ORIGINAL RESEARCH

Cannabis Use by Veterans and Potential Interactions With Antineoplastic Agents: Analysis and Literature Review

Tsvetelina Todorova, DO[a]; Elizabeth John, MD[a]; Srishti Sareen, MD[a]; Vaishnavi Tandra, MD[a]; Jessica Davis, DO[b]; Lindsey Lands, MD[a]; Alva Weir III, MD[a]

Background: Extensive research exists investigating medicinal uses of cannabis products in patients with malignancies, though it is largely unknown how cannabis use may affect the efficacy of antineoplastics. Like many antineoplastic agents, cannabis is metabolized by cytochrome P450, leading to potential interactions. This observational study sought to identify the prevalence of cannabis use in patients with a malignancy at the Veterans Affairs Memphis Healthcare System and analyze the potential for interactions between various products and the antineoplastic agents the patients received.
Methods: A retrospective review of electronic health records was completed for patients who used cannabis products during treatment and accepted the invitation to complete a survey during January 2024. Prescribed antineoplastic medications for each patient were recorded, and data were compiled and analyzed for potential interactions.
Results: A total of 132 patients agreed to participate in the survey, and 50 (38%) acknowledged use of cannabis products within the previous 90 days. Forty-five patients (90%) who used cannabis within 90 days were male, and 29 patients (58%) were aged 60 to 65 years. Thirty-five patients (70%) predominantly inhaled cannabis. Twenty-one patients (42%) had chemotherapy, the most common treatment, 19 patients (38%) had immunotherapy, 12 patients (24%) used targeted therapy, and 10 patients (20%) used endocrine therapy.
Conclusions: This analysis found a notable prevalence of patients with cancer concurrently using cannabis while being prescribed an antineoplastic. The results of this study suggest a potential decrease in the efficacy of immunotherapy and endocrine therapy and an increase in toxicity with chemotherapy and targeted agents when used alongside cannabis products.

Author affiliations can be found at the end of this article.
Correspondence: Tsvetelina Todorova (ttodorov@uthsc.edu)

Fed Pract. 2026;43(suppl 2). Published online May 16. doi:10.12788/fp.0619

Cannabis has a long history of use for medicinal and recreational purposes. Research illustrates the potential benefits and increased prevalence of cannabis use in patients with cancer.[1] Cannabis products have been shown to possess antineoplastic and palliative activity, improving nociceptive and neuropathic pain in addition to chemotherapy-related nausea and vomiting.[2-5] Despite these developments and changing social attitudes toward cannabis, there remains a lack of comprehensive data on patient perspectives regarding its use, especially in regions where cannabis remains illegal. This knowledge gap is notable among veterans undergoing cancer treatment in states where cannabis is prohibited. Up to 57% of veterans report lifetime marijuana use, making it crucial to understand this population's cannabis use patterns and potential interactions with cancer treatments.[6]

This observational study sought to determine the prevalence of cannabis use among patients undergoing cancer treatment at the US Department of Veterans Affairs (VA) Memphis Healthcare System and evaluate the potential risks associated with combining cannabis products with anticancer therapies.

METHODS

This prospective observational study identified cannabis use among veterans receiving antineoplastic therapy at the Lt. Col. Luke Weathers Jr. VA Medical Center (WJVAMC) and analyzed potential interactions between cannabis products and their cancer treatments. Participants included adults aged > 18 years undergoing antineoplastic therapy at WJVAMC who consented to the study. Data collection involved a written survey approved by the WJVAMC Institutional Review Board and verbal consent from participants. The survey asked participants about their cannabis use in the previous 90 days, including details on quantity, frequency, and method of consumption (eg, inhalation, oral, topical). No incentives were offered for participation.

Surveys from 50 patients who used cannabis were analyzed and their electronic health records were reviewed for sex, age, diagnosis, and antineoplastic regimen. This information was securely stored. A literature review was conducted using PubMed and the Cochrane Library to explore potential interactions between cannabis and the antineoplastic agents that were prescribed to patients in the study, focusing on toxicity, efficacy, or synergistic effects.

Patients were categorized into 4 groups based on treatment: cytotoxic chemotherapy, immunotherapy, endocrine therapy, and targeted therapy. Patients undergoing multiple types of therapies were included in each applicable category.

