Open Access Review Article

January - February 2026, Vol. 02 (1), 15-21

INTERNATIONAL JOURNAL OF PHARMACEUTICAL RESEARCH AND MEDICINAL PLANTS

(An International Peer Reviewed Journal)

# Repurposing Phenytoin and Its Derivatives for Ovarian Cancer Management: Current Insights and Future Directions

**Harshada Subhash Bhosale**

Savitribai Phule Pune University, Maharashtra, India

**ARTICLE INFO:**

**Received:** 16th Jan. 2026; **Received** in revised form: 28th Jan. 2026; **Accepted:** 18th Feb. 2026; **Available online:** 27th February 2026.

**Keywords:** Phenytoin, Hydantoin derivatives, Ovarian cancer, Drug repurposing, Sodium channels, Apoptosis, Nano-drug delivery, Chemoresistance.

**DOI:** https://doi.org/10.61280/journalmedicinalplants.v2i1.243

***Corresponding Author:** Harshada Subhash Bhosale

**ABSTRACT**

Ovarian cancer is one of the most serious gynecological malignancies because most patients are diagnosed at an advanced stage when treatment options become limited. Although surgery and chemotherapy remain the primary treatments, many patients eventually develop resistance to drugs such as cisplatin and paclitaxel, resulting in poor long-term outcomes. Drug repurposing has gained attention as an effective strategy because it allows the use of existing approved medicines for new therapeutic purposes, reducing both development time and cost. Phenytoin, traditionally used as an antiepileptic drug, has shown promising anticancer effects by blocking voltage-gated sodium channels, reducing tumor cell invasion, and inducing apoptosis in various cancer types. Hydantoin derivatives, structurally related to phenytoin, also demonstrate enhanced cytotoxicity and better molecular targeting. This review provides an overview of current insights into the potential role of phenytoin and its derivatives in managing ovarian cancer, highlighting their mechanisms of action, preclinical evidence, limitations, and future therapeutic opportunities.

## Introduction

Ovarian cancer is the eighth most common malignancy among women worldwide and remains a leading cause of gynecological cancer-related mortality due to late diagnosis and nonspecific early symptoms (1). More than 70% of patients are diagnosed at an advanced stage, where metastatic spread and disease recurrence are common, resulting in poor clinical outcomes (2). Although platinum-based chemotherapy initially produces favorable responses, the majority of patients eventually develop chemoresistance, which significantly limits long-term treatment success (3).

Drug repurposing has emerged as an effective strategy for identifying new therapeutic applications for existing approved drugs, thereby reducing development time, cost, and early-phase toxicity risks (4). This approach is particularly valuable in ovarian cancer, where rapid and effective therapeutic alternatives are urgently required (5). Phenytoin, a well-established antiepileptic drug, has attracted increasing attention due to its ability to block voltage-gated sodium channels, which are aberrantly expressed in several cancer types, including ovarian

cancer (6). Inhibition of these channels suppresses cancer cell motility, proliferation, and metastatic potential (7).

Hydantoin derivatives, including phenytoin, have demonstrated significant anticancer activity in multiple experimental models (8). Structural modification of the hydantoin scaffold has led to the development of compounds with enhanced cytotoxicity and the ability to target multiple oncogenic pathways simultaneously (9). Given the biological complexity of ovarian cancer, phenytoin-based compounds represent promising candidates for anticancer drug repurposing and development (10).

## Chemical Structure and Pharmacological Basis

Phenytoin belongs to the hydantoin class of heterocyclic compounds, characterized by a five-membered imidazolidine-2,4-dione ring containing two carbonyl groups that contribute to its chemical stability and biological activity (11). The presence of two phenyl groups at the C-5 position enhances lipophilicity, facilitating efficient membrane permeability and intracellular accumulation (12). These physicochemical properties enable phenytoin to interact with intracellular signaling pathways involved in cancer growth and metastasis (11). The hydantoin nucleus is highly amenable to structural modification, and substitutions at the N-1, N-3, or C-5 positions can significantly improve binding affinity, cytotoxicity, and selectivity toward cancer cells (13). Medicinal chemistry studies have shown that the incorporation of electron-withdrawing groups, halogens, or aromatic moieties enhances anticancer potency in hydantoin analogues (14). These features establish phenytoin as a versatile scaffold for the development of novel anticancer agents targeting ovarian cancer (10).

