# Transcript: Alcohol is STILL a Drug: An Exploratory Webinar Series – November 2021

Presenter: Randall Webber
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ANN SCHENSKY: Welcome, everyone. We'll get started in a minute or so.

OK, we have a couple of people still coming in, but we will get started. Again, welcome everyone to our series Alcohol Is Still A Drug. This is our third session of the series. They will occur monthly till June except for January of 2022.

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Just a couple of housekeeping details. This presentation will be recorded and posted to our websites within a couple of weeks. Please put any questions in the Q&A pod at the bottom of your screen, and the presenter will address them at the end of the session.

If you have any technical difficulties, please individually message either Kristina Spannbauer or Stephanie Behlman. And certificates of attendance will be sent to all who attend the full session. They take about two weeks, and they'll be sent via email.

We are excited today that our presenter is Randall Webber. Randall Webber has worked in the addiction field for the past 45 years as a counselor, program director, and trainer. He has provided training on street pharmacology, addiction science, counselor ethics, and substance misuse treatment strategies.

Randall has authored or co-authored numerous publications and has held teaching positions at several colleges and universities. He serves on the board of directors at the Illinois Association of Addiction Professionals. So I'm going to turn it over to you Randall.

RANDALL WEBBER: All right, thank you very much. Let me share my screen, and then we'll get going.

OK, psychotherapy in the treatment of alcohol use disorders. We're going to look at a number of medications that are used to treat alcohol use disorder.

ANN SCHENSKY: Randall, I don't mean to interrupt you. I don't think you have it in presentation mode yet.

RANDALL WEBBER: OK.

ANN SCHENSKY: If you click-- OK, perfect. It looks good now.

RANDALL WEBBER: Is that better?

ANN SCHENSKY: Yeah.

RANDALL WEBBER: OK, great. So the two types of medication that we're going to talk about this morning are, first of all, medications that are used to facilitate alcohol withdrawal, and secondly, those that are used to reduce or stop drinking.

A little bit about the neurophysiology of alcohol withdrawal before we get started with the medications that are used to treat it. When alcohol enters the body, it's a depressant drug. We can also say it's an inhibitory drug. It tends to sedate the body. When alcohol comes in in large enough quantities regularly enough, the body adjusts in order to maintain homeostasis.

Homeostasis is a basic set of regulatory functions. Your blood sugar should be within certain levels. Your breathing should be within a certain range, your heartbeat. And when alcohol enters the body, it produces

a sedating effect. It slows down breathing, et cetera. And so inhibitory systems, the sedating systems of the body, down regulate. In other words, there are fewer sedating effects because alcohol is providing that sedation.

At the same time there's what's called upregulation of excitatory systems, the neurochemical systems that tend to stimulate the body. This is because the body is trying to fight against the sedating effects of alcohol. When alcohol use is ceased abruptly, all of these changes are unmasked. And so the person has very little sedating neurotransmitters and a lot of excitatory neurotransmitters. So naturally, they're anxious, and they have other symptoms, which we're going to take a look at.

These symptoms include craving, tremor, which can originally be the kind of tremor that would make a guy think twice about shaving. The tremor can get very course later on in withdrawal and be so severe that the person would have trouble holding a coffee cup. Anxiety, sweating, sometimes profuse sweating, insomnia, nausea. When you combine sweating and nausea and perhaps are not taking in enough liquid, you have a recipe for dehydration.

Later on, if the withdrawal gets worse, you may see rapid pulse, an increase in blood pressure, fever, DTs, delirium tremens, delusions, and vivid, frightening hallucinations, and ultimately, seizures, which may be the ultimate cause for the person's death.

Some patients with alcohol use disorder don't need medical detoxification. In fact, probably a large number of them. In working with clients over the years that I've been in the field, I found that very few needed detoxification. Most of them could stop without medication. They may be very nervous, very anxious, depressed, but they don't show any of the physical signs that would require medication.

But those people who do need medication have worse withdrawal symptoms, and they do need something to help to temper those symptoms. For more severe withdrawal, the drugs of choice are benzodiazepines. And so we're talking about various different benzodiazepines, including Klonopin, Xanax, Ativan. The ones that are preferred with for detoxification of alcohol withdrawal are those with the longer half life. A half life is the amount of time that half of the drug is still in your body. It's a measure of how long the drug lasts.

And these two neuro-trans-- I'm sorry, benzodiazepines, Librium and Valium, have longer half lives, and Librium, in particular, lasts a long time. And so compared to something like Xanax, where you have to dose the person every four hours, with equilibrium you can provide the medication every 12 hours, and there's more stability, physical stability. Phenobarbital is less commonly used. It was once a medication that was often employed but not anymore.

So once the person gets through withdrawal, then we can begin thinking about medications used to reduce or stop drinking. And these would include Disulfiram, which is commonly known as Antabuse, Naltrexone, Acamprosate, the anticonvulsants, and Baclofen.

