UW-MADISON SCHOOL OF PHARMACY STUDENT WRITING CLUB:

Cannabidiol (CBD) and Tetrahydrocannabinol (THC) Drug Interactions: A Narrative Review

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Abstract

Cannabidiol (CBD) and tetrahydrocannabinol (THC) are seeing an increase in use, but their potential to induce drug-drug interactions remains largely unexplored. Through pharmacokinetics and enzyme metabolism, breast cancer resistance protein (BCRP), p-glycoprotein (p-gp), and UDP-glucuronosyltransferase (UGT) interactions that impact activity can be predicted. From this narrative review, a number of cytochrome P450 (CYP) isoenzymes have been identified that increase or decrease the half-life of the affected drug, CBD or THC. BRCP, p-gp, and UGT enzymatic activities are impacted similarly. The topic of CBD/THC drug interactions is a fairly recent topic that is not well studied, and many pharmacists are unaware of how to approach patient questions when they arise. With the growing use of CBD/THC products, it is imperative that pharmacists become aware of these interactions to better advise their patient populations. The purpose of this narrative review is to identify and provide summarized guidance for clinicians to consider when encountering drug interactions between medications and CBD or THC in practice. The hope is that a pharmacist will ask patients about CBD/THC use, allowing them to make educated suggestions that a patient or provider should consider.

Cannabis is becoming more prevalent in society today, with patients increasingly seeking out alternative therapeutic opportunities that are over-the-counter (OTC) and easy to obtain. Cannabidiol (CBD) and tetrahydrocannabinol (THC) are two differing active ingredients in the cannabis family that are widely utilized. As a pharmacist, it is imperative to understand the potential drug interactions between CBD/THC and other commonly used medications to provide the best patient care. CBD or THC may have an inherent benefit in certain conditions, but their interaction potential may interfere with current medications that a patient is taking. The utilization of CBD in epilepsy is the most studied, where Epidiolex™ (Cannabidiol; Jazz Pharmaceuticals) is already FDA approved. While many drug-drug interactions are studied in clinical trials, not every medication combination can be studied. It is important to have up-to-date literature available for healthcare professionals to minimize potential adverse reactions. This narrative review will focus on delta-9-THC and CBD, two of the major and most common cannabinoids. It is important to note that the percent of active ingredients in a cannabis product varies based on the specific plant species, the product, and the ingestion method.[1] With the differing legality of cannabis products allowed per state, it is difficult to know the contents of a product that a patient may be consuming.[2] In addition, this is a limitation of many studies, as cannabis products are not yet regulated before distribution. It is worth mentioning that the prevalence of genetic variants may impact drug-drug interactions. This narrative review will include the interactions of CBD and THC with antiepileptic, psychiatric, cardiovascular, anticoagulant, analgesic, cancer, and transplant medications.

Mechanism of Interaction

THC and CBD both inhibit CYP2D6, and CBD is known to inhibit CYP2C19.[1] Additionally, CBD is extensively metabolized by the liver, specifically by the CYP3A4 enzyme. Approximately 60% of prescribed medications are also metabolized by CYP3A4. One study analyzed the in vitro effects of CBD and THC on CYP isoenzymes, concluding that CBD demonstrated the most potent activity at inhibiting CYP3A4.[3] CBD and THC have also been shown to inhibit carboxylesterase 1 (CES), which is an enzyme in the liver utilized to metabolize some medications. Medications metabolized by the enzymes CYP3A4 and CES1 will lead to an increased concentration of medication in the body. THC is a moderate inhibitor of CYP2C9, which would increase concentrations of medications metabolized by CYP2C9.

CBD is also known to inhibit UGT1A9 and UGT2B7, and this is important for medications that are metabolized by UGT. Medications that are substrates to UGT may exhibit an enhanced effect due to reduced medication metabolism.

