

Table of Contents

- About Drugs
- The Synapse
- The Serotonin-Melatonin Pathway
- Serotonin-Melatonin Issues
- SSRI's: Selective Serotonin Reuptake Inhibitors
- Serotonin-Norepinephrine Reuptake Inhibitors
- Dopamine-Norepinephrine-Epinephrine
- Stimulants Effects on Dopamine-Norepinephrine-Epinephrine
- Stimulant Withdrawal Issues
- Antipsychotics Effects on Dopamine-Norepinephrine-Epinephrine
- Antipsychotic Withdrawal Issues
- Catecholamine Methyl Transferase (COMT)
- Reactive Hypoglycemia
- Endorphin-Opiate Connection
- Endorphin Deficiency
- Addressing Endorphin Deficiency
- GABA Pathway and Benzodiazepines
- GABA Pathway and Benzodiazepine Issues
- Alcohol
- Anxiety Related Alcohol Addicted
- Hypoglycemic Alcohol Addicted
- Allergy Alcohol Addicted
- Endorphin Deficient Alcohol Addicted
- Addressing Alcohol Issues
- Closing Comments
- Disclaimer

About Drugs...

Almost all drugs act by poisoning one or more of the body's functions in order to achieve a desired effect. This includes recreational drugs, alcohol, and medications. Most drugs also impact the liver detoxification systems (the CYP Enzymes) in a way that indicates the extent to which they are poisons. In this eBook, we are going to learn about:

- Neurochemical pathways
- Some theories on how drugs may work
- Nutritive ways to assist deficiencies and withdrawal

The Synapse

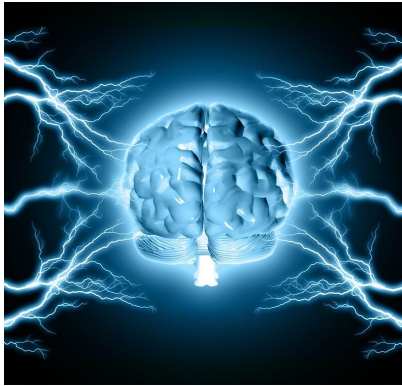
Nerve transmission occurs in the following way: The originating nerve "talks" to the receptive nerve across a synapse. The synapses are breaks in the chain of nerves where chemicals can flow from one neuron to another. Chemically, there is an opportunity to either increase or decrease the intensity of the nerve impulse at the synapse. Some chemicals inhibit the intensity of a nerve impulse while others stimulate the impulse to have greater intensity.

Neurotransmitters are chemicals which are stored in the originating nerve in an area called vesicles. A nerve transmission comes down the nerve and can cause a release of the neurotransmitter from these vesicles. The neurotransmitter flows across the synapse. The receptors on the other side of the synapse are activated by the neurotransmitter and then transmit that information to the next neuron. After the neurotransmitter activates the receptors, it is recycled back into the vesicles to be used again. This recycling process is very important to the sustainability of the neurochemicals and without it the vesicles become empty.

The Serotonin-Melatonin Pathway

Serotonin is manufactured from the base amino acid tryptophan. Tryptophan is able to cross the blood-brain barrier and then convert into 5HTP, which is then converted into serotonin. Serotonin can further be converted into melatonin. Usually, when people try to boost their serotonin levels naturally, they either take tryptophan or 5HTP to do so. Niacin is also helpful to assist in this pathway as tryptophan and 5HTP can degrade into niacin if people do not have enough niacin in the first place.

Serotonin is popularized as the body's natural antidepressant; people are often under the impression that it is serotonin that gives them an emotional lift. That is not true. Dopamine is what provides an uplifting sensation. Serotonin is better framed as an anti-rumination neurochemical. People with low serotonin typically present as anxious, impulsive, and obsessively ruminate. And since serotonin is the precursor to melatonin, they also tend to have trouble sleeping.



Serotonin modifies the impulsive reactionary thoughts of the brainstem and amygdala. The brain stem has a similar capacity to that of a reptile brain: Reptiles kill, devour, and breed. Serotonin assists in modifying those impulsive processes. So instead of killing a person who thinks differently than us, our reasoning ability, with the help of serotonin, modifies this impulse to diplomatically figure out how that person is different from us and possibly seek some common ground. Rather than devouring a chocolate cake, we may savor it a bit as we share it with a friend. And, rather than forcing copulation, we may court a person we are interested in instead.

Sugar and alcohol are particularly toxic to serotonin metabolism. You may notice that people who consume lots of sugar or alcohol tend to be impulsive. Coffee and caffeine trigger a dump of sugar stored in the liver, which has the same effect. Sugar, alcohol and low-quality carbohydrates and caffeine often contribute to serotonin deficiency. A study was conducted in California prisons by Stephen Schoenthaler with the help of then Governor Arnold Schwarzenegger, regarding sugar. It found that restricting sugar significantly decreased incidences of violence.

