

## EXPLORING CANCER-RELATED THERAPEUTIC ROLES OF PSYCHIATRIC MEDICATIONS: MECHANISTIC INSIGHTS AND CLINICAL RELEVANCE

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### ABSTRACT

Cancer continues to impose a substantial global health burden, while the development of novel anticancer agents remains costly, time-consuming, and inefficient. Drug repurposing has therefore emerged as a strategic alternative, enabling the identification of new oncological applications for established drugs with known pharmacokinetic and safety profiles. Psychiatric medications, including antidepressants, antipsychotics, mood stabilizers, and monoamine oxidase inhibitors, have attracted increasing interest due to accumulating evidence of their anticancer potential. Preclinical and translational studies demonstrate that several psychiatric drugs exert antineoplastic effects by modulating key molecular pathways involved in tumour growth and progression, such as apoptosis, autophagy, epigenetic regulation, immune and inflammatory signalling, angiogenesis, and cellular metabolism. Agents such as valproic acid, selective serotonin reuptake inhibitors, thioridazine, and penfluridol have shown significant antiproliferative and pro-apoptotic activity across diverse cancer models. In parallel, depression is highly prevalent among patients with cancer and is associated with impaired treatment adherence, reduced quality of life, and poorer clinical outcomes, highlighting the dual relevance of psychiatric drugs in oncology. This review synthesizes current evidence on the anticancer mechanisms of psychiatric medications, evaluates their therapeutic implications in cancer-associated depression, and discusses safety considerations, drug–drug interactions, and challenges related to clinical translation. Overall, psychiatric drug repurposing represents a promising, cost-effective strategy that may complement conventional anticancer therapies and contribute to more comprehensive, patient-centred cancer management. Further well-designed clinical trials are required to establish efficacy and define optimal therapeutic roles.

**KEYWORDS:** Psychiatric drug repurposing, Anti-cancer mechanisms, Antidepressants, Antipsychotics, Cancer-associated depression, Apoptosis and autophagy, Translational oncology.

## INTRODUCTION

Cancer is one of the main causes of the world's illness burden and a significant public health issue. By 2030, the International Agency for Research on Cancer (IARC) projects that population ageing and increase alone will result in 13 million cancer-related deaths and 21.7 million new cancer cases. Future burdens are further increased by genetic participation, chemical and radiation exposure, poor dietary habits and other risk factors. Alkylating drugs, antimetabolites, antibiotics, topoisomerase inhibitors, and mitotic antagonists are the principal chemotherapy therapies used today, and they haven't changed much in thirty years. The US Food and Drug Administration's (FDA) list of approved drugs is consistently dominated by anticancer treatments, notwithstanding a brief decrease in 2016.

Discovering a novel treatment for cancer is still an extremely difficult task. The FDA only approves about ten new cancer medications each year. One of the biggest obstacles to cancer treatment discovery is the lengthy and expensive nature of new drug development. It can take several years to demonstrate a new medication's effectiveness and safety. It takes 15 years and US\$802 million to launch a new medication, according to an analysis of 68 randomly chosen authorised medications. Additionally, the overall cost of pre-approval is rising at a 7.4% yearly pace.<sup>[1]</sup> The difficulties in creating new medications point to the necessity of investigating fresh, creative, and reasonably priced methods of treating cancer in humans. The technique known as "drug repurposing" or "drug repositioning," which involves changing the indications of currently available medications from one therapeutic area to involve the treatment for various diseases, reduces the time needed for clinical usage based on the outcomes of prior drug clinical trials and toxicological testing. Compared to conventional drug development, this novel approach to drug discovery has several advantages.<sup>[2,3]</sup> It has demonstrated notable *in vitro* efficacy against both susceptible and multidrug-resistant *Mycobacterium TB* strains.<sup>[4]</sup> Methicillin-susceptible *Staphylococcus aureus* (MSSA) inside cells.<sup>[5]</sup> MRSA, or methicillin-resistant *S. aureus*.<sup>[6]</sup> Due to its negative side effects, thioridazine's use as an antipsychotic medication has decreased; nonetheless, studies and the new finding of its antibacterial properties show that its therapeutic usefulness is feasible and reliable. To further understand additional possible clinical applications of this drug, more research could be done. Other examples include haloperidol and its derivative, bromperidol, which are currently being used to treat a variety of fungal infections.<sup>[7]</sup> Penfluridol, a first-generation typical antipsychotic medication, has been shown to prevent the growth of pancreatic tumours by triggering autophagy-mediated apoptosis.<sup>[8]</sup>