## Mechanism of Anticancer Action of Phenytoin

One of the primary anticancer mechanisms of phenytoin involves inhibition of voltage-gated sodium channels, particularly Nav1.5, which is frequently overexpressed in ovarian, breast, and colon cancers (6). While these channels regulate electrical signaling in excitable tissues, their aberrant activation in cancer cells promotes migration, invasion, and metastatic progression (15). By blocking sodium influx, phenytoin reduces membrane depolarization and suppresses cancer cell motility, thereby limiting metastatic dissemination (6).

In addition to sodium-channel blockade, phenytoin interferes with key intracellular signaling pathways associated with cancer cell survival and chemoresistance. Specifically, it inhibits the PI3K/AKT and MAPK pathways, which play central roles in ovarian cancer proliferation and resistance to chemotherapy (16). Suppression of these pathways induces growth arrest and sensitizes cancer cells to apoptosis (17). Furthermore, phenytoin increases reactive oxygen species production, disrupts mitochondrial integrity, and activates caspase-dependent apoptotic cascades, collectively contributing to cancer cell death (18).

Phenytoin also suppresses epithelial–mesenchymal transition (EMT), a process that enables cancer cells to acquire invasive and stem-like properties (19). Sodium-channel inhibition downregulates mesenchymal markers such as vimentin and N-cadherin while restoring epithelial markers including E-cadherin (20). As a result, cancer cell migration is reduced and metastatic spread is limited (21).

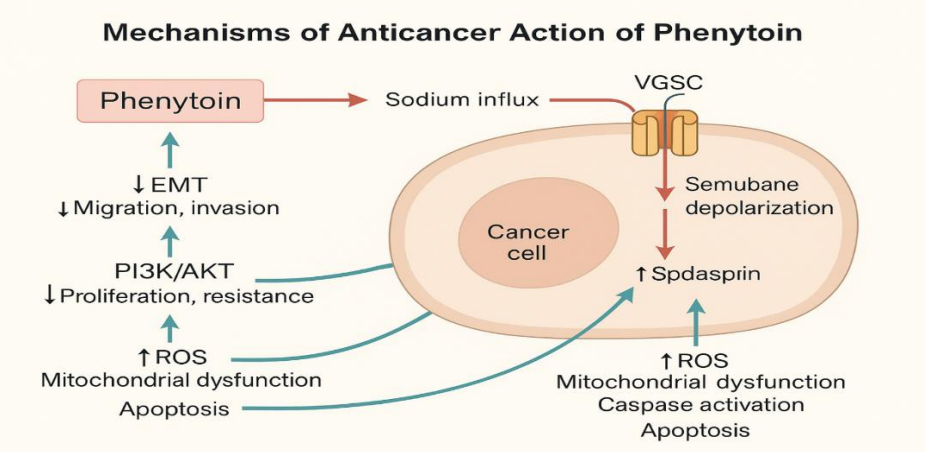


**Figure 1:** Mechanism of Anticancer Action of Phenytoin

### Evidence of Phenytoin Activity in Cancer Models

In vitro studies demonstrate that phenytoin significantly reduces cancer cell proliferation, migration, and invasion across multiple malignancies, including breast, colon, lung, prostate, and ovarian cancers (22). In ovarian cancer cell lines such as SKOV3 and OVCAR3, phenytoin induces dose-dependent reductions in cell viability, promotes apoptosis, and suppresses migratory behavior (23). These findings support the hypothesis that sodium-channel inhibition weakens ovarian cancer progression (24).

In mouse xenograft models, phenytoin-treated tumors exhibit reduced tumor volume, diminished angiogenesis, and fewer metastatic nodules compared with untreated controls (25). Importantly, phenytoin administration was not associated with significant systemic toxicity, highlighting its favorable safety profile (26). Epidemiological observations further suggest that epileptic patients receiving sodium channel-blocking antiepileptic drugs may show reduced cancer incidence, supporting further clinical investigation (27,28).