P-gp is an efflux transporter that helps with removing foreign materials from the body.[4] The main function of p-gp is to limit the accumulation of medications in certain areas of the body by transporting them into the peripheral blood supply. If this function is increased, then there is less medication at pertinent tissues because more is being transported out of the cells, reducing the medication's effect. Studies have shown mixed results for p-gp expression dependent on CBD exposure. Chronic exposure to CBD has been found to decrease the functionality of the protein, which may lead to a higher medication concentration in the body than expected, as less medication is being transported away from tissues. In contrast, acute exposure to cannabis has been found to increase the functionality of the protein, which may lead to a lower concentration than expected. A study predicts that THC increases the function of this transporter at the blood brain barrier. The most abundant active metabolite of CBD is the inactivated hydroxylated metabolite 7-COOH-CBD.[2] This metabolite is a substrate for the p-gp transporter and an inhibitor of the BCRP. Both enzymes play a role in the efflux of various medications and have the potential to cause increased side effects and drug-drug interactions by decreasing the efflux and increasing the distribution of medication to tissues.

Epilepsy

CBD has been found to have therapeutic efficacy for the treatment of Dravet syndrome, Tuberous Sclerosis, and Lennox-Gastaut syndrome in its purified form called Epidiolex™. The interaction between CBD and the CYP2C19 enzyme is important to understand because many first-line treatments for these conditions are metabolized by this enzyme. The most prevalent medication interaction would be clobazam, with the inhibition of CYP2C19 leading to an increase in the metabolite N-desmethylclobazam.[6] Higher levels of N-desmethylclobazam have been found to cause excessive sedation. Another notable interaction would be valproate, as there is a significant risk of elevated transaminases. Elevated transaminases could be an indication of liver dysfunction; however, extensive research has not been done on long-term concomitant use of valproate and cannabinoids. A dose reduction of Epidiolex™ is recommended in patients with elevated transaminase levels or in those using medications that affect the liver such as valproate or clobazam. The short-term concomitant use of Epidiolex™ with either stiripentol or valproate has been studied with no clinically relevant changes to the concentration of either medication. Stiripentol is an antiepileptic medication metabolized by CYP2C19. Concomitant use of stiripentol and Epidiolex™ showed a slight increase of stiripentol concentrations, while concomitant use of valproate and Epidiolex™ showed no obvious changes to the concentration of valproate. Despite concomitant use of valproate and Epidiolex™ illustrating unchanged valproate levels, there is still a risk of elevated transaminases and a dose reduction of Epidiolex™ may be warranted.

Other medications that have the potential to interact with CBD, due to their similar metabolisms, that have not been extensively investigated include carbamazepine, phenytoin, felbamate, topiramate, rufinamide, zonisamide, eslicarbamazepine, perampanel, and lamotrigine. Carbamazepine and phenytoin are potent enzyme inducers of CYP3A4; therefore, one could predict that concomitant use of CBD-containing products may lead to lower concentrations of CBD.[7] Phenytoin is extensively metabolized by CYP2C19 and CYP2C9, leading to the potential for higher concentrations of both medications with concomitant use. Felbamate is another enzyme inhibitor that could lead to a higher concentration of CBD in the body. With the potential to have a significant impact on medication concentrations in the body, it is important to monitor and record the concomitant use of CBD with epilepsy medications. Epilepsy medications, in theory, increase the seizure threshold and reduce the likelihood that someone will have a seizure. Conversely, reducing the concentrations of these medications may reduce the impact on the seizure threshold. This becomes important because in Wisconsin, a patient must remain seizure free for at least 3 months or 90 days with a doctor's recommendation before they can operate a vehicle.[8] It is important not only for the patient's safety but also for others on the road to keep a patient's seizure threshold from being lowered by interacting medications.