Melatonin, which is our sleep neurohormone, is created from serotonin inside the brain. You cannot take melatonin and have it cross the blood brain barrier and get the melatonin into the brain. Melatonin has to be broken down into tryptophan, and then builds itself back up to melatonin inside the brain. If a person takes melatonin and gets some benefit, this is a good sign; it means the pathway is at least functioning somewhat. If not, then it is very possible that some toxic impairment is occurring that will have to be addressed. This pathway is also subject to toxic impairment from mercury, which we discuss in our YouTube video entitled Neurotoxicity. Link: <https://youtu.be/Q-7yyYxG9hs>

When a pathway is blocked, taking supplements is generally not so effective. There are also genetic factors that can increase or decrease serotonin-melatonin production. Exposure to a chronic stressful environment will deplete serotonin. Chronic pain such as from fibromyalgia or other ailments can also act as a stressor that negatively impacts serotonin.

Serotonin-Melatonin issues present as:

Classic tell-tale signs:

Ruminating thoughts
Impulsivity
Insomnia

Associated issues, but may have other causes:

Carb and sugar cravings
Anxiety

Some Ways To Address Serotonin-Melatonin issues¹:

Supplement:

100-500 mg niacin, 500-1500 mg Tryptophan 2x/day, and possibly 3-6 mg Melatonin.

Toxic Impairment:

Address any toxic burden, specifically mercury amalgams in teeth.

Diet:

Consume high-protein foods; eliminate sugar, alcohol and simple carbs.

Environment:

Decrease exposure to stress

SSRI's: Selective Serotonin Reuptake Inhibitors

Serotonin is an inhibitory neurochemical. It lessens the intensity of a nerve impulse. It helps us dampen thoughts that may become compulsions without this dampening. It can also dampen a pain impulse, which is not its primary function but may explain why certain antidepressants are often used off-label for pain management. Inhibitory neurochemicals have the ability to overlap each other and can perform similar functions. When a person is deficient in one inhibitory neurochemical, they are typically deficient in all of them because the other ones have likely been used up trying to compensate for the original deficiency. These individuals often lack the ability to turn an intense impulse into a milder one.



When Serotonin is called upon, it comes out from the vesicles, is released into the synapse, and inhibits the nerve impulse. It is then broken down by the serotoninase enzyme so it can be reuptaken back into the vesicles and used again later.

Selective Serotonin Reuptake type antidepressants, which we will refer to as SSRI's, are generally not what you would regard as mood elevators. They are more implicated in reducing the probability of someone following through on suicidal ideation. Increasing serotonin may reduce the impulsivity associated with hurting oneself or others.

SSRI's could give someone a break from certain symptoms like impulsivity, anxiety and sleep issues. Unfortunately, like most medications, the effect may not be sustainable as these types of drugs are not feeding the nervous system more neurotransmitters, they are spending them. And when the nervous system is completely spent, the problem could become substantially worse. SSRI's have even been known to increase suicidality, which is what generated public outcry forcing manufacturers to include a black box warning.

SSRI's are thought to work by poisoning the serotoninase enzyme. The serotoninase enzyme is what allows the serotonin to be recycled. This forces the serotonin to stay in the synapse and continue to exert an inhibitory effect. If the serotonin is not reuptaken as it should be, over time it is probable that it then breaks down into its metabolites to the point where there are none left and the vesicles are exhausted. When there is no serotonin left to reuptake, a reuptake inhibitor would, in theory, be useless.

Tryptophan is the serotonin precursor. We use it in conjunction with a SSRI taper. The use of both an antidepressant and tryptophan is meant only for someone who is actively reducing their antidepressant. The sustained combination of both could produce a rare, but potentially fatal disorder called Serotonin Syndrome. For that reason, unless you are actively tapering your SSRI, do not use both in combination. For withdrawal, it is helpful to build up serotonin as the medication withdrawal is occurring to lessen the withdrawal side effects.

One possible symptom of antidepressant withdrawal is known as *brain zaps*. These feel like electrical discharges in the head and sometimes the body. The recommendation for someone experiencing these is to slow the taper and work on building up serotonin reserves. Brain zaps are said to not be damaging, but they are certainly not a *good* sign either.

Serotonin-Norepinephrine Reuptake Inhibitors (SNRI's)

These medications, in theory, reuptake serotonin and norepinephrine. Norepinephrine is an excitatory neurochemical that is closely associated with dopamine. Dopamine and

norepinephrine are more associated with what one thinks of mood elevation and antidepressants.