### Hydantoin Derivatives and Their Anticancer Potential

Hydantoin derivatives often display enhanced anticancer activity compared with phenytoin itself (29). Structural modifications such as halogenation, aromatic substitution, or heterocycle incorporation significantly improve cytotoxic potency and selectivity (30,31). Several hydantoin derivatives induce oxidative stress, mitochondrial dysfunction, and caspase-mediated apoptosis (32).

Other derivatives disrupt DNA synthesis and cell-cycle progression, leading to arrest at the G0/G1 or G2/M phases (33). Certain analogues inhibit tubulin polymerization, resulting in mitotic arrest and cell death (34). Metal-based hydantoin derivatives, particularly platinum–hydantoin complexes, demonstrate enhanced DNA binding and pro-apoptotic activity, offering promise against platinum-resistant ovarian cancer (35,36). Hybrid compounds such as β-carboline–hydantoin conjugates further suppress EMT and cancer cell invasion (37).

**Table 1:** Anticancer Activities of Hydantoin Derivatives

| Type of Hydantoin Derivative | Structural Modification | Primary Anticancer Mechanism | Effects on Cancer Cells |
|---|---|---|---|
| Basic hydantoin derivatives | Small substitutions on hydantoin ring | Increased oxidative stress; mitochondrial disruption | ↑ ROS, ↑ apoptosis, ↓ proliferation |
| Halogenated / aromatic hydantoins | Halogens, aromatic rings, heterocycles | Enhanced receptor binding and cytotoxicity | ↑ Selectivity, ↑ cell penetration, ↓ tumor growth |
| Cell-cycle inhibitory hydantoins | Modifications at N-1 / N-3 / C-5 | Inhibition of DNA synthesis; cell-cycle arrest | G0/G1 or G2/M arrest, ↓ mitosis |
| Tubulin-inhibiting hydantoins | Bulky aromatic ligands | Disruption of tubulin polymerization | Failed chromosome separation; mitotic arrest; apoptosis |
| Metal-based hydantoin complexes | Platinum (Pt) and other metal conjugation | DNA binding and enhanced DNA damage | Strong apoptosis in resistant tumors; useful in platinum-resistant ovarian cancer |

## Combination Therapy Potential with Chemotherapeutic Agents

Combination therapy remains a cornerstone of cancer treatment due to its ability to target multiple pathways simultaneously (38). Phenytoin may enhance the efficacy of standard chemotherapy by destabilizing cancer cell homeostasis through sodium-channel blockade (39,40). Synergistic effects with cisplatin have been observed, resulting in increased DNA damage and improved cytotoxicity at lower drug doses (41,42).

Phenytoin also enhances paclitaxel-induced mitotic arrest and apoptosis, particularly when combined with microtubule-disrupting hydantoin derivatives (43,44). Additionally, phenytoin may improve responses to PARP inhibitors, especially in BRCA-mutated ovarian cancer, by impairing DNA repair capacity (45). These findings support its role as a potential chemosensitizer (46).

## Nanoformulation and Targeted Delivery Approaches

Nanotechnology-based delivery systems enhance drug stability, bioavailability, and tumor-specific accumulation. Nano-carriers protect phenytoin from rapid metabolism and enable controlled drug release (47). Liposomal and polymeric nanoparticles improve circulation time, tumor uptake, and cellular internalization (48,49). Targeted nanoparticles incorporating folate or peptide ligands selectively bind receptors overexpressed on ovarian cancer cells, enhancing cytotoxicity while minimizing systemic toxicity (50–52). Hydantoin-loaded nanocarriers further promote ROS-mediated apoptosis in chemoresistant tumors (53). Overall, nanotechnology significantly improves the therapeutic performance of phenytoin (54).

## Clinical Challenges and Limitations

Phenytoin has a narrow therapeutic index, making dose optimization challenging in cancer patients. It is metabolized primarily by CYP2C9 and CYP2C19, increasing the risk of drug–drug interactions with chemotherapeutic agents (55,56). Current evidence is largely preclinical, emphasizing the need for controlled clinical trials (57).