### Psychiatric Drugs Anti-Cancer Effects

The growing severity of the worldwide cancer burden and the high expense of creating new therapies have raised interest in the study and creation of innovative, reasonably priced antineoplastic drugs. In addition to their antipsychotic effects, psychiatric medications, which have been used for decades to treat a variety of mental illnesses, are now known to have strong anticancer effects against a wide range of cancers. Epidemiological research examining the connection between schizophrenia and cancer has produced contradictory findings for over a century.<sup>[9]</sup> After adjusting for known risk and demographic characteristics, a population-based study conducted in the United States similarly showed that people with schizophrenia had a lower cancer risk than the general population.<sup>[10]</sup> Due to genetic, environmental, and other complicating factors, some studies have concluded that people with schizophrenia had a higher or comparable relative risk for cancer compared to the general population.<sup>[11]</sup> Antipsychotic drugs' molecular anticancer actions are yet unclear. The main conditions for which valproic acid is prescribed include migraine headaches, epilepsy, and bipolar disorder. Based on the inhibition of histone deacetylase (HDAC), it has also been found to be a promising antineoplastic medication. To assess its anticancer properties against various tumours, more than 80 clinical studies have

been started.<sup>[12]</sup> shown that it specifically causes cytotoxicity and antiproliferative action in leukemic cells by inhibiting mitochondrial DNA polymerase and reducing ATP generation. Additionally, another team discovered that thioridazine is cytotoxic to the gastric cancer cell lines NCIN87 and AGS via the mitochondrial mechanism.<sup>[13]</sup> demonstrated that in biopsy-like Burkitt lymphoma cells, SSRIs directly cause apoptosis-associated cytotoxicity. In a different study, the viability and proliferation of live human colon carcinoma cell lines and colorectal carcinoma cell-xenografted mice were dose-dependently inhibited by paroxetine and sertraline.<sup>[14]</sup>

**Table 1: Psychiatric Drug with Potential Anti-Neoplastic Effects.**

Class	References	Principal Indications For Its Application	Major mechanism of action	Mechanism of anti-tumor effects
Olanzapine Pimozide	(15)	Schizophrenia Bipolar illness Tourette's syndrome Tics that are resistant	An antagonist of the 5-HT7 receptors and the D2, D3, and D4 receptors	Destroys cancer cells by upsetting the balance of cholesterol.
Valproic acid (Valproate, VPA)	(12,16–18)	Bipolar illness Epilepsy Headaches from migraines	Suppression of GABA reuptake and blockade of voltage-gated sodium, potassium, and calcium channels.	Reduces the growth of cancer cells and triggers apoptosis by inhibiting histone deacetylase; it also causes differentiation and prevents angiogenesis.
Selective serotonin reuptake inhibitors (SSRI) Citalopram Fluoxetine Paroxetine Sertraline	(19–21)	Depression Anxiety disorders that are generalised Obsessive-compulsive disorder Disorders related to eating Recovery from a stroke Early ejaculation	Inhibition of serotonin reuptake	Reduces cancer cell proliferation and triggers apoptosis; down-regulates pAKT to facilitate the synergistic anti-proliferative interactions with other chemotherapy medicines.
MAO inhibitors Selegiline Phenelzine Tranylcypromine	(22,23)	Unusual depression Panic attacks Disorder of the borderline personality	Monoamine oxidase inhibition, which stops monoamine neurotransmitters from being broken down.	Inhibits BHC110/LSD1, a crucial chromatin modifying enzyme that can demethylate histones.