Norepinephrine flows into the synapse and increases the intensity of a nerve impulse. It is then reuptaken naturally to be used again. Norepinephrine is associated with memory and the ability to focus on a task. Low norepinephrine could result in a lethargic depression, an inability to find reward in life, and attention problems that such as difficulty focusing on tasks like reading and comprehension. Excessive norepinephrine most often presents as anxiety. We will discuss more about norepinephrine when we talk about dopamine.

SNRI's are meant to increase the availability of serotonin and norepinephrine. Theoretically, they would lower someone's impulsivity and help stimulate that individual out of depression. That sounds wonderful. Unfortunately, this intended effect, again for reasons mentioned earlier, may not be sustainable. Also, for someone that is anxious, an SNRI can markedly increase anxiety.

Antidepressants have varying half-lives, with Effexor being the shortest at less than one day, and Prozac the longest at possibly 5 days or more. The height of the withdrawal tends to hit right after the half-life. Antidepressants with a shorter half-life are generally more problematic if someone were to go cold-turkey, as they may leave the system at a rate faster than the person can adjust to. On the average, a person can expect withdrawal manifestations from an antidepressant to occur within 24 hours to 5 days after the taper and generally speaking, around day two or three. Once someone surmounts their first taper, whatever pattern of symptoms they had, the timing and onset of those symptoms and then the relief, will be similar to what one can expect on each taper. This withdrawal pattern is consistent for all medications with the special exception being antipsychotics, which we will explain in detail later.

Dopamine - Norepinephrine - Epinephrine

The Dopamine - Norepinephrine - Epinephrine pathway is also called the catecholamine pathway. The amino acid precursors to this pathway are DLPA and tyrosine. Using DLPA is a bit of a softer precursor, whereas tyrosine is more direct.

Dopamine is the primary neurochemical of reward. It is what helps us seek a "job well done", a rewarding, intimate relationship, and creative thought. Too little dopamine would leave a person feeling flat and lethargic, with little perception of life's rewards. Too much dopamine may make a person feel manic.



Norepinephrine is what allows us to focus and to learn. Too little means a poor attention span, and too much means anxiety.

Stimulants Effect on Dopamine - Norepinephrine - Epinephrine

Persons with too low of a perception of reward, or an inability to focus, may have an addictive biochemistry that craves stimulants. Stimulants include amphetamines, cocaine, coffee, sugar, ADHD medications, and on some level SNRI's like Effexor, Wellbutrin and Cymbalta. These stimulants are generally not sustainable in their action. For instance, cocaine certainly increases a person's perception of reward to the point where everything becomes rewarding, but after continued use, there is no amount of cocaine, money, fast cars, or reward of sex that can produce the same feeling.

Here is how that works: Normally, when dopamine is released, it takes something that we have associated reward with and amplifies its attractiveness. So it increases stimulation around that object or feeling. After the stimulus has been transmitted, the dopamine transport system breaks down the dopamine from the synapse and delivers it back to the vesicles so that it can be used again. This recycling is the normal process. Genetic and even behavioral conditioning or trauma can alter the way this process works. Some people produce more dopamine, some people break it down differently, some people find reward in the smallest things and some people eclipse their desire for reward with fear.

When you introduce a stimulant at the synapse, it blocks the dopamine transport system, which is the reuptake mechanism for dopamine. The dopamine stays in the synapse and continues to exert a stimulating response, which certainly feels rewarding, for a while.

As with serotonin, when the dopamine is forced to stay out in the synapse, it degrades into its metabolites. Then, there is no dopamine left for the stimulant to use and the stimulant becomes ineffective.

Additionally, there is another mechanism called *down regulation*. Down regulation is where the receptors become less responsive and it takes more and more of the drug to exert an effect. In the case of dopamine, the dopamine receptors become dampened in their ability to transmit the perception of reward. The logical fix is to feed the nervous system what it needs, not to spend what it doesn't have.

To Address Stimulant Withdrawal Issues:

Supplement: 500 - 1500 mg Tyrosine, or if mild anxiety is present, then begin with 500-1500 mg DLPA and 50 mg of B6. If extreme anxiety exists, wait until the anxiety passes. Do not ever use this supplement regimen during an antipsychotic or benzo withdrawal.

Toxic Impairment: Check for thyroid issues. Stop smoking.

Diet: Eliminate sugar and caffeine. Pay special attention to removing allergen foods.

Environment: Increase physical activity. Meditation practices. Work on being okay with being “in the down”.

Typically, ADHD medications or recreational stimulant withdrawal can be done abruptly. Occasionally, if extreme depression is a prominent feature for someone using ADHD medications, then tapering is appropriate.