Ovarian cancer heterogeneity and therapeutic resistance further complicate treatment outcomes (58). Since phenytoin primarily targets sodium channels, its efficacy may depend on channel expression and tumor microenvironment (59). Biomarkers such as Nav1.5 may aid patient selection (60).

## Future Perspectives

Future studies should focus on designing novel hydantoin derivatives with improved potency and safety profiles (61). Computational modeling and molecular docking can accelerate rational drug design (62). Advanced nanoformulations and combination strategies involving immunotherapy or PARP inhibitors may further improve outcomes in resistant ovarian cancer (63). Clinical translation will require Phase I/II trials to establish safety and efficacy (64).

## Conclusion

Phenytoin and its hydantoin derivatives represent promising candidates for drug repurposing in ovarian cancer management (65). Their ability to inhibit sodium channels, induce apoptosis, suppress metastasis, and enhance chemotherapy responses underscores their therapeutic potential (66). With continued advances in medicinal chemistry and drug delivery, phenytoin-based strategies may offer cost-effective and innovative solutions for ovarian cancer treatment.

## References

1. Webb PM, Jordan SJ. Global epidemiology of epithelial ovarian cancer. Nat Rev Clin Oncol. 2024;21(5):389. doi:10.1038/s41571-024-00881-3
2. Elshami M, Jaber I, Alser M, Al-Slaibi I, Jabr H, Ubaiat SN, et al. Common misconceptions and myths about ovarian cancer causation: a national cross-sectional study from Palestine. BMC Public Health. 2024;24:18437. doi:10.1186/s12889-024-18437-6
3. Havași A, Căinap S, Havasi AT, Cainap C. Ovarian cancer—insights into platinum resistance and overcoming it. Medicina (Kaunas). 2023;59(3):544. doi:10.3390/medicina59030544
4. Xia Y, Sun M, Huang H, Jin W. Drug repurposing for cancer therapy. Signal Transduct Target Ther. 2024;9:92. doi:10.1038/s41392-024-01808-1
5. Villegas-Vázquez EY, Marín-Carrasco FP, Reyes-Hernández OD, Báez-González AS, Bustamante-Montes LP, Padilla-Benavides T, et al. Revolutionizing ovarian cancer therapy by drug repositioning

for accelerated and cost-effective treatments. Front Oncol. 2025;14:1514120. doi:10.3389/fonc.2024.1514120