In order to talk about Antabuse, we need to understand something about alcohol's metabolic pathway. Alcohol is broken down into acetaldehyde by the enzyme alcohol aldehyde dehydrogenase. Acid aldehyde is broken down into acetic acid by aldehyde dehydrogenase. And then from acetic acid it's converted into carbon dioxide and water and then leaves the body.

Our focus is going to be on acetaldehyde. Acetaldehyde is a very toxic substance that can produce very unpleasant effects. And so if it's not converted to acetic acid, it produces all kinds of unpleasant effects. And so Disulfiram blocks the conversion of acetaldehyde to acetic acid. It does this by inhibiting aldehyde dehydrogenase. In other words, it eliminates the enzyme that could convert acetaldehyde to acetic acid. So the person who stuck with a large, liberal acetaldehyde circulating in their body.

When this happens, the person has taken antabuse. It's inhibited aldehyde dehydrogenase. Drinking produces these aversive effects, sometimes called DER, disulfiram- ethanol reaction. The person would experience flushing, dizziness, sweating, a drop in blood pressure, which would make them feel faint, nausea and vomiting, blurred vision, confusion.

With higher doses of Antabuse and more alcohol, if the person has a high dose of Antabuse, and they drink four shots really quickly before the acid aldehyde reaction kicks in, then ultimately, within a few minutes they may experience extremely rapid or even slowed heart rate, more extreme drop in blood pressure, cardiovascular collapse, congestive failure, where the lungs are unable to rid themselves of the fluids that can accumulate there.

And then, finally, convulsions. And so using Disulfiram and drinking can be fatal, but it rarely is. Usually the person simply feels unpleasant.

I'm sorry. I was having a little trouble with my slides. Somehow we won't get off that slide.

KRISTINA SPANNBAUER: You could try stopping your share screen and restarting it, sharing again, and picking up from that slide. Maybe that will help.

RANDALL WEBBER: For heaven's sake. This is never good.

KRISTINA SPANNBAUER: If you notice when you move your mouse in the bottom left, there are also arrows that will advance. You could try clicking those. See if that works. Bottom left side.

RANDALL WEBBER: OK, so I'm sorry that we've lost some minutes. We don't have very much time, but I'll continue on. Most reactions to the Disulfiram last about 30 minutes. And so the person is miserable for about 30 minutes, and then the reaction goes away. But if they drink again, they will again get that DER response.

The efficacy of Disulfiram depends on patient compliance. This is kind of obvious. If the client does not take the Disulfiram it's not going to work. Disulfiram may limit the length of relapse because when the person relapses they get sick, and then the relapse stops. But there's a lack of methodologically sound evidence that Antabuse prevents relapse. It will minimize the length of relapse, but it doesn't seem to prevent relapse.

There are also a lack of guidelines about which psycho-social interventions are best suited to enhance compliance. And this is a good time to mention that with all these medications psycho-social interventions, counseling, et cetera, should be added. These are not stand alone medications in most cases. They're adjuncts to treatment just the way that Methadone and Buprenorphine are adjuncts to opioid treatment.

So the clients need to be monitored for optic neuritis, which is inflammation of the optic nerve, peripheral neuropathy, numbness or tingling in the hands, feet, or extremities, and hepatotoxicity, in other words, liver dysfunction. And the liver function can be monitored by simple blood tests.

OK, let's talk a little bit about Naltrexone, sometimes sold under the brand name ReVia. Naltrexone is well tolerated physically. It doesn't produce adverse effects on the liver or other organs. Alcohol detox has to be completed before Naltrexone begins to be administered. And Naltrexone is an opioid antagonist.

Wait a minute. We're talking about alcohol. Why are we throwing in an opiate antagonist? An antagonist is a drug like Naloxone, often called Narcan, that will reverse an opioid overdose or block the effect of opioids, so that if the Naltrexone is in place, the opioid molecules cannot attach to their receptor site in the brain, and sort of blocks the effect of opioids. So why are we talking about antagonists?

Well, alcohol works in part through the endogenous, the naturally occurring opioid system. This is part of the way that it produces its effect. And so when the opioid system is blocked by Naltrexone, drinking becomes less pleasurable. And the research indicates that Naltrexone can of reduce craving, drinking days, and the length of relapse compared to placebo.

There is a depot form of Naltrexone that is injected intramuscularly that works for 30 days. So this eliminates the daily administration or the person needing to take Naltrexone daily. Everybody can forget to take medication. Sometimes people conveniently forget to take medication because they don't want it to work. If you don't take Naltrexone, it doesn't work. But the depot form works for 30 days, and so the person does not have to remember to take the drug every day.

The research is telling us that Naltrexone combined with psycho-social interventions works better than Naltrexone alone, and that really kind of reiterates what I just said. Some of the common psycho-social interventions are CBT, cognitive behavioral therapy, motivational enhancement therapy, and 12-step facilitation, all of which are indicated by the National Institute on Drug Abuse as evidence-based strategies.

All right, we're going to move ahead and talk about Acamprosate. Acamprosate is an amino acid derivative. It increases GABA, the principal inhibitory or sedating neurotransmitter, and it decreases glutamate, which is the principal excitatory or speedy neurotransmitter.