Antipsychotic Effects on Dopamine - Norepinephrine - Epinephrine

Antipsychotic medications often work by holding back dopamine. Remember, too much dopamine can result in mania. Blocking dopamine with an antipsychotic is an all too effective way to bring someone out of mania and psychosis. However it also limits the person's ability to access the normal perception of reward.

This is what an antipsychotic looks like at the synapse: Dopamine is normally released from the vesicles into the synapse and affects the receptors so as to produce stimulation. In the presence of an antipsychotic, less dopamine is able to reach the receptors and so therefore very little stimulation is happening, so the perception of reward is muted.

Now we get to a concept very much like the down regulation we see with stimulants, but in this case what happens is known as *up regulation*. The tendency of the nervous system to up or down regulate is collectively referred to as neuroplasticity.

In the case of antipsychotics, what tends to happen is that the dopamine receptors up regulate. So in effect, the synapse builds *more* dopamine receptors. In this way, the little bit of dopamine that gets thru has a higher chance of exciting something. And so in this way, the drugs tend to become less effective, and the person needs more and more of the drug to combat the original symptoms.

With this up regulation happening in the synapse, getting off an antipsychotic can be very difficult and needs to be handled with extra caution. Even if the person was misdiagnosed, they can still have a challenging time getting off of these drugs.

This is why: What do you think will happen when you start reducing the drug and when you start getting a more normal amount of dopamine hitting an excessive number of dopamine receptors? The result is that you will often get a rather sudden escalation of mania or psychosis. Oftentimes when this happens, conventionally we think, “Well of course, look at what happens when they come off the drug. They need this drug!” Thus the person becomes their diagnosis forever. Most likely what is happening is that the nervous system just needs time to re-regulate itself and that if the person goes slow enough, they can adapt. A person coming off antipsychotics too fast often reports lack of sleep, failure to eat, and acting tangentially. This can be tough because once a person gets into these states, they may be resistant to slowing things down and you may lose them. This is largely what makes antipsychotics the most difficult drug class to withdraw from.



To Address Antipsychotic Withdrawal Issues

Supplements: Niacin 1000-5000 mg daily. Always include approximately twice as much Vitamin C as Niacin. Use BH4 and phosphatidylserine to help convert dopamine.

Toxic Impairment: Stop smoking if possible, but at least limit it. Sudden onset of chain smoking is not a good sign and can mean the person is starting to spin out. Do not smoke marijuana or do any recreational drugs, especially stimulants or hallucinogens during this time.

Diet: Eliminate sugar and caffeine. Seriously, even a cup of coffee during the withdrawal can land a person in the hospital. What was tolerated before on meds as far as stimulants almost certainly cannot be tolerated during the withdrawal. Understand this well. What habits a person may have had *on* antipsychotics are not going to serve them while coming off. Eat protein based foods and eat 3 times per day. The proteins help ground a person and control blood sugar swings at this time. Vegan, and vegetarians may have a bit of a rough go at this. If vegan it's important to include heavy vegan proteins and limit light foods like fruits.

Environment: Increase physical activity, specifically cardiovascular. Gym membership, regular yoga classes, jogging; these kinds of activities are going to help redirect all of that energy. If a person is still lethargic from the drugs, start with walking and then move up to something more demanding. Do anything to redirect the attention out into the world; physical activity, hikes, manual labor, group activities. It is best to limit reading, especially religious material just during this time. The concepts of god and religion during an antipsychotic withdrawal can fuel psychosis. Even limiting meditation practices during this time is advised. The idea is to get out of the head and become externalized into the environment as much as possible.

Additionally: Allow one month of withdrawal for every 1 year on antipsychotic medication if psychosis is a feature. This general rule is specific to long term users of antipsychotics and have psychosis a feature. If this is the case, the suggestion is to withdraw as much as is practical during the stay at ATMC, to find the lowest possible dose. Continue at that lowest possible dose for a period of time at home from 3 months to a year to make sure life's feelings are getting integrated. Consider Depakote as a buffer. Depakote can help soften the withdrawal. Depakote can be used throughout the duration of the antipsychotic withdrawal. Once the antipsychotic withdrawal is over, the depakote withdrawal is almost certainly much easier comparatively. If an antipsychotic was introduced to help with sleep or for a reason other than psychosis, and is at low dose, then a person can generally come off faster and without the use of Depakote.