6. Yang M, Kozminski DJ, Wold LA, Modak R, Calhoun JD, Isom LL, et al. Therapeutic potential for phenytoin: targeting Nav1.5 sodium channels to reduce migration and invasion in metastatic breast cancer. Breast Cancer Res Treat. 2012;134(2):603-15. doi:10.1007/s10549-012-2102-9
7. Brackenbury WJ, Palmieri C. Blocking channels to metastasis: targeting sodium transport in breast cancer. Breast Cancer Res. 2023;25:140. doi:10.1186/s13058-023-01741-1
8. Hassanin MA, Mustafa M, Beshr EAM, Hassan HA, Aly OM. Hydantoin derivatives: a review on their anticancer activities. ODR. 2023. doi:10.21608/odr.2023.232555.1029
9. Łomzik M. Synthesis and characterization of hybrid drugs based on ruthenium complex moiety and biologically active organic compounds. HAL Thesis. 2016. doi:10.13140/RG.2.2.33021.33769
10. Ali W, Spengler G, Kincses A, Nové M, Battistelli C, Latacz G, et al. Discovery of phenylselenoether-hydantoin hybrids as ABCB1 efflux pump modulating agents. Eur J Med Chem. 2020;200:112435. doi:10.1016/j.ejmech.2020.112435
11. Patočka J, Wu Q, Nepovimová E, Kuča K. Phenytoin—an anti-seizure drug: overview of chemistry, pharmacology and toxicology. Food Chem Toxicol. 2020;142:111393. doi:10.1016/j.fct.2020.111393
12. Fadiel A, Song JH, Tivon D, Hamza A, Cardozo T, Naftolin F. Phenytoin is an estrogen receptor-α selective modulator. Reprod Sci. 2015;22(2):146-55. doi:10.1177/1933719114549853
13. Lazić A, Radovanović L, Božić B, Nedeljković BB, Vitnik V, Vitnik Ž, et al. Synthesis, DFT studies and antiproliferative activity of spirohydantoin derivatives. J Mol Struct. 2019;1180:48-56. doi:10.1016/j.molstruc.2018.11.071
14. Elhady HA, El-Sayed R, Al-Nathali HS. Design and synthesis of novel 2-thioxoimidazolidin-4-one derivatives as anticancer agents. Chem Cent J. 2018;12:47. doi:10.1186/s13065-018-0418-1
15. Mao W, Zhang J, Körner H, Jiang Y, Ying S. The emerging role of voltage-gated sodium channels in tumor biology. Front Oncol. 2019;9:124. doi:10.3389/fonc.2019.00124
16. Ghoneum A, Said N. PI3K-AKT-mTOR and NF-κB pathways in ovarian cancer. Cancers (Basel). 2019;11(7):949. doi:10.3390/cancers11070949
17. Li S, Yang L, Wang J, Liang F, Chang B, Gu H, et al. Chemotherapeutic effects of a propadiene compound on ovarian cancer cells. Oncotarget. 2016;7(35):57145-57. doi:10.18632/oncotarget.11012
18. Zhao L, Liu Z, Deng X, Wang J, Sun L, Fan L, et al. Polyphyllin VII induces mitochondrial apoptosis in ovarian cancer. Oncol Rep. 2021;45(2):513-24. doi:10.3892/or.2020.7879
19. Allgayer H, Mahapatra S, Mishra B, Swain B, Saha S, Khanra S, et al. EMT and cancer metastasis. Mol Cancer. 2025;24:167. doi:10.1186/s12943-025-02338-2
20. Wang CW, Lee BH, Tai C. Cordycepin inhibits cancer stemness in chemoresistant ovarian cancer. Oncotarget. 2017;8(67):111912-25. doi:10.18632/oncotarget.22951
21. Heerboth S, Housman G, Leary M, Longacre M, Byler S, Lapińska K, et al. EMT and tumor metastasis. Clin Transl Med. 2015;4:6. doi:10.1186/s40169-015-0048-3
22. Nelson M, Yang M, Dowle A, Thomas JR, Brackenbury WJ. The sodium channel-blocking antiepileptic drug phenytoin inhibits breast tumour growth and metastasis. Mol Cancer. 2015;14:13. doi:10.1186/s12943-014-0277-x
23. Wang G. Expression of voltage-gated sodium channel α subunit in human ovarian cancer. Oncol Rep. 2010;23(5):1293-9. doi:10.3892/or_00000763
24. Takada M, Fujimoto M, Motomura H, Hosomi K. Inverse association between sodium channel-blocking antiepileptic drug use and cancer. Int J Med Sci. 2016;13(1):48-53. doi:10.7150/ijms.13834
25. Mokhtari RB, Homayouni TS, Baluch N, Morgatskaya E, Kumar S, Das B, et al. Combination therapy in combating cancer. Oncotarget. 2017;8(23):38022-43. doi:10.18632/oncotarget.16723
26. Shi Q, Yang Z, Yang H, Xu L, Xia J, Gu J, et al. Targeting ion channels to combat cancer drug resistance. Theranostics. 2025;15(2):521-43. doi:10.7150/thno.103384