### Depression Prevalence in Cancer

Depending on the type and stage of the disease, the timing and method of evaluation, the diagnostic criteria used, and the demographics of the population under study, the reported prevalence of depressive symptoms in cancer has varied. Depression symptoms are more common in cancer patients nearing the end of their lives, as well as in certain types of cancer such lung, pancreatic, gastric, and oropharyngeal malignancies.<sup>[24,25]</sup> In the general population, depression has been found to be two to three times more common in women than in males. However, this difference has not been seen in cancer, possibly because the burden of the disease may be similarly distributed by sex.<sup>[26]</sup> There is a range of depression symptoms, with nonpathologic sorrow at the milder end, minor or subthreshold depression in the middle, and major depression at the more severe end. The American Psychiatric Association's DSM-IV-TR (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision) states that Depressed mood, also known as anhedonia, is one of the five symptoms of serious depression that must be present for at least two weeks. Other symptoms include disturbed eating or sleep, psychomotor agitation or retardation, low energy, feelings of guilt or worthlessness, trouble focussing, or suicidal thoughts. Only two to four of these symptoms must be present for at least two weeks to be identified as minor depression; three to four symptoms must be present consistently for at least two

years to be diagnosed as dysthymia. A so-called adjustment disorder, which is defined as a state of substantial suffering that is more than expected from exposure to a stressor, may also include depressive symptoms.<sup>[27]</sup>

### **Ambiguity of Diagnostic**

The somewhat arbitrary and frequently unclear distinctions between true sadness and subthreshold and significant depression present diagnostic issues in cancer-related depression. The diagnostic significance of psychological and physical symptoms is also often unclear. Fatigue, anorexia, sleeplessness, and cognitive impairment are only a few of the symptoms of cancer and its treatment that are similar to those of depression. Suicidality, or the wish to die quickly, can also be a symptom of depression, though it can also occur in demoralised people who are not clinically depressed.<sup>[28]</sup>

### **Etiological Methods**

We have proposed<sup>14</sup> that the development of depression in cancer patients could be viewed as a last common pathway that arises from the interplay of various individual, psychological, and disease-related factors.<sup>[29]</sup> Younger age, a personal or familial history of depression, a lack of social support, increased attachment anxiety, poor communication with medical carers, and maladaptive coping mechanisms are among the individual and psychological factors that increase the risk of depression in this setting.<sup>[30]</sup> One of the best and most reliable indicators of depressive symptoms is the physical burden of cancer, which is represented by factors including functional disability, disease stage, and the quantity and intensity of physical symptoms.<sup>[31]</sup> Since depression is more common in pancreatic cancer than in other cancer types, it has long been thought that the two conditions are specifically related.<sup>[32]</sup> There was early conjecture that pancreatic cancer was more likely than other cancer kinds to be diagnosed with depression.<sup>[33]</sup>

### **Treatment**

When treating depression in cancer patients, it is important to address not just the depressive symptoms but also the psychosocial and disease-related variables that contribute to the development of depression in this setting. These include the management of pain and other uncomfortable physical symptoms, the interaction with oncologists and other medical professionals, the network of social support, and the personal experience of disease. Those with more severe depression respond well to antidepressant drugs.<sup>[34]</sup>

Studies on pharmacologic and psychotherapy treatments for depression in cancer patients have yielded conflicting and unclear findings. The methodologic limitations of published research, such as small sample sizes and failure to specify depression severity, as well as other variations in inclusion criteria, demographic, disease-related, and treatment characteristics of the samples studied, and follow-up duration, may contribute to this.<sup>[35]</sup>

### **Drug Interactions and Toxicity**

Recently, there has been discussion concerning how all kinds of antidepressant medications may exacerbate suicidality, especially in teenagers and young adults. Because antidepressants may change the pharmacokinetics of other drugs commonly prescribed for cancer patients, drug interactions should be taken into account when using antidepressants.<sup>[36]</sup> Therefore, when medication for depression is indicated in this population, drugs that have little to no effect on the 2D6 isoenzyme (such as citalopram or venlafaxine) should be utilised.<sup>[37]</sup>