RESULTS

A total of 132 patients agreed to participate. Fifty patients (38%) acknowledged using cannabis products within 90 days. The patients that used cannabis products within 90 days of the survey reported the following malignancies: 8 patients (16%) had prostate cancer, 3 patients (6%) had hepatocellular carcinoma, 7 patients (14%) had pancreatic carcinoma, 5 patients (10%) had multiple myeloma, 3 patients (6%) had chronic lymphocytic leukemia, 9 patients (18%) had non-small cell lung cancer, 3 patients (6%) had breast cancer, 3 (6%) patients had bladder cancer, 2 patients (4%) had renal cell carcinoma, 1 (2%) patient had chronic myeloid leukemia, 1 (2%) patient had renal amyloid, 1 patient (2%) had supraglottic squamous cell carcinoma, 1 patient (2%) had esophageal carcinoma, 1 (2%) patient had small cell lung cancer, 1 (2%) patient had gastric cancer, and 1 patient (2%) had follicular lymphoma.

Five (10%) of the cannabis users were female, and 45 (90%) were male. Twenty-nine patients (58%) were aged 66 to 75 years, 16 (32%) were aged 56 to 65 years, 3 (6%) were aged 46 to 55 years, and 2 (4%) were aged 76 to 85 years.

Thirty-five patients (70%) inhaled cannabis as opposed to using it via other formulations or a combination (eg, inhalation and topical). Thirty-eight percent of patients used cannabis once daily, 24% used < 1 daily, and 28% used it ≥ 2 times daily. Five patients (10%) did not report the frequency of their cannabis use. Among the patients who reported cannabis use, 21 (42%) were undergoing cytotoxic chemotherapy, 19 (38%) were undergoing immunotherapy, 12 (24%) were undergoing targeted therapy, and 10 (20%) were undergoing endocrine therapy. Some patients were treated with multiple types of antineoplastic agents and were counted in multiple categories (Table 1).

Following a literature review of cannabis and antineoplastic agents, patients were evaluated for the potential effects of cannabis on their treatment. The literature review revealed that 31% of cytotoxic chemotherapy agents received by patients in this study might have increased toxicity, and 19% could have reduced efficacy when combined with cannabis. Among immunotherapy agents received by patients in this study, 70% might have decreased efficacy when combined with cannabis use. For targeted therapies, 35% could have increased toxicity, and 70% of endocrine agents could potentially have decreased efficacy (Table 2).

TABLE 1. Therapies Used

Type	Specific agent
Cytotoxic chemotherapy	Melphalan, carboplatin, paclitaxel, etoposide, 5-fluorouracil, irinotecan, oxaliplatin, pemetrexed, docetaxel, cabazitaxel, T-DM1, gemcitabine, cyclophosphamide
Immunotherapy	Pembrolizumab, nivolumab, atezolizumab, ipilimumab, avelumab, lenalidomide, durvalumab
Targeted therapy	Zanubrutinib, ibrutinib, sorafenib, acalabrutinib, dabrafenib, trametinib, trastuzumab, bevacizumab, daratumumab, imatinib
Endocrine therapy	Anastrozole, darolutamide, abiraterone, tamoxifen, enzalutamide

DISCUSSION

This prospective study corroborates previous research by demonstrating that more than one-third of patients receiving oncology care at WJVAMC use cannabis, most often inhaled. Cannabis use was observed among patients undergoing various cancer therapies, including cytotoxic chemotherapy, immunotherapy, targeted therapy, and endocrine therapy. The most common malignancies among cannabis users at WJVAMC include patients with lung cancer, prostate cancer, pancreatic cancer, and multiple myeloma. Cannabis use in patients with pancreatic cancer and multiple myeloma was significantly out of proportion to their prevalence at WJVAMC. This could potentially be due to their drastic effect on quality of life.

Cannabis use increased the risk of toxicity in patients treated with cytotoxic chemotherapy and targeted therapy. Cannabis use potentially decreased efficacy for patients treated with cytotoxic chemotherapy and/or immunotherapy. Cannabis use did not increase the risk of toxicity or efficacy in patients treated with endocrine therapy.