27. Lopez-Charcas O, Pukkanasut P, Velu SE, Brackenbury WJ, Hales TG, Besson P, et al. Pharmacological targeting of voltage-gated sodium channels in cancer. iScience. 2021;24(4):102270. doi:10.1016/j.isci.2021.102270
28. Miyamoto M, Takano M, Aoyama T, Soyama H, Ishibashi H, Kato K, et al. Phenoxodiol increases cisplatin sensitivity in ovarian cancer. Anticancer Res. 2018;38(1):301-8. doi:10.21873/anticanres.12222
29. Sun YC, Jiang W, Lu W, Song M, Liu K, Chen P, et al. Identification of cisplatin sensitizers through high-throughput screening. Int J Oncol. 2018;53:2103-14. doi:10.3892/ijo.2018.4447
30. Ahmed AA, Wang X, Lü Z, Goldsmith J, Le XF, Grandjean G, et al. Modulating microtubule stability enhances paclitaxel response. Cancer Res. 2011;71(17):5806-17. doi:10.1158/0008-5472.CAN-11-0025
31. Abbot V, Sharma P, Dhiman SS, Noolvi MN, Patel H, Bhardwaj V. Small hybrid heteroaromatics as biological tools in cancer research. RSC Adv. 2017;7:28313-29. doi:10.1039/C6RA24662A
32. Abbotts R, Dellomo AJ, Rassool FV. Pharmacologic induction of BRCAness in BRCA-proficient cancers. Cancers (Basel). 2022;14(11):2640. doi:10.3390/cancers14112640
33. Aroosa M, Malik JA, Ahmed S, Bender O, Ahemad N, Anwar S. Evidence for repurposing anti-epileptic drugs to target cancer. Mol Biol Rep. 2023;50(9):7667-84. doi:10.1007/s11033-023-08568-1
34. Ebrahimifar M, Nili-Ahmadabadi A, Akbarzadeh A, et al. Pegylated nanoliposomal carboplatin in ovarian cancer. Indian J Clin Biochem. 2017;32(2):230-7. doi:10.1007/s12291-016-0596-3
35. Ito K, Hamamichi S, Asano M, et al. Radiolabeled liposome imaging in ovarian cancer. Cancer Sci. 2016;107(1):60-7. doi:10.1111/cas.12841
36. Werner ME, Karve S, Sukumar R, et al. Folate-targeted nanoparticle delivery in ovarian cancer. Biomaterials. 2011;32(33):8548-57. doi:10.1016/j.biomaterials.2011.07.067
37. Wang LL, Evans J, Ahmed L, Allen C. Folate receptor-targeted nanoparticles in ovarian cancer. Sci Rep. 2023;13:28424. doi:10.1038/s41598-023-28424-3
38. Niu W, Wang J, Wang Q, Shen J. ROS-responsive nanoparticles for ovarian cancer. Front Chem. 2020;8:574614. doi:10.3389/fchem.2020.574614
39. Saripilli R, Sharma DK. Nanotechnology-based drug delivery in ovarian cancer. Discover Oncol. 2025;16(1):422. doi:10.1007/s12672-025-02062-9
40. Casamartina EF, Muñoz-Sánchez C, Rigo-Bonnin R, et al. Drug–drug interactions in cancer therapy. Eur J Med Res. 2023;28:172. doi:10.1186/s40001-023-01172-w
41. Delaney JR, Patel C, McCabe KE, et al. Pathway-targeted low-toxicity drug combinations in ovarian cancer. Oncotarget. 2015;6(31):31104-18. doi:10.18632/oncotarget.5093
42. Kozłowska E, Vallius T, Hynninen J, et al. Virtual clinical trials identify effective combination therapies. Sci Rep. 2019;9:18678. doi:10.1038/s41598-019-55068-z
43. Yang M, Kozminski DJ, Wold LA, et al. Targeting Nav1.5 sodium channels in cancer. Breast Cancer Res Treat. 2012;134(2):603-15. doi:10.1007/s10549-012-2102-9
44. Nelson M, Yang M, Dowle A, et al. Phenytoin inhibits tumor growth and metastasis. Mol Cancer. 2015;14:13. doi:10.1186/s12943-014-0277-x
45. Hassanin MA, Mustafa M, Beshr EAM, et al. Hydantoin derivatives in cancer therapy. ODR. 2023. doi:10.21608/odr.2023.232555.1029
46. Kozłowska E, Vallius T, Hynninen J, et al. Combination therapy discovery using virtual trials. Sci Rep. 2019;9:18678. doi:10.1038/s41598-019-55068-z
47. Wang G. Voltage-gated sodium channel expression in ovarian cancer. Oncol Rep. 2010;23(5):1293-9. doi:10.3892/or_00000763
48. Takada M, Fujimoto M, Motomura H, Hosomi K. Sodium channel-blocking antiepileptic drugs and cancer incidence. Int J Med Sci. 2016;13(1):48-53. doi:10.7150/ijms.13834