So you can imagine if a person is anxious and depressed, and all of a sudden there's more of a sedating neurotransmitter and less of the transmitter that makes you anxious and shaky, that would be good. The reduction in glutamate, the reduction in this speedy neurotransmitter, is thought to be the basis for its effect on alcohol use disorder, but it also increases beta endorphins. And so that might also enhance the person's mood.

Side effects are rare. They're generally mild. They pass quickly. Diarrhea and bloating, pruritus, which is simply itching. And sometimes Acamprosate is

used with Disulfiram, and it seems to-- we seem to see better outcomes when Acamprosate is mixed with Disulfiram.

It can also be a combined with Naltrexone. And there are no contraindications, no warnings against combining these two. Studies, research studies, vary in their outcomes. And I wish I had time to talk about the research, but unfortunately we are limited in time. But there's no clear advantage to combining the two except versus placebo.

So Acamprosate works well by itself. Naltrexone works by itself. If you combine the two, there is an advantage versus placebo. But otherwise, there really is no clear advantage. So psycho-social interventions are an advantage, and we mentioned that already.

The anticonvulsants are use occasionally to treat alcohol use disorder. As we progress through these slides, we're going to get to medications that are less and less effective in treating alcohol use disorder, but they're still out there. And so I thought I would mention them.

So anticonvulsants, including Topiramate, Carbamazepine, they reduce glutamate levels. Again, reducing that kind of speedy anxiety producing neurotransmitter. Some studies, but not all, have found Carbamazepine superior to placebo in reducing time to first heavy drinking day, drinks per drinking day, and the number of consecutive drinking days. So there is a decrease in the amount of time until the person starts drinking heavily. And then once they start drinking, there's a decrease in the number of drinks and the number of days consecutively that they drink.

Similar results with other anticonvulsants, such as Topiramate. Now these medications are not FDA approved for the treatment of AUD, and so they're used, as they say, off label.

Baclofen. Baclofen is a muscle relaxant or anti spasmodic. It's often used to treat multiple sclerosis. It acts as a GABA-B agonist, in other words, it stimulates GABA production. But it works at a particular sub receptors. Neurotransmitter receptors have sub receptors. So we have GABA-A and the GABA-A2 receptor is called the benzodiazepine receptor and seems to be responsible for the action of that particular group of drugs.

This is GABA-B, and it doesn't have quite the ability to produce GABA that the other GABA agonists do. It's got a good safety profile for people with liver disease, which is always good because so many people who drink heavily have hepatotis, hepatitis, or even cirrhosis.

So inconsistent research result, that's why it's not used as often. There may be a reduction in-- or increase in the time to relapse, more days occur before relapse, decrease in alcohol intake, and abstinence. These are all outcomes that have been reported in the literature, but there isn't really good research to back them up. In other words, the research is inconsistent and shows kind of

it does work, it doesn't work. And it also is not FDA approved for the treatment of AUD.

Well, let's talk about co-occurring psychiatric disorders. There is such a thing as subacute withdrawal. We can call it alcohol post-acute withdrawal syndrome. It occurs with many drugs. In the case of alcohol, you would see, perhaps, anxiety and depression. Anxiety, even though depression can be severe in some cases, we're not talking about severe depression here, but we are talking about a fair level of anxiety. And of course, this is very unpleasant.

In treating co-occurring psychiatric disorders, clinicians have to differentiate between subacute withdrawal and diagnosable psych disorders. So let's say that a person has generalized anxiety disorder, and they drink in order to manage their anxiety. When they stop drinking, they become anxious. Now is this a subacute withdrawal symptom, or is this a return of the original symptoms for which the person began using alcohol?

Sometimes it's difficult to tell, and a seasoned clinician can tell the difference between these two. Co-occurring psychiatric disorders have been treated with antidepressants, lithium, neuroleptics, and benzodiazepines.

Doctors have to be careful in prescribing to make sure that they avoid contraindications, bad reactions between drugs. In other words potentially bad cross reactions between different drugs, and also, benzodiazepine use disorder. Benzodiazepines and alcohol have much of the same effect. Neurochemically they work in slightly different ways, but the ultimate effect is about the same. And so benzodiazepines may be considered to be alcohol in a pill.

So what we're saying is that if a person has an alcohol use disorder, it's very easy for them to develop benzodiazepine use disorder if they are prescribed that drug. And so, we have to watch out for this secondary addiction.

Well, we'll see if there are any questions, but otherwise, I appreciate your being here today. I will post my PowerPoint slides on my website, RandallWebber.com. Just look for resources, and then workshop downloads, and you can download the slides for yourself.

OK, let me turn it back over then.

ANN SCHENSKY: Thank you so much. We do not currently have any questions in the Q&A. Also, I just wanted to let people know that we will post the slides on our website, along with the recording of this presentation. So if anyone has any quick questions, you can feel free to just put them in the chat very quickly. Otherwise, I would like to thank you all for your time, and especially you, Mr. Webber, for your time this morning and your expertise.

RANDALL WEBBER: You're very welcome.