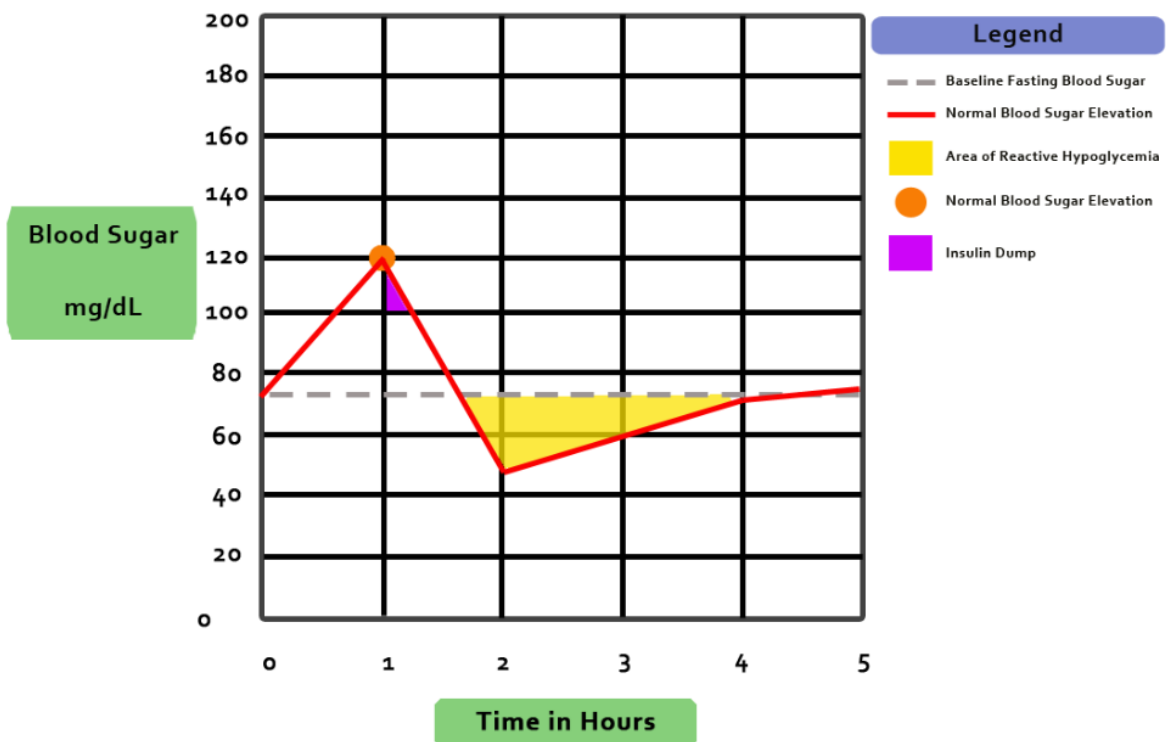
COMT

COMT stands for catecholamine methyl transferase. COMT helps break down dopamine, and to some degree is involved in the process of making it. Much of the population has a genetic polymorphism that makes it difficult to make and then break down dopamine. It manifests as a difficulty in finding a reward-level of dopamine and then when they do get it, they have a hard time breaking it down, and can turn manic. Sounds like bipolar right? Well, COMT

impairments are often found in persons who crave stimulation and therefore stimulants, and people who have been diagnosed bipolar and schizoaffective.

Reactive Hypoglycemia

What looks like bipolar, schizoaffective, or a mood disorder can also be attributed to blood sugar issues. There is a type of hypoglycemia that often defies detection, known as reactive hypoglycemia.



Let's look at a graph to describe this phenomenon. The vertical line is the blood sugar level and the horizontal line is the number of hours. At the beginning we see a normal, "fasting" blood sugar level present upon waking. Most people think that this fasting blood sugar is how you determine hypoglycemia. Rarely do you find hypoglycemia on a fasting level. The real test is a glucose tolerance test where a person drinks a thick sugary liquid and the blood sugar is plotted over a 5 hour period.

When a reactive hypoglycemic eats a sugary meal, the blood sugar rises, as it should. Then, an overwhelming amount of insulin is released by the pancreas. The excessive insulin release actually drops the blood sugar level down below the fasting level, then the person becomes hypoglycemic for a few hours and then comes back to the fasting level.

This situation would be better named hyperinsulinism as that is what is occurring. Keep in mind, coffee can set up this same mechanism. As this situation progresses, it often turns to a diabetic condition. In the early days of ATMC, we did 200 glucose tolerance tests on our population and found that 70% were reactive hypoglycemic. You could reasonably assume that 70% of mental health hospital admits have, at least in part, blood sugar issues as a component. Sugar acts like a drug in your system as should be used sparingly.

Endorphin - Opiate Connection

Endorphins are the body's natural pain-killers. Our bodies can produce what is essentially morphine. Opiate drugs take the place of this natural neurochemistry, and long term use of opiates will shut off the natural production. Then when a person comes off of the opiates, there is nothing to block pain and even the slightest touch, or even digestion, can register as *extreme* pain. This is one reason why these drugs are so addictive. Because coming off of them hurts.