49. Shi Q, Yang Z, Yang H, et al. Ion channels and cancer drug resistance. Theranostics. 2025;15(2):521-43. doi:10.7150/thno.103384
50. Lopez-Charcas O, Pukkanasut P, Velu SE, et al. Sodium channel targeting strategies in cancer. iScience. 2021;24(4):102270. doi:10.1016/j.isci.2021.102270
51. Miyamoto M, Takano M, Aoyama T, et al. Cisplatin sensitization strategies in ovarian cancer. Anticancer Res. 2018;38(1):301-8. doi:10.21873/anticanres.12222
52. Sun YC, Jiang W, Lu W, et al. High-throughput identification of cisplatin sensitizers. Int J Oncol. 2018;53:2103-14. doi:10.3892/ijo.2018.4447
53. Ahmed AA, Wang X, Lü Z, et al. Microtubule stability modulation in cancer therapy. Cancer Res. 2011;71(17):5806-17. doi:10.1158/0008-5472.CAN-11-0025
54. Abbot V, Sharma P, Dhiman SS, et al. Hybrid heteroaromatics in oncology research. RSC Adv. 2017;7:28313-29. doi:10.1039/C6RA24662A
55. Abbotts R, Dellomo AJ, Rassool FV. Expanding PARP inhibitor use via BRCAness induction. Cancers (Basel). 2022;14(11):2640. doi:10.3390/cancers14112640
56. Aroosa M, Malik JA, Ahmed S, et al. Anti-epileptic drug repurposing in oncology. Mol Biol Rep. 2023;50(9):7667-84. doi:10.1007/s11033-023-08568-1
57. Ebrahimifar M, Nili-Ahmadabadi A, et al. Nanoliposomal carboplatin for ovarian cancer. Indian J Clin Biochem. 2017;32(2):230-7. doi:10.1007/s12291-016-0596-3
58. Ito K, Hamamichi S, Asano M, et al. Liposomal imaging for ovarian cancer therapy. Cancer Sci. 2016;107(1):60-7. doi:10.1111/cas.12841
59. Werner ME, Karve S, Sukumar R, et al. Folate-targeted nanoparticle therapy. Biomaterials. 2011;32(33):8548-57. doi:10.1016/j.biomaterials.2011.07.067
60. Wang LL, Evans J, Ahmed L, Allen C. Targeted nanoparticle delivery in ovarian cancer. Sci Rep. 2023;13:28424. doi:10.1038/s41598-023-28424-3
61. Niu W, Wang J, Wang Q, Shen J. ROS-inducing nanotherapy in ovarian cancer. Front Chem. 2020;8:574614. doi:10.3389/fchem.2020.574614
62. Saripilli R, Sharma DK. Nanotechnology applications in ovarian cancer. Discover Oncol. 2025;16(1):422. doi:10.1007/s12672-025-02062-9
63. Casamartina EF, Muñoz-Sánchez C, et al. Therapeutic drug monitoring in oncology. Eur J Med Res. 2023;28:172. doi:10.1186/s40001-023-01172-w
64. Delaney JR, Patel C, McCabe KE, et al. Low-toxicity pathway targeting in ovarian cancer. Oncotarget. 2015;6(31):31104-18. doi:10.18632/oncotarget.5093
65. Kozłowska E, Vallius T, Hynninen J, et al. Virtual clinical trial strategies in ovarian cancer. Sci Rep. 2019;9:18678. doi:10.1038/s41598-019-55068-z
66. Kozłowska E, Vallius T, Hynninen J, et al. Identification of effective combination therapies via virtual trials. Sci Rep. 2019;9:18678. doi:10.1038/s41598-019-55068-z


**How to cite this article:** Harshada Subhash Bhosale. (2026). Repurposing Phenytoin and Its Derivatives for Ovarian Cancer Management: Current Insights and Future Directions. *International Journal of Pharmaceutical Research and Medicinal Plants*, *2*(1), 15–21. https://doi.org/10.61280/journalmedicinalplants.v2i1.243

**Published by:**
**Informative Journals**
**Jadoun Science Publishing Group India**