Antineoplastics/Cannabis Interactions

The potential interactions between cannabis and antineoplastic therapies administered at WJVAMC are worth exploring. While this review aims to shed light on possible interactions, it is important to acknowledge that much of the data is preliminary and derived from in vitro studies. The interactions should be interpreted as potential risks rather than established facts. Additional research is needed to confirm these interactions and effectively guide clinical practices. Understanding these dynamics is essential to optimize patient care and manage the complex interplay between cannabis use and cancer treatment.

Originating from Central Asia, the cannabis plant contains > 400 medicinally relevant compounds, of which about 100 are cannabinoids (CBs). Key CBs are cannabidiol (CBD), a nonpsychoactive compound, and Δ-9-tetrahydrocannabinol (THC), a psychoactive compound. THC can make up 20% to 30% of the dry weight of female cannabis flowers.[7]

CBs act through the endocannabinoid system, involving CB1 and CB2 receptors, endogenous CBs like anandamide (AEA) and 2-arachidonoylglycerol, and various enzymes. These endogenous CBs, derived from arachidonic acid, play roles in cell growth and proliferation.[8] In some studies, AEA has induced apoptosis in neuroblastoma cells and inhibited proliferation in breast cancer cells. However, other research suggests AEA may block apoptosis under certain conditions.[9]

CB receptors are transmembrane proteins that interact with CBs differently depending on tissue type and CB structure. Synthetic CBs are designed to target specific receptors, while natural CBs may act as both agonists and antagonists.[10]

Cytochrome P450 Metabolism

The human cytochrome P450 (CYP) 3A subfamily affects the metabolism of many therapeutic drugs, including cancer therapeutics.[11] The various compositions of cannabis are primarily metabolized by the CYP450 pathway, the same as many cancer-directed pharmacologic treatments. CBs act as both CYP inducers and inhibitors. THC, for example, is a CYP inducer whereas CBD is a CYP inhibitor; both are found in the various compounds available for consumption.[12,13] Pharmacology research has suggested potential interactions and effects on established adverse symptoms, but clinical data are lacking, and current research revealing interactions are only recognized in vitro.[14]

The Antineoplastic Activity of Cannabis

CBs can affect various cancer-related pathways such as PKB, AMPK, CAMKK-β, mTOR, PDHK, HIF-1 α, and PPAR-γ. Δ-9-THC can selectively induce apoptosis in tumor cells without harming normal cells, though the exact mechanism remains unclear. Promising results from early mouse studies led to a 2006 human study where intracranial Δ-9-THC in patients with recurrent glioma yielded a median survival of 24 weeks, with 2 patients surviving > 1 year.[15]

In a 2022 review article, Cherkasova et al highlighted potential clinical benefits of cannabis across various cancers. They found that upregulated CB1 receptors in colon cancer might enhance the effect of 5-fluorouracil. However, many studies are preliminary and therefore not definitive.[10]

Additional research is needed to refine these findings. Challenges include variability in cannabis formulations, the complex tumor microenvironment, and the legal and psychoactive issues surrounding cannabis use. These factors complicate the design of multicenter randomized studies and may deter patients from disclosing cannabis use, thereby hindering efforts to fully understand its therapeutic potential.

Cannabis/Cytotoxic Chemotherapy Interactions

The chemotherapy agents used in this study included carboplatin, paclitaxel, 5-fluorouracil, etoposide, irinotecan, oxaliplatin, pemetrexed, docetaxel, cabazitaxel, T-DM1, gemcitabine, and cyclophosphamide. There is a paucity of research regarding the interactions between cytotoxic chemotherapy and cannabis. Most studies focused on CBD due to its inhibition of the CYP450 pathway, which is used for metabolizing cytotoxic chemotherapies. Through this mechanism, CBD could potentially increase the concentrations of chemotherapeutic agents, enhancing their toxicity.

When combined with irinotecan, cannabis can pose risks. Δ-9-THC undergoes first-pass metabolism in the liver, mediated by the CYP450 system and CYP3A4. The glucuronidation of irinotecan is mediated by uridine diphosphate glycosyltransferase, leading to its recirculation within the hepatic system and potentially increased toxicity due to prolonged drug presence. Cannabis may also compete with drug binding to albumin, altering the plasma concentrations of irinotecan and its conversion to the metabolite SN38.[16]

Cannabis products can affect chemotherapy levels by interacting with cellular transporters. The MRP1 transporter family, encoded by the *ABCC* gene family, is expressed mainly in the lung, kidney, skeletal muscle, and hematopoietic stem cells. A 2018 study investigating the effects of THC, CBD, and CBN on MRP1 transporters found that the presence of a cannabis component increased the concentration of vincristine 3-fold. Additional studies suggest the interaction with the CB1 receptor may lead to changes in the expression of MRP1 transporters.[17]

CBD inhibits the BCRP transporter, which functions as an efflux pump for methotrexate. Consequently, CBD can increase methotrexate levels, potentially enhancing efficacy but

TABLE 2. Patients With Potential Interactions With Cannabis[a]

Therapy type	Decreased efficacy, No.	Increased toxicity, No.	Potentiation, No.	No interaction, No.
Immunotherapy	14	0	0	6
Chemotherapy	8	13	2	1
Targeted therapy	0	5	0	9
Endocrine therapy	7	1	1	1

[a]Patients undergoing combination therapy with multiple antineoplastic agents were counted more than once.

also worsening adverse effects.[18]

In pancreatic cancer, CBD specifically interacts with gemcitabine. CB1 and CB2 receptors are upregulated, and CBD inhibits the GPR55 receptor. These interactions may enhance the antineoplastic effect of gemcitabine, reducing cell cycle progression and growth.[19]

CBD also interacts with temozolomide (TMZ) by affecting extracellular vesicles used by cells for pro-oncogenic signaling and immune system evasion. Experiments on patient-derived glioblastoma cells, both chemotherapy-resistant and chemotherapy-sensitive, found that CBD increases the formation of extracellular vesicles with reduced levels of miR21 (pro-oncogenic) and elevated levels of miR126 (antioncogenic).[20] CBD has also been found to decrease prohibitin levels, a protein associated with TMZ resistance.

In patients with glioblastoma, CBD combined with chemotherapeutic agents like TMZ, carmustine, doxorubicin, and cisplatin has shown increased sensitivity and improved tumor response. CBD is also known to inhibit NF-kB, a pathway that sustains tumor viability despite chemotherapy.[21] Additionally, CBD inhibits the P-glycoprotein system, affecting chemotherapy efflux from neoplastic cells.[14] In vitro studies have found that CBD is synergistic with bortezomib in inhibiting cancer cell viability. In another glioblastoma model, CBD enhanced the antiproliferative effects of both TMZ and carmustine.[14]

Different cannabis formulations may vary in how they interact with various cytotoxic chemotherapeutic agents. Some may potentiate the effects of chemotherapy and act synergistically to inhibit tumor growth, while others may lead to increased toxicity.[10] More research is needed to determine which formulations, in combination with specific agents and doses, may have significant interactions that warrant adjustments in chemotherapy dosing.

Cannabis/Immunotherapy Interactions

Cannabis is an immunosuppressant. Data suggest the use of cannabis during immunotherapy worsens treatment outcomes in patients with cancer.[22] Exogenous (THC) and endogenous (AEA) CBs negatively affect antitumor immunity by impairing the function of tumor-specific T cells via CB2 and by inhibiting the Jak1-STATs signaling in T cells through CNR2. Xiong et al found that THC reduces the therapeutic effect of anti-PD-1 therapy.[22]

In a prospective observational clinical study, Bar-Sela et al analyzed 102 patients with advanced cancer—of which 68 were cannabis users—that were started on immune checkpoint inhibitor therapy. The study found that cannabis users on anti-PD-1 (nivolumab, pembrolizumab), anti-CTLA-4 (ipilimumab), and anti-PD-L1 (durvalumab, atezolizumab) had a significant decrease in time to treatment progression and overall survival vs cannabis nonusers.[23] However, a 2023 study by Waissengrin et al found that concomitant use of medical cannabis with pembrolizumab had no harmful effect in advanced non-small cell lung cancer.[24] Time to treatment progression of cannabis users did not differ from cannabis nonusers.[25]

Cannabis/Endocrine Therapy Interactions

In addition to having direct antineoplastic activity on tumor cells, data exist that show how cannabis affects the endocrine system. In animal models, cannabis has been found to suppress the whole hypothalamic-pituitary-adrenal axis as well as other hormones like thyroid, prolactin, and growth hormone. In breast cancer, cannabis competes with estrogen for the estrogen receptor and suppresses growth.[26]

The endocrine agents used by patients with cancer in this study were antiandrogens like abiraterone, enzalutamide, tamoxifen and anastrozole. Abiraterone is metabolized by CYP450 isoenzymes and uridine diphosphate glycosyltransferases. Cannabis inhibits both processes and therefore may lead to increased toxicities.[27] Conversely, enzalutamide is a strong CYP3A inducer, and cannabis use during enzalutamide therapy may significantly increase the toxic effects of cannabis.