This is how endorphins work at the synapse: A pain impulse come down the pipeline, and endorphins are released to soften the amount of pain that is felt. The stimulus could be something as benign as a soft touch to the arm. Without endorphins, this touch might register as pain. Opiates do the same thing as endorphins. What happens however is that the receptors, over time, down regulate to the drug because the body perceives pain as its friend, saving the body from damage. It then takes more and more of the drug in order to achieve a pain limiting response.

The way to relieve this conundrum is twofold. We taper the amount of the drug while building up the person's neurochemistry with endorphin precursors. This makes it so that the neurochemistry can adapt to the shift and the person is relieved from experiencing ridiculous amounts of pain. With this preloading of endorphin building amino acids the tapering process can usually be achieved in about two to four weeks. Suboxone is sometimes used as an intermediary.

Chronic pain situations can also exhaust endorphins as the body is trying to continually keep up with a dampening pain response. Allergen foods can also exhaust endorphins. When an allergic response is triggered, there is inflammation. The body tries to dampen the pain from the inflammation using endorphins. People can get addicted to allergen foods because they get addicted to the endorphin release. This is termed *allergic addicted*. People who have exhausted their endorphins will often crave opiates and inflammatory foods.

Endorphins also block emotional pain, or specifically the amount of emotion we pick up from other people. Our emotional brain is called our limbic brain. All mammals have this limbic brain, including our dogs and cats. It is normal and natural to be able to sense someone's

emotions from a distance. It is this ability that allows us to avoid threats, and be drawn toward bonding experiences that increase our chances of survival. We can tell if the person in the car next to us has road rage, and that can trigger an anger response in us. We have a whole different feeling if they are attracted to us and are trying to get our phone number. We can feel if a person is anxious, bored, excited, sad and mad. If someone is low in endorphins, they tend to “overfeel” people, which can be uncomfortable.



Low endorphin people can feel others more. It can be a wonderful gift in many ways. Some of our best artists are geared this way. They can feel the emotions of others and can resonate with them in amazing ways. In the case of some of our most famous celebrities, the continual feeling of everyone putting attention on them can grind them down, spending what little endorphins they have. Often, these folks then turn to pain-killers and sedatives to cope, as in the cases of Michael Jackson and Whitney Houston. The world can be unkind to these gifted beings. They are exactly the kinds of people who have the most difficulty managing all of the attention.

Extreme emotional trauma, especially prolonged threats, can burn out endorphins too. When someone is in a war situation, or is at threat of being traumatized, it is natural for the neurochemistry to want to perceive threats faster so as to avoid getting killed. So an adaptive mechanism to ongoing threats is to lower the endorphins so that a person can feel what is going on around them more. The lowered endorphins allow for this, but over time, a person begins to feel threats even when there is none. This is a possible contributing mechanism behind PTSD. PTSD has been shown at our clinic to respond favorably to endorphin repair.

There is a genetic disposition for low endorphins. Those who have the A1 allele on the D2 dopamine receptor of the 11th chromosome do not transmit endorphins as well. So basically, they are low endorphin and may have an addictive biochemistry that drives them toward

opiates. Cutting is also a sign of low endorphins as the pain causes a release of endorphins and helps provide an emotional distance from whatever the person is feeling. The hallmark of low endorphins is extreme emotional sensitivity, specifically the inability to block others' emotions. A diet low in protein and high in sugar can also debilitate someone into a low endorphin state, as can emotional trauma. High arsenic levels have also appeared very frequently in those with pain and sensitivity issues.

Classic Tell-Tale Signs of Endorphin Deficiency:

- Over-feeling others' emotions to the point of frequent discomfort.
- Chronic pain
- Cutting
- Opiate addiction

To Address Endorphin Deficient Issues:

Supplements: Endorphin PAKS by NuPathways. NAC.

Toxic Impairment: Clear up Arsenic using NAC. Stop smoking if possible. Cigarettes contain arsenic. Persons smoking are *3-4 times as likely to relapse on opiates* according to the National Institute on Drug Abuse.

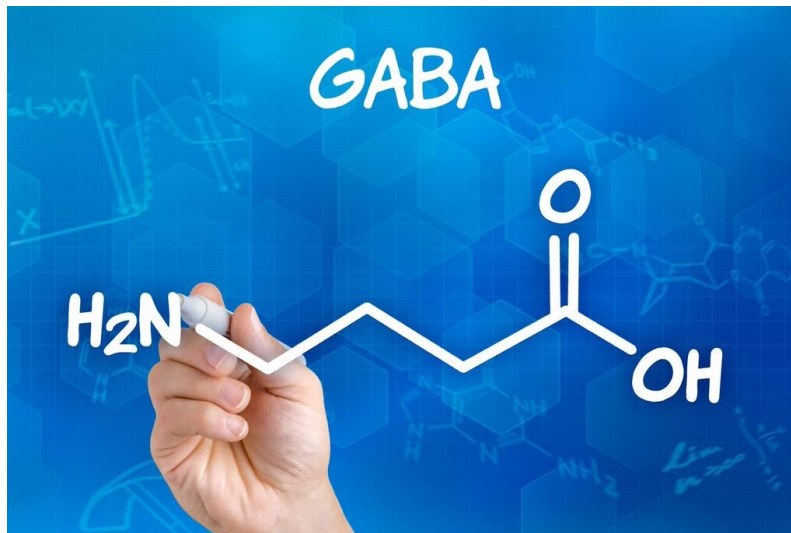
Diet: Eliminate sugar and caffeine. Include protein-based foods. Sugar poisons endorphins and proteins build them. If vegan, include the heaviest vegan proteins that you can and limit light foods like fruits. Remove foods that the person shows an addictive tendency toward.