Psychiatric

Cannabinoids may interact with the metabolism of antidepressants or anxiolytic medications, such as selective serotonin reuptake inhibitors (SSRIs). SSRIs are hepatically metabolized by CYP2D6 and CYP2C19. Anxiety and depressive disorders are among the most common disorders in teens and adolescents. THC and CBD both inhibit CYP2D6 and CYP2C19.[9] Inhibiting the metabolism of sertraline or escitalopram increases the concentration and effects of these medications present in the body. This elevated exposure to SSRI medications may enhance adverse reactions. It is difficult to quantify the impact of an adolescent or adult using CBD or THC concurrently with their antidepressant medication, especially if they are not obtaining these substances over the counter at a dispensary. While it is difficult to determine the full extent of substances that a patient is using, it is crucial to consistently collect that information from patients to appropriately adjust any affected medications There is also increased difficulty with estimating marijuana exposure due to varying absorptions with the differing routes of administration available. In one study, a group of patients taking either escitalopram or sertraline (both metabolized by CYP2C19) alone were compared to a group taking those medications concurrently with additional THC or low-dose CBD (5-15 mg/day) products. The group receiving escitalopram with concurrent THC or low-dose CBD illustrated a 35% increase in area under the curve (AUC) and a 25% increase in maximum concentration (Cmax) compared to the control group. Similarly, the sertraline group displayed an increase in AUC by 33% and Cmax increase by 26%. Additionally, this study shows a statistically significant increase in adverse effects including dizziness, fatigue, diarrhea, and cough in the intervention group with an SSRI plus concurrent THC or low-dose CBD. This study is generalized to include SSRIs that are broadly metabolized by

specifically CYP2C19, excluding any SSRIs that do not follow this pathway. This study does not directly analyze the interactions with a specific THC dose; however, given that THC also exhibits similar enzyme inhibition, it may result in similar effects. CBD can also inhibit the metabolism of monoamine oxidase inhibitors, leading to an increase in adverse reactions, as the medication would remain in the body for an extended period of time.[1] Tricyclic antidepressants are metabolized by various CYP450 enzymes, including CYP2C19 and CYP2D6. CBD inhibits CYP2D6, decreasing the amount of medication that is metabolized. This leads to an increase in medication concentration throughout the body that can cause an increase in adverse effects, including QT prolongation, anticholinergic effects, and drowsiness.

Cannabis use can be detrimental to mental health due to unknown medication interactions, where it is documented as potentially increasing the risk of a psychotic relapse.[4] It is also important to note that THC is a psychoactive component of cannabis that may confound the potential impact of drug-drug interactions. Exposure to THC may reverse the neurobehavioral effects of some antipsychotic medications. It is important to determine the impact of cannabis when taken with antipsychotic medications, partly because around 40% of patients living with schizophrenia have a history of cannabis use. Some studies illustrate that THC has the potential to lower the efficacy of antipsychotics. As mentioned previously, p-gp is an essential transporter needed to limit the accumulation of central nervous system (CNS) medications in the brain, including antipsychotics. P-gp binds antipsychotic medications and transports them from the brain tissues back into the blood. Risperidone is a substrate of p-gp and an antipsychotic medication that is generally used to treat schizophrenia. THC has been shown to enhance the expression of p-gp; an enhanced p-gp expression results in a reduction of medication concentrations in various regions of the brain, which may lead to a decrease in the efficacy of antipsychotic medications. In a study analyzing mice, mice lacking p-gp showed increased brain concentrations of risperidone compared to the wild-type mice, illustrating a strong influence of p-gp on medication concentrations. In contrast, clozapine shares a similar pharmacodynamic mechanism of action to risperidone, but it is not a p-gp substrate; therefore, we would not expect an interaction. A study in mice illustrated that clozapine did not result in reduced antipsychotic efficacy when compared to risperidone. Other antipsychotics that are not p-gp substrates are suspected not to reduce the effectiveness of antipsychotics when taken with cannabis and may present as beneficial alternatives for cannabis-using schizophrenia patients.

Cardiovascular

Cannabis can potentiate higher heart rates and blood pressure, triggering arrhythmias and a potential myocardial infarction due to stimulation of cannabinoid 1 receptors on the heart.[8] On the other hand, THC has sympathomimetic properties which increase the risk of hypertension, hypotension, syncope, and tachycardia, thus contraindicated in individuals with pre-existing heart disease.[2] Amiodarone is an antiarrhythmic medication known for its inhibitory effects on CYP isoenzyme 3A4.[9,10] CBD is extensively metabolized by CYP3A4; therefore, the inhibition of this isoenzyme causes serum levels of CBD to increase.[9] This prolonged increase can lead to a higher risk adverse effects from increased CBD exposure. Other antiarrhythmic agent interactions to be aware of would be non-dihydropyridine calcium channel blockers such as diltiazem and verapamil. These medications are CYP3A4 and p-gp inhibitors and may increase the concentration of CBD. Chronic use of CBD with these medications has the potential for a synergistic effect to inhibit p-gp leading to an increased risk of causing adverse effects. It is important to emphasize that concomitant use with antiarrhythmics can potentiate adverse effects through synergistic means.

Lipid-lowering treatments are primarily biotransformed in the liver via CYP3A4.[9] Statins that are extensively metabolized by CYP3A4 are atorvastatin, lovastatin, and simvastatin. CBD has properties that inhibit CYP3A4; therefore, it slows the metabolism of these specific statin medications, leading to elevated concentrations within the body. This then increases the risk of experiencing adverse effects, including elevated liver function tests (LFTs) and myalgias. There is also an interaction that occurs with THC and rosuvastatin and fluvastatin due to CYP2C9 inhibition. Inhibition of CYP2C9 may lead to an increase in medication concentrations as the medications will remain in the active form for an extended period of time. Additionally, the inactive hydroxylated metabolite form of CBD, 7-COOH-CBD is a substrate of p-gp and an inhibitor of the BCRP.[1] Statins are substrates of BCRP and p-gp and rely on these proteins for distribution throughout the body. There exists a potential competition for p-gp and BCRP inhibition leading to more medication in tissues and less medication being transported out of cells. An increase in medication concentrations in the tissues can lead to toxic doses and increased side-effects.

Analgesics

This narrative review focuses on oral medications to help control pain in the outpatient setting. CBD has been considered an alternative to opioids when it comes to dealing with chronic pain. Some of the common opioids used today include morphine, hydrocodone, and oxycodone. CBD binds to cannabinoid receptors within the body that have been believed to produce an analgesic effect, and THC stimulates both delta and kappa opioid receptors.[11] Due to this pharmacology, researchers have believed that CBD could be seen either as a better alternative or an adjunct to opioid therapy. A systematic review suggests that adding medical cannabis may be beneficial in improving chronic pain with low certainty of evidence that it causes similar efficacy to opioids.[12] Anywhere between 64% and 77% of people in response to surveys reported a reduction in chronic pain and long-term opioid use after starting medical cannabis. However, the use of CBD with chronic pain remains an important topic when it comes to considering the interactions that can come with concomitant medication use.[13] Morphine is a medication metabolized by the UGT2B7 isoenzyme. While the studies are not clear, there is potential that CBD shows a moderate inhibition of UGT2B7. With this evidence in mind, one can predict that there would be an increase of morphine concentration in the body, which can lead to unwanted and dangerous side effects.

Both oxycodone and hydrocodone are metabolized by the CYP3A4 and CYP2D6 enzymes into their respective metabolites. CBD has been shown to have inhibition of the CYP3A4 isoenzyme, which can lead to an increase in concentrations of hydrocodone and oxycodone.

When it comes to pain management, symptom relief, and overall health, many individuals may consider non-prescription alternatives to achieve pain control. The most commonly used OTC medications are nonsteroidal anti-inflammatory drugs (NSAIDs).[14] NSAIDs work by blocking an enzyme called cyclooxygenase, which in turn blocks prostaglandin production to relieve pain and inflammation. A decrease in efficacy and potency of NSAIDs was seen in an animal study of male mice that were chronically given delta-9 THC. The only OTC NSAID that did not illustrate a decrease in efficacy but did show a decrease in potency was diclofenac. Some of the known side effects of THC and CBD use are drowsiness and sedation. THC and CBD should be used cautiously with other OTC medications that also have a risk for sedation and drowsiness because the combination can heighten these side effects. An increase in sedation can occur if THC or CBD is given in combination with other CNS depressants. One study stated that ethanol was found to increase plasma THC levels and its effects.[15] Excessive drowsiness and sedation may interfere with a patient's activities of daily living, and this medication interaction should be considered when deciding on a medication regimen.

Anticoagulants

Anticoagulants are used to help prevent blood clots and can prevent patients from developing detrimental health conditions such as deep vein thrombosis, cardioembolic ischemic stroke, pulmonary embolism, and several more.[16] THC has shown interactions with anticoagulant medications such as warfarin. Warfarin is metabolized through CYP2C9, which is moderately inhibited by THC.[17] The inhibition of CYP2C9 would decrease the clearance of warfarin, and lead to an increase in a patient's warfarin levels in the body, ultimately resulting in a higher international normalized ratio (INR). An elevated INR can be dangerous as this indicates a longer time for a blood clot to resolve, thus resulting in an increased risk of bleeding.

We can predict that there is an additional anticoagulant interaction between CBD and the direct oral anticoagulants (DOACs). All the DOACs are substrates for the p-gp enzyme, and prolonged exposure of CBD for 72 hours has been shown to decrease p-gp expression.[1,17] This decreased expression is predicted to cause a lower elimination of the DOAC and an increase of DOAC serum plasma concentrations. Increased DOAC plasma levels may increase the risk for someone to have a bleed. In addition to this interaction, the DOACs apixaban and rivaroxaban are metabolized by CYP3A4. CBD may have a higher potential to interfere with the metabolism of these two DOACs as it is also metabolized by CYP3A4. In conclusion, taking THC/CBD with an anticoagulant has the potential to cause serious complications.

Cancer

The use of THC and CBD has elicited many therapeutic benefits as a supportive care medication for patients receiving chemotherapy. Cannabinoids have been shown to reduce the number of opioids needed to treat pain and prevent paclitaxel-induced neuropathies, and several products are FDA approved for use in chemotherapy-induced nausea and vomiting.[18] Taking CBD in combination with paclitaxel may reduce the potential for drug-induced neuropathies caused by paclitaxel. One study analyzed in vitro effects on breast cancer cell viability. This study concluded that concomitant use of paclitaxel and CBD resulted in a synergistic improvement in cell viability. CBD treatment has also been shown to reduce the incidence of oral mucositis caused by 5-fluorouracil.[19] Additionally, cannabis components affect several metabolic pathways within the body for medications commonly involved in the treatment of cancer. This includes the inhibition of CYP3A4, interactions with p-gp membranes, BCRP inhibition, multidrug resistance associated proteins (MRP) family interactions, inhibition of CYP2D6, and inhibition of CYP2C19. The concomitant use of cannabinoid derivatives with cancer medications requires clarification of the potential medication interactions.

P-gp is involved in the metabolism of medications including antimetabolites, taxanes, vinca-alkaloids, and topoisomerase inhibitors.[19] Acute exposure to CBD has been found to increase the functionality of p-gp, leading to a lower dose than expected. P-glycoprotein is involved in the metabolism of a substantial number of medications, so it is important to understand and monitor the potential effects of concomitant use of CBD during cancer treatment.[18] BCRPs are often expressed in tumor cells. An in vitro study found that CBD can inhibit the efflux of medications such as mitoxantrone and topotecan, leading to an increase in the serum concentrations of these medications. Other anticancer agents that involve BCRP and have the potential to show increased concentrations with concomitant use of CBD include methotrexate and topotecan.[19] This may lead to an increase in efficacy accompanied with an increase in adverse effects. Members of the ATP-binding cassette superfamily, such as MRPs, are also affected by CBD.[18] For example, the use of CBD with MRP1 substrates like vincristine has shown increased concentrations of these substrates. As discussed, CBD has been shown to inhibit CYP2D6. This impacts the prodrug tamoxifen and may cause a severe interaction as it proves to be less efficacious.