There is evidence that molecular pathways involving CB receptors and estrogens overlap, which may lead to interactions when antiestrogens are used in cannabis users with hormone receptor-positive breast cancer.[26] In preclinical studies, tamoxifen has been shown to act as an inverse agonist on CB1 and CB2 receptors, though the significance of this finding is unclear. There is no research evaluating the effects of CBs on tamoxifen treatment. However, CBD has been found to potentiate the effectiveness of anastrozole or exemestane in breast cancer cell lines.[28] Dobovišek et al demonstrated no inhibitory effect of CBD on the activity of tamoxifen, fulvestrant, or palbociclib in breast cancer cell lines.[29] The interactions between hormone receptor-positive breast cancer and cannabinoids are complex, and the clinical significance of these interactions remains difficult to identify.

Cannabis/Targeted Therapy Interactions

The targeted therapies used by patients in this study included zanubrutinib, ibrutinib, sorafenib, acalabrutinib, dabrafenib, trametinib, trastuzumab, bevacizumab, daratumumab, and imatinib. Compared to other classes of cancer treatments, most studies have not demonstrated decreased efficacy or increased toxicity of targeted anticancer drugs when used concomitantly with CBD.[29]

Trastuzumab is a recombinant humanized monoclonal antibody that targets the proto-oncogene *HER2/neu*. It is used to treat select patients with metastatic breast cancer. Studies have shown that cannabis use does not attenuate the effectiveness of trastuzumab in HER2-positive and triple-negative breast cancer subtypes.[29] One study found that CBD, in combination with chemotherapeutics and Bruton tyrosine kinase inhibitors, such as ibrutinib and zanubrutinib, has synergistic potential for treating diffuse large B-cell lymphoma and mantle cell lymphoma cell lines. This synergy is attributed to the CB1 antagonist activity of cannabis against diffuse large B-cell lymphoma and mantle cell lymphoma cell lines.[30,31]

Moreover, combining cannabinoids with bevacizumab (a monoclonal anti-VEGF antibody) has been shown to decrease tumor growth and intratumoral hypoxia in clinically relevant human glioblastoma models. This effect is mediated through the downregulation of HIF-1α.[32] Long-term studies evaluating the potential harmful or synergistic potential of CBD on targeted anticancer therapy are needed.

CONCLUSIONS

This exploratory study of patients receiving cancer therapy at WJVAMC found a significant prevalence of concurrent cannabis use among patients undergoing antineoplastic treatments. Given that many antineoplastic agents are metabolized by the CYP450 enzyme system, the findings of this study suggest that concurrent cannabis use may pose risks of suboptimal therapeutic outcomes due to potential interactions affecting drug metabolism. These interactions could impact the efficacy and toxicity of the antineoplastic therapies, potentially leading to diminished therapeutic effects or exacerbated adverse reactions.

Patients should be informed regarding the potential decreased efficacy of immunotherapy with concurrent use of cannabis products. They should also be aware of the possibility of increased toxicity with other treatment modalities, though the exact impact on efficacy remains unclear. This highlights the necessity of caution when combining cannabis with prescribed cancer treatments.

While this study identified possible interactions, its data are preliminary and highlight the need for more rigorous research. Future studies should include larger, well-designed cohorts to compare outcomes between cannabis users and nonusers. Such research is essential to fully elucidate the clinical implications of cannabis use during cancer treatment, address the high prevalence of cannabis use among patients with cancer, and mitigate potential risks associated with combining cannabis products with antineoplastic therapies. This will ensure that treatment strategies are optimized for safety and efficacy in this complex patient population.

Author affiliations

[a]Lt. Col. Luke Weathers Jr. Veterans Affairs Medical Center, Memphis, Tennessee
[b]Alice and Carl Kirkland Cancer Center, Jackson, Tennessee

Author disclosures

The authors report no actual or potential conflicts of interest regarding this article.