Environment: Increase physical activity, specifically weightlifting. Muscle building exercises break muscle tissue in order to expand. This releases endorphins which is a good thing. Remove oneself from emotionally charged living situations or work places. Repeated insults to one's emotional stability can thwart endorphin building efforts.

If coming off of opiates: Consider switching to Suboxone. Induct Suboxone to a stabilizing level, and then taper off of the Suboxone over the course of two weeks. Otherwise, a person can taper the existing opiate while building up endorphins using the Endorphin PAKS. If on Methadone, the best cessation plan is to obtain a pain diagnosis and then be put on the equivalent of a short-acting opiate like Oxycodone for 3 days. After those 3 days, the person can then convert to Suboxone and taper off over a two-week period.

Dark chocolate also has an effect on endorphins. Chocolate contains phenethylamine, also known as (PEA). PEA is a brain chemical that is released when you feel like you are falling in love. PEA triggers a release of endorphins.

There are also two alcoholic types that are associated with endorphin deficiency that we will cover near the end of this eBook.



GABA Pathway and Benzodiazepines

GABA is the main inhibitory neurochemical of the central nervous system. GABA is the main counterbalance for the excitatory neurochemical called glutamate. When someone is low in GABA, they have a difficult time dampening stimulation and will tend to be anxious and may even demonstrate seizures. GABA is basically the brakes of the nervous system.

We will use the synapse as an example. When an excitatory impulse comes down the originating nerve, the synapse has the opportunity to ramp up the impulse, or decrease it. If GABA is released, the impulse is lessened.

Benzodiazepines are thought to do two things as sedatives. They make the synapse more permeable to GABA, so there is a much greater inhibitory effect. They also diminish the amount of calcium that can flow into synaptic areas. Calcium assists in muscle contraction, and having less calcium involved is part of why benzodiazepines act as a muscle relaxant.

Of course over time we experience down regulation. The receptors become less receptive to GABA, and therefore it takes more and more of the drug to achieve the same effect. Then, when a person is coming off of the drug, they are getting less GABA, which will register as

anxiety and typically gut issues. Additionally, the person coming off of benzos will get an influx of Calcium ions, which will trigger muscle contractions. This is the likely mechanism behind seizures when the withdrawal is too rapid.

Some people get put on a benzo because of a challenging or traumatic life situation. When the life situation has passed, they taper the drug, go through withdrawal to varying degrees and then get thru it. Other people seem to suffer indefinitely once the drug is discontinued and get into a situation often termed protracted withdrawal. There is some theory around damaged benzo receptors and that the person is experiencing ongoing suffering as these receptors go thru the process of repair. This is a plausible and viable theory. However we at The Alternative to Meds Center pose another possible mechanism. That being the role of toxicity.

Some toxins act as excitotoxins, as seen in the grasshopper portion of our Neurotoxicity video on ATMC's YouTube channel. These types of toxins dysregulate the neurochemical balance and then a person can become symptomatic. A person who is poisoned with excitotoxins generally presents with a reasonably constant type of anxiety that does not generally come and go. It is like a background roar in the nervous system. An analogy might be a game of catch. If the pitcher throws a soft pitch, a person can catch it with their bare hand. This would be analogous to normal interaction in the nervous system. If the pitcher pitches a 100-mile per hour fastball, very soon you are going to need a glove. The 100-mph fastball would represent the excitotoxic nervous system and the glove, a benzodiazepine. If a person is excitotoxic, then tapering the drug is like thinning the glove. At some point, the fastballs are going to start hurting.

Our approach is to get the pitcher to pitch a slower ball by removing the excitotoxic burden. We are also feeding the nervous system what it needs for receptor repair in the event of damaged receptors. This approach works very successfully in our population and we have helped many people with protracted withdrawal recover in much shorter times than typically considered the norm. Unfortunately for some that have been tapering at home for a long duration, and are suffering unreasonably, the best recommendation is to go back up to a stabilizing level of the benzo.