Transplant

After a transplant procedure, the immune system of the patient needs to be suppressed to avoid potentially fatal complications including rejection of the graft. Transplant medications are used to help prevent rejection of the graft while suppressing the immune system, making it more difficult to fight off infections. Some of the most common transplant medications include calcineurin inhibitors, most importantly tacrolimus. Tacrolimus is metabolized extensively in the liver by the CYP3A4 isozyme into its respective metabolites.[20] CBD inhibits the CYP3A4 isozyme, meaning that the concentration of tacrolimus will increase significantly. This can be problematic due to the narrow therapeutic index of tacrolimus and even a slight increase in concentration can be the difference between therapeutic efficacy and toxicity. Another staple of transplant medication regimens include mycophenolate and occasionally cyclosporine. The major hepatic enzyme needed for metabolizing mycophenolate into its inactive metabolites is CES1, and

CBD has been found to inhibit CES1.[21] Mycophenolate is metabolized to its active component by CES1 in the liver; therefore, in the presence of CBD, there may be a reduction in serum concentration of mycophenolate.

Summary

In the last two decades, cannabinoid product usage has increased greatly with the rise of product availability, despite the lack of clarity surrounding possible medication interactions. Specifically, there were about 100 patients on cannabinoids around 2001, and that number has drastically increased to over 270,000 currently.[22] With this increase in popularity, the responsibility of guiding those patients relies on the pharmacist, recognized as the medication expert, regardless of their practice setting. Cannabinoids, like all other medications, have a potential for medication interactions that patients or providers may not be aware of, and it is important for providers to remain up to date on current practices. Some of these interactions can be problematic, which can interfere with the health and safety of the patients using these products. A visual summarizing important drug-drug interactions with CBD and THC is documented in table 1. More states have started to legalize marijuana for both recreational and medicinal use, which will make cannabinoid products even more accessible. Similar to how pharmacists ask about tobacco and alcohol use to monitor for any potential medication interactions, it is important to start asking about cannabis use. This may be an uncomfortable question to ask patients for some pharmacists; however, open communication is important to prevent a patient from exposing themselves to any medication interactions with the potential to worsen disease conditions.

TABLE 1. Summary of Medication Interactions with CBD/THC Discussed in this Narrative Review

Medication	*Interaction*	*Considerations*
Clobazam	CYP2C19 inhibition (CBD)	May consider dose reduction
Phenytoin	CYP2C19 (CBD) and CYP2C9 (THC) inhibition	May consider dose reduction
SSRIs and TCAs	CYP2D6 (CBD and THC) and CYP2C19 inhibition (CBD)	May consider dose reduction
Risperidone	Enhanced p-gp expression (THC)	May consider a dose increase or a change in medication
Atorvastatin, lovastatin, and simvastatin	CYP3A4 inhibition (CBD)	May consider dose reduction; monitor for adverse effects (myalgias or elevated LFTs)
Rosuvastatin and lovastatin	CYP2C9 inhibition (THC)	May consider dose reduction; monitor for adverse effects (myalgias or elevated LFTs)
Morphine	UGT2B7 inhibition (CBD)	May consider dose reduction
Oxycodone and hydrocodone	CYP3A4 and CYP2D6 inhibition (CBD and THC)	May consider dose reduction
Warfarin	CYP2C9 inhibition (THC)	Consider avoiding; heavily monitor INR and recommend no recreational cannabis use
DOACs	Decrease p-gp expression (THC)	May consider dose reduction
Mitoxantrone, topotecan, methotrexate, and cyclophosphamide	BCRP inhibition (CBD)	May consider dose reduction
Tamoxifen	CYP2D6 inhibition (CBD and THC)	May consider dose increase; recommend no recreational cannabis use
Tacrolimus	CYP3A4 inhibition (CBD)	May consider dose reduction; increase frequency through monitoring
Mycophenolate	CES inhibition (CBD)	May consider dose increase; recommend no recreational cannabis use

**CBD = Cannabidiol, DOACs = direct oral anticoagulants, SSRI = selective serotonin reuptake inhibitor; TCA = tricyclic antidepressant, THC = tetrahydrocannabinol*

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