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Ethics and consent

This study was reviewed and approved by the Lt. Col. Luke

Weathers Jr. Veterans Affairs Medical Center Institutional Review Board.

References

1. Steele G, Arneson T, Zylla D. A comprehensive review of cannabis in patients with cancer: availability in the USA, general efficacy, and safety. *Curr Oncol Rep*. 2019;21:1-10. doi:10.1007/s11912-019-0757-7
2. Brown D, Watson M, Schloss J. Pharmacological evidence of medicinal cannabis in oncology: a systematic review. *Support Care Cancer*. 2019;27:3195-320. doi:10.1007/s00520-019-04774-5
3. Abrams DI. Integrating cannabis into clinical cancer care. *Curr Oncol*. 2016;23:S8-S14. doi:10.37.47/co.23.3099
4. Serafimovska T, Darkovska-Serafimovska M, Stefkov G, Arsova-Sarafinovska Z, Balkanov T. Pharmacotherapeutic considerations for use of cannabinoids to relieve symptoms of nausea and vomiting induced by chemotherapy. *Folia Medica (Plovdiv)*. 2020;62:668-678. doi:10.3897/folmed.62e51478
5. Bar-Sela G, Zalman D, Semenysty V, Ballan E. The effects of dosage-controlled cannabis capsules on cancer-related cachexia and anorexia syndrome in advanced cancer patients: pilot study. *Integr Cancer Ther*. 2019;18:1534735419881498. doi:10.1177/1534735419881498
6. Pederson ER, Villarosa-Hurlocker MC, Prince MA. Use of protective behavioral strategies among young adult veteran marijuana users. *Cannabis*. 2018;1:14-27.
7. Schilling S, Melzer R, McCabe PF. Cannabis sativa. *Curr Biol*. 2020;30:R8-R9. doi:10.1016/j.cub.2019.10.039
8. McDougle DR, Kambalyal A, Meling DD, Das A. Endocannabinoids anandamide and 2-arachidonoylglycerol are substrates for human CYP2J2 epoxygenase. *J Pharmacol Exp Ther*. 2014;351:616-627. doi:10.1124/jpet.114216598
9. Movsesyan VA, Stoica BA, Yakovlev AG, et al. Anandamide-induced cell death in primary neuronal cultures: role of calpain and caspase pathways. *Cell Death Differ*. 2004;11:1121-1132. doi:10.1038/sj.cdd.4401442
10. Cherkasova V, Wang B, Gerasymchuk M, Fiselier A, Kovalchuk O, Kovalchuk I. Use of cannabis and cannabinoids for treatment of cancer. *Cancers (Basel)*. 2022;14:5142. doi:10.3390/cancers14205142
11. Engels FK, Ten Tije AJ, Baker SD, et al. Effect of cytochrome P450 3A4 inhibition on the pharmacokinetics of docetaxel. *Clin Pharmacol Ther*. 2004;75:448-454. doi:10.1016/j.clpt.2004.01.001
12. Alsherbiny MA, Li CG. Medicinal cannabis-potential drug interactions. *Medicines (Basel)*. 2018;6:3. doi:10.3390/medicines6010003
13. Stout SM, Cimino NM. Exogenous cannabinoids as substrates, inhibitors, and inducers of human drug metabolizing enzymes: a systematic review. *Drug Metab Rev*. 2014;46:86-95. doi:10.3109/03602532.2013.849268
14. Opitz BJ, Ostroff ML, Whitman AC. The potential clinical implications and importance of drug interactions between anticancer agents and cannabidiol in patients with cancer. *J Pharm Pract*. 2020;33:506-512. doi:10.1177/0897190019828920
15. Guzmán M, Duarte MJ, Blázquez C, et al. A pilot clinical study of D9-tetrahydrocannabinol in patients with recurrent glioblastoma multiforme. *Br J Cancer*. 2006;95:197-203. doi:10.1038/sj.bjc.6603236
16. Kopjar N, Fuchs N, Brčić Karačonji I, et al. High doses of Δ^9-tetrahydrocannabinol might impair irinotecan chemotherapy: a review of potentially harmful interactions. *Clin Drug Investig*. 2020;40:775-787. doi:10.1007/s40261-020-00954-y
17. Bouquié R, Deslandes G, Mazaré H, et al. Cannabis and anticancer drugs: societal usage and expected pharmacological interactions - a review. *Fundam Clin Pharmacol*. 2018;32:462-484. doi:10.1111/fcp.12373
18. Buchtova T, Lukac D, Skrott Z, Chroma K, Bartek J, Mistrik M. Drug-drug interactions of cannabidiol with standard-of-care chemotherapeutics. *Int J Mol Sci*. 2023;24:2885. doi:10.3390/ijms24032885
19. Sharafi G, He H, Nikfarjam M. Potential use of cannabinoids for the treatment of pancreatic cancer. *J Pancreat Cancer*. 2019;5:1-7. doi:10.1089/pancan.2018.0019
20. Kosgodage US, Uysal-Onganer P, MacLatchy A, et al. Cannabidiol affects extracellular vesicle release, miR21 and miR126, and reduces prohibitin protein in glioblastoma multiforme cells. *Transl Oncol*. 2019;12:513-522. doi:10.1016/j.tranon.2018.12.004
21. Elbaz M, Nasser MW, Ravi J, et al. Modulation of the tumor microenvironment and inhibition of EGF/EGFR pathway: novel anti-tumor mechanisms of cannabidiol in breast cancer. *Mol Oncol*. 2015;9:906-919. doi:10.1016/j.molonc.2014.12.010
22. Xiong X, Chen S, Shen J, et al. Cannabis suppresses antitumor immunity by inhibiting JAK/STAT signaling in T cells through CNR2. *Signal Transduct Target Ther*. 2022;7:99. doi:10.1038/s41392-022-00918-y
23. Bar-Sela G, Cohen I, Campisi-Pinto S, et al. Cannabis consumption used by cancer patients during immunotherapy correlates with poor clinical outcome. *Cancers (Basel)*. 2020;12:2447. doi:10.3390/cancers12092447
24. Waissengrin B, Leshem Y, Taya M, et al. The use of medical cannabis concomitantly with immune checkpoint inhibitors in non-small cell lung cancer: a sigh of relief? *Eur J Cancer*. 2023;180:52-61. doi:10.1016/j.ejca.2022.11.022
25. Sarsembayeva A, Schicho R. Cannabinoids and the endocannabinoid system in immunotherapy: helpful or harmful? *Front Oncol*. 2023;13:1296906. doi:10.3389/fonc.2023.1296906
26. Kisková T, Mungenast F, Suváková M, Jäger W, Thalhammer T. Future aspects for cannabinoids in breast cancer therapy. *Int J Mol Sci*. 2019;20:1673. doi:10.3390/ijms20071673
27. Woerdenbag HJ, Olinga P, Kok EA, et al. Potential, limitations and risks of cannabis-derived products in cancer treatment. *Cancers (Basel)*. 2023;15:2119. doi:10.3390/cancers15072119
28. Almeida CF, Teixeira N, Valente MJ, Vinggaard AM, Correia-da-Silva G, Amaral C. Cannabidiol as a promising adjuvant therapy for estrogen receptor-positive breast tumors: unveiling its benefits with aromatase inhibitors. *Cancers (Basel)*. 2023;15:2517. doi:10.3390/cancers15092517
29. Dobovišek L, Novak M, Krstanović F, Borštnar S, Turnšek TL, Debeljak N. Effect of combining CBD with standard breast cancer therapeutics. *Adv Cancer Biol Metastasis*. 2022;4:100038. doi:10.1016/j.adcanc.2022.100038
30. Strong T, Rauvolfova J, Jackson E, Pham LV, Bryant J. Synergistic effect of cannabidiol with conventional chemotherapy treatment. *Blood*. 2018;132:5382. doi:10.1182/blood-2018-99-116749
31. Maggi F, Morelli MB, Tomassoni D, et al. The effects of cannabidiol via TRPV2 channel in chronic myeloid leukemia cells and its combination with imatinib. *Cancer Sci*. 2022;113:1235-1249. doi:10.1111/cas.15257
32. Obad N, Janji B, Prestegarden L, et al. ATPS-59 improving efficacy of bevacizumab treatment in glioblastoma by targeting hif1 alpha. *Neuro Oncol*. 2015;17:v31. doi:10.1093/neuonc/nov204.59