### Repurposing Psychiatric Drugs: Challenges and Restrictions

The anticancer effects of mental drugs against various cancers have been validated by numerous research. However, there are still obstacles and restrictions in the repurposing of these medications that must be taken into account before they are used in further clinical settings. The carcinogenicity of numerous kinds of psychiatric medications, such as antipsychotics, antidepressants, anticonvulsants, benzodiazepines/sedative-hypnotics, and amphetamines, was summarised in a systematic analysis and evaluation of mental medications from FDA rodent studies.<sup>[38]</sup> The results of clinical research on the impact of mental medications on cancer risk are also debatable. The use of SSRIs was linked to a lower risk of epithelial ovarian cancer when compared to non-use and other antidepressants, according to a study that evaluated the relationships between different antidepressants and the risk using Danish national registries.<sup>[39]</sup>

### Directions for the Future

Recently, a range of customised novel interventions have been developed for patients with various cancer-related issues and at various stages of the disease (e.g., a geriatric-specific group psychoeducational intervention, CALM for patients with metastatic cancer, and Dignity Therapy for patients near the end of life). There will soon be more proof of their advantages in treating and preventing depression in cancer patients. The creation and assessment of more focused pharmaceutical therapies to treat depression in cancer patients may be made possible by the identification of cytokine-mediated and other biologic pathways to depression in these patients.<sup>[40]</sup>

### DISCUSSION

Psychiatric medications exhibit a number of qualities that make them appealing options for repurposing in malignancies from a translational standpoint. Numerous antidepressants and antipsychotics have the potential to directly affect tumour biology because they alter intracellular signalling pathways related to cell survival, apoptosis, autophagy, and treatment resistance.<sup>[41]</sup> Immune dysregulation, chronic inflammatory conditions, oxidative stress, and neuroendocrine imbalance are among the molecular pathways that are increasingly recognised to be shared by psychiatric disorders and cancer growth, as evidenced by the integration of oncological and psychiatric findings. Through changes in cytokine signalling, inhibition of antitumor immunity, and stimulation of the hypothalamic-pituitary-adrenal axis, psychological stress and depression have been demonstrated to affect tumour initiation and progression, thereby producing a milieu favourable to cancer growth.<sup>[42]</sup> Furthermore, as effective therapy of psychiatric complications has been linked to better adherence to therapy, quality of life, and potentially survival outcomes, the significant frequency of depression among patients with cancer further supports their therapeutic relevance.<sup>[43]</sup> Crucially, when compared to de novo antitumor drug discovery, medication repurposing offers useful benefits such as lower research costs, proven safety profiles, and quicker clinical translation.<sup>[44]</sup> Emerging evidence suggests that certain psychiatric drugs may enhance the efficacy of chemotherapy and radiotherapy by sensitizing cancer cells and overcoming resistance mechanisms, thereby supporting their use as adjuvant agents.<sup>[45]</sup>

### CONCLUSION

In conclusion, because psychiatric drugs can alter important physiological and molecular pathways involved in tumour progression, an increasing amount of translational evidence points to them as attractive candidates for repurposing in cancer therapy. The therapeutic promise of these medicines beyond their conventional indications is reinforced by the significant confluence between mental health and cancer highlighted by the common molecular pathways underlying psychiatric illnesses and malignancies. Psychiatric medications may help develop more thorough and patient-centered

cancer management approaches by targeting both tumour biology and psychosocial comorbidities. Their clinical relevance is further reinforced by their proven safety profiles, affordability, and capacity to improve responsiveness to traditional anticancer treatments. When taken as a whole, these findings highlight the necessity of ongoing multidisciplinary research and well planned clinical trials in order to confirm effectiveness, improve treatment plans, and completely include mental medication repurposing into oncological practice going forward.