This is often a difficult conversation to have with someone. The rationale is that if the person is so debilitated and sleepless that they cannot engage in our program, then they are not

going to be able to experience true relief, as they will be unable to address the toxic burden. A person who has lowered their benzos to the point of pain and physical distress is often the condition we find people in when they are seeking help. They are sort of painted into a corner. Fortunately, once they raise back up to a stabilizing dose and engage in the program, they get the benefit of unburdening themselves of their neurotoxic burden. When it comes time to eliminate the meds, they can do so at a *much* faster rate with *far* fewer complications.

We have tried a lot of methods, including the Ashton Method, which involves converting to Valium as it is longer acting and is technically easier to taper from. We have found in most cases that it is better taper off of the med that the person is accustomed to. Remember, it will be easier after the toxic burden is lowered. You want to spread out the dosing so that the person is not experiencing interdosing withdrawal. For instance, if a person is taking Ativan at night, since Ativan has a short half-life, they may experience withdrawal during the morning or day. It is best to spread the medication out two or more times a day to avoid interdosing withdrawal.

By anyone's standard, even a slow taper of a benzo can be difficult. Usually, the withdrawal from a benzo medication cut begins on about the second day and may last till the third to fifth day. It is typically accompanied by anxiety, fear, insomnia and gut issues. If the acute withdrawal lasts longer than this, there is likely underlying neurotoxic poisoning that needs to be cleared up. There is a bridge medication that is considered safe called Gabapentin. Basically Gabapentin is the naturally occurring GABA attached to a pentane molecule. It is essentially a prescription vitamin, and is definitely a lesser evil than a benzo. Gabapentin does not use liver enzymes to be broken down, which means the body is not registering it as a toxin. It can give the person just enough of a buffer to endure the benzo withdrawal. Once the person withdraws from the benzo, the Gabapentin can be tapered much, much easier. Gabapentin has a very short half-life in the body and needs to be taken throughout the day to support the withdrawal. Since it has such a short half-life, Gabapentin is not effective at giving someone a full night's sleep. For sleep, a person may consider Vistiril which is an antihistamine or Trazodone which is an older tricyclic antidepressant. These meds are just for the purpose of softening the withdrawal and are lifted once the withdrawal is complete.

The bridge medications are optional. A non-prescription activated GABA product that is effectually similar to Gabapentin is called PheniTropic, also called Phenibut. They are used in a similar way as Gabapentin.

Addressing GABA Pathway and Benzodiazepine Issues

Supplements: PheniTropic 3X per day, Taurine 500-1000 mg am/pm, Tryptophan 500-1500 am/pm, Niacin 100 mg, Theanine 200 mg am/pm.

Toxic Impairment: Clear up neurotoxins, specifically pesticide and insecticides. Avoid diet sodas, aspartame and *any* stimulants. Mercury can often be implicated in these cases as can other toxic metals.

Diet: Eat organic whenever possible. Consider a microbiome cleanse and bone broth if “benzo belly” is present.

Environment: Increase physical activity, specifically walking in a relaxed area. Try not to make big life decisions. During this time, a person is probably in fight or flight mode and this will taint their perspective on life. We have successfully handled many 20+ year benzo cases and it has been a rewarding but often challenging journey. You *can* get thru this, you just have to be strategic and go at your own pace and not take your current thoughts too seriously, they will pass.

Consider bridging with Gabapentin. When done correctly, this may be just the tool to round off the edges of this withdrawal and make it tolerable.

Rapid detox for benzodiazepines is lunacy. We have unfortunately stopped working with folks who have just exited a rapid detox. It is not necessary to throw someone into crisis under the pretense that they are an addict. People coming into the center in that sort of crisis are basically unmanageable. It is not necessary to throw someone into crisis during withdrawal. We would much rather take someone on a stabilized medication level and do the tapering ourselves as we will support them at a pace that they can safely handle.

Alcohol

In all cases, it is important to grasp the addictive biochemistry of a situation. There are several types of alcoholics, a concept first posed by Joan Mathews Larson, and which we will be expanding upon here.

1. Anxiety related
2. Hypoglycemic
3. Allergic addicted
4. Endorphin deficient

It is important to know what kind of alcoholic a person is so that you can create a working strategy. Without knowledge of the addiction and correction of the underlying addictive biochemistry, a person often falls back into the same trap.



Anxiety Related

Most everyone who has a drinking problem has anxiety. The type of anxiety we are highlighting here involves a reasonably constant type of anxiety that does not come or go. This type of person is self-medicating to lessen anxiety so that they can function. We are not talking about the anxiety that comes up first thing in the morning or directly after their alcohol levels drop. We are talking about people that have anxiety for long periