Azadirachta indica leaf extract. Journal of Traditional and Complementary Medicine. 2021 May 1;11(3):197-208.

[13] Bray F, Ferlay J, Soerjomataram I, Siegel RL, Torre LA, Jemal A. Global cancer statistics 2018: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA: a cancer journal for clinicians. 2018 Nov;68(6):394-424.

[14] Salehi B, Del Prado-Audelo ML, Cortés H, Leyva-Gómez G, Stojanović-Radić Z, Singh YD, Patra JK, Das G, Martins N, Martorell M, Sharifi-Rad M. Therapeutic applications of curcumin nanomedicine formulations in

cardiovascular diseases. Journal of clinical medicine. 2020 Mar 10;9(3):746.

[15] Deshpande AM, Sastry KV, Bhise SB. A contemporary exploration of traditional Indian snake envenomation therapies. Tropical Medicine and Infectious Disease. 2022 Jun 16;7(6):108.

[16] Kuttan R, Bhanumathy P, Nirmala K, George MC. Potential anticancer activity of turmeric (Curcuma longa). Cancer letters. 1985 Nov 1;29(2):197-202.

[17] Simoes e Silva AC, Miranda AS, Rocha NP, Teixeira AL. Neuropsychiatric disorders in chronic kidney disease. Frontiers in pharmacology. 2019 Aug 16;10:932.

[18] Vyas A, Dandawate P, Padhye S, Ahmad A, Sarkar F. Perspectives on new synthetic curcumin analogs and their potential anticancer properties. Current pharmaceutical design. 2013 Apr 1;19(11):2047-69.

[19] Akter S, Rahman MA, Hasan MN, Akhter H, Noor P, Islam R, Shin Y, Rahman MH, Gazi MS, Huda MN, Nam NM. Recent advances in ovarian cancer: therapeutic strategies, potential biomarkers, and technological improvements. Cells. 2022 Feb 13;11(4):650.

[20] Newman DJ, Cragg GM. Natural products as sources of new drugs over the last 25 years. Journal of natural products. 2007 Mar 23;70(3):461-77.

[21] Sharma H, Gupta N, Garg N, Dhankhar S, Chauhan S, Beniwal S, Saini D. Herbal Medicinal Nanoformulations for Psoriasis Treatment: Current State of Knowledge and Future Directions. The Natural Products Journal. 2024 Oct 1;14(7):61-79.

[22] Caunii A, Pribac G, Grozea I, Gaitin D, Samfira I. Design of optimal solvent for extraction of bio–active ingredients from six varieties of Medicago sativa. Chemistry Central Journal. 2012 Dec;6:1-8.

[23] Aratanechemuge Y, Komiya T, Moteki H, Katsuzaki H, Imai K, Hibasami H. Selective induction of apoptosis by ar-turmerone isolated from turmeric (Curcuma longa L) in two human leukemia cell lines, but not in human stomach cancer cell line. International journal of molecular medicine. 2002 May 1;9(5):481-4.

[24] Romijn JC, Verkoelen CF, Schroeder FH. Application of the MTT assay to human prostate cancer cell lines in vitro: establishment of test conditions and assessment of hormone-stimulated growth and drug-induced cytostatic and cytotoxic effects. The prostate. 1988; 12(1):99-110.

[25] Richardson DL, Eskander RN, O'Malley DM. Advances in ovarian cancer care and unmet treatment needs for patients with platinum resistance: a narrative review. JAMA oncology. 2023 Jun 1.

[26] Lv ZD, Liu XP, Zhao WJ, Dong Q, Li FN, Wang HB, Kong B. Curcumin induces apoptosis in breast cancer cells and inhibits tumor growth in vitro and in vivo. International journal of clinical and experimental pathology. 2014;7(6):2818.