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#### REFERENCES

1. Huang J, Zhao D, Liu Z, Liu F. Repurposing psychiatric drugs as anti-cancer agents. *Cancer Lett*, 2018 Apr; 419: 257–65.
2. Thanacoody HKR. Thioridazine: resurrection as an antimicrobial agent? *Br J Clin Pharmacol*, 2007; 64(5): 566–74.
3. Amaral L, Viveiros M, Kristiansen J. ‘Non-Antibiotics’: Alternative Therapy for the Management of MDRTB and MRSA in Economically Disadvantaged Countries. *Curr Drug Targets*, 2006; 7(7): 887–91.
4. Ordway D, Viveiros M, Leandro C, Bettencourt R, Almeida J, Martins M, et al. Clinical Concentrations of Thioridazine Kill Intracellular Multidrug-Resistant *Mycobacterium tuberculosis*. *Antimicrob Agents Chemother*, 2003; 47(3): 917–22.
5. Ordway D, Viveiros M, Leandro C, Arroz MJ, Amaral L. Intracellular activity of clinical concentrations of phenothiazines including thioridazine against phagocytosed *Staphylococcus aureus*. *Int J Antimicrob Agents*, 2002 July; 20(1): 34–43.
6. Kristiansen M. Phenothiazines alter resistance of methicillin-resistant strains of *Staphylococcus aureus* (MRSA) to oxacillin in vitro. *Int J Antimicrob Agents*, 2003 Sept; 22(3): 250–3.
7. Holbrook SYL, Garzan A, Dennis EK, Shrestha SK, Garneau-Tsodikova S. Repurposing antipsychotic drugs into antifungal agents: Synergistic combinations of azoles and bromperidol derivatives in the treatment of various fungal infections. *Eur J Med Chem*, 2017 Oct; 139: 12–21.
8. Ranjan A, Srivastava SK. Penfluridol suppresses pancreatic tumor growth by autophagy-mediated apoptosis. *Sci Rep*, 2016 May 18; 6(1): 26165.
9. Csatory LaszloK. CHLORPROMAZINES AND CANCER. *The Lancet*, 1972 Aug; 300(7772): 338–9.
10. Cohen ME, Dembling B, Schorling JB. The association between schizophrenia and cancer: a population-based mortality study. *Schizophr Res*, 2002 Oct; 57(2–3): 139–46.
11. Chou FHC, Tsai KY, Su CY, Lee CC. The incidence and relative risk factors for developing cancer among patients with schizophrenia: A nine-year follow-up study. *Schizophr Res*, 2011 July; 129(2–3): 97–103.
12. Michaelis M, Doerr H, Cinatl Jr. J. Valproic Acid As Anti-Cancer Drug. *Curr Pharm Des*, 2007 Nov 1; 13(33): 3378–93.
13. Mu J, Xu H, Yang Y, Huang W, Xiao J, Li M, et al. Thioridazine, an antipsychotic drug, elicits potent antitumor effects in gastric cancer. *Oncol Rep*, 2014 May; 31(5): 2107–14.

14. Gil-Ad I. Evaluation of the potential anti-cancer activity of the antidepressant sertraline in human colon cancer cell lines and in colorectal cancer-xenografted mice. *Int J Oncol* [Internet]. 1992 [cited 2025 Dec 10]; Available from: [http://www.spandidos-publications.com/ijo/article.jsp?article\\_id=ijo\\_33\\_2\\_277](http://www.spandidos-publications.com/ijo/article.jsp?article_id=ijo_33_2_277)
15. Wiklund ED, Catts VS, Catts SV, Ng TF, Whitaker NJ, Brown AJ, et al. Cytotoxic effects of antipsychotic drugs implicate cholesterol homeostasis as a novel chemotherapeutic target. *Int J Cancer*, 2010 Jan; 126(1): 28–40.
16. Berendsen S, Broekman M, Seute T, Snijders T, Van Es C, De Vos F, et al. Valproic acid for the treatment of malignant gliomas: review of the preclinical rationale and published clinical results. *Expert Opin Investig Drugs*, 2012 Sept; 21(9): 1391–415.
17. Ryu CH, Yoon WS, Park KY, Kim SM, Lim JY, Woo JS, et al. Valproic Acid Downregulates the Expression of MGMT and Sensitizes Temozolomide-Resistant Glioma Cells. *J Biomed Biotechnol*, 2012; 2012: 1–9.
18. Osuka S, Takano S, Watanabe S, Ishikawa E, Yamamoto T, Matsumura A. Valproic Acid Inhibits Angiogenesis In Vitro and Glioma Angiogenesis In Vivo in the Brain. *Neurol Med Chir (Tokyo)*, 2012; 52(4): 186–93.
19. Serafeim A, Holder MJ, Grafton G, Chamba A, Drayson MT, Luong QT, et al. Selective serotonin reuptake inhibitors directly signal for apoptosis in biopsy-like Burkitt lymphoma cells. *Blood*, 2003 Apr 15; 101(8): 3212–9.
20. Gil-Ad I. Evaluation of the potential anti-cancer activity of the antidepressant sertraline in human colon cancer cell lines and in colorectal cancer-xenografted mice. *Int J Oncol* [Internet]. 1992 [cited 2025 Dec 10]; Available from: [http://www.spandidos-publications.com/ijo/article.jsp?article\\_id=ijo\\_33\\_2\\_277](http://www.spandidos-publications.com/ijo/article.jsp?article_id=ijo_33_2_277)
21. Liu F, Huang J, Ning B, Liu Z, Chen S, Zhao W. Drug Discovery via Human-Derived Stem Cell Organoids. *Front Pharmacol* [Internet]. 2016 Sept 22 [cited 2025 Dec 10];7. Available from: <http://journal.frontiersin.org/Article/10.3389/fphar.2016.00334/abstract>
22. Bennani-Baiti IM, Machado I, Llombart-Bosch A, Kovar H. Lysine-specific demethylase 1 (LSD1/KDM1A/AOF2/BHC110) is expressed and is an epigenetic drug target in chondrosarcoma, Ewing's sarcoma, osteosarcoma, and rhabdomyosarcoma. *Hum Pathol*, 2012 Aug; 43(8): 1300–7.
23. Baxter E, Windloch K, Gannon F, Lee JS. Epigenetic regulation in cancer progression. *Cell Biosci*, 2014 Dec; 4(1): 45.
24. Brintzenhofe-Szoc KM, Levin TT, Li Y, Kissane DW, Zabora JR. Mixed Anxiety/Depression Symptoms in a Large Cancer Cohort: Prevalence by Cancer Type. *Psychosomatics*, 2009 July; 50(4): 383–91.
25. Li M, Fitzgerald P, Rodin G. Evidence-Based Treatment of Depression in Patients With Cancer. *J Clin Oncol*, 2012 Apr 10; 30(11): 1187–96.
26. Miller S, Lo C, Gagliese L, Hales S, Rydall A, Zimmermann C, et al. Patterns of depression in cancer patients: an indirect test of gender-specific vulnerabilities to depression. *Soc Psychiatry Psychiatr Epidemiol*, 2011 Aug; 46(8): 767–74.
27. American Psychiatric Association, editor. *Diagnostic and statistical manual of mental disorders: DSM-IV*; includes ICD-9-CM codes effective 1. Oct. 96. 4. ed., 7. print. Washington, DC; 1998. 886 p.
28. Rodin G, Lo C, Mikulincer M, Donner A, Gagliese L, Zimmermann C. Pathways to distress: The multiple determinants of depression, hopelessness, and the desire for hastened death in metastatic cancer patients. *Soc Sci Med*, 2009 Feb; 68(3): 562–9.
29. Salmon RM. The American Psychiatric Publishing Textbook of Psychosomatic Medicine. *J Clin Psychiatry*, 2007 Dec 15; 68(12): 1990.

30. Li M, Boquiren V, Lo C, Rodin G. Depression and anxiety in supportive oncology. In: Supportive Oncology [Internet]. Elsevier; 2011 [cited 2025 Dec 11]. p. 528–40. Available from: <https://linkinghub.elsevier.com/retrieve/pii/B9781437710151000527>
31. Rodin G, Zimmermann C, Rydall A, Jones J, Shepherd FA, Moore M, et al. The Desire for Hastened Death in Patients with Metastatic Cancer. *J Pain Symptom Manage*, 2007 June; 33(6): 661–75.
32. Li M, Fitzgerald P, Rodin G. Evidence-Based Treatment of Depression in Patients With Cancer. *J Clin Oncol*, 2012 Apr 10; 30(11): 1187–96.
33. Kenner BJ. Early Detection of Pancreatic Cancer: The Role of Depression and Anxiety as a Precursor for Disease. *Pancreas*, 2018 Apr; 47(4): 363–7.
34. Fournier JC, DeRubeis RJ, Hollon SD, Dimidjian S, Amsterdam JD, Shelton RC, et al. Antidepressant Drug Effects and Depression Severity: A Patient-Level Meta-analysis. *JAMA*, 2010 Jan 6; 303(1): 47.
35. Rodin G, Lloyd N, Katz M, Green E, Mackay JA, Wong RKS, et al. The treatment of depression in cancer patients: a systematic review. *Support Care Cancer*, 2007 Feb 5; 15(2): 123–36.
36. Sandson NB, Armstrong SC, Cozza KL. An Overview of Psychotropic Drug-Drug Interactions. *Psychosomatics*, 2005 Sept; 46(5): 464–94.
37. Zembutsu H, Sasa M, Kiyotani K, Mushiroda T, Nakamura Y. Should CYP2D6 inhibitors be administered in conjunction with tamoxifen? *Expert Rev Anticancer Ther*, 2011 Feb; 11(2): 185–93.
38. Amerio A, Gálvez JF, Odone A, Dalley SA, Ghaemi SN. Carcinogenicity of psychotropic drugs: A systematic review of US Food and Drug Administration–required preclinical in vivo studies. *Aust N Z J Psychiatry*, 2015 Aug; 49(8): 686–96.
39. Mørch LS, Dehlendorff C, Baandrup L, Friis S, Kjær SK. Use of antidepressants and risk of epithelial ovarian cancer. *Int J Cancer*, 2017 Dec; 141(11): 2197–203.
40. Holland J, Poppito S, Nelson C, Weiss T, Greenstein M, Martin A, et al. Reappraisal in the eighth life cycle stage: A theoretical psychoeducational intervention in elderly patients with cancer. *Palliat Support Care*, 2009 Sept; 7(3): 271–9.
41. Avendaño-Félix M, Aguilar-Medina M, Bermudez M, Lizárraga-Verdugo E, López-Camarillo C, Ramos-Payán R. Refocusing the Use of Psychiatric Drugs for Treatment of Gastrointestinal Cancers. *Front Oncol*, 2020 Aug 14; 10: 1452.
42. Antoni MH, Lutgendorf SK, Cole SW, Dhabhar FS, Sephton SE, McDonald PG, et al. The influence of bio-behavioural factors on tumour biology: pathways and mechanisms. *Nat Rev Cancer*, 2006 Mar; 6(3): 240–8.
43. Pasquini M, Biondi M. Depression in cancer patients: a critical review. *Clin Pract Epidemiol Ment Health*, 2007; 3(1): 2.
44. Pushpakom S, Iorio F, Eyers PA, Escott KJ, Hopper S, Wells A, et al. Drug repurposing: progress, challenges and recommendations. *Nat Rev Drug Discov*, 2019 Jan; 18(1): 41–58.
45. Vlachos N, Lampros M, Voulgaris S, Alexiou GA. Repurposing Antipsychotics for Cancer Treatment. *Biomedicines*, 2021 Nov 28; 9(12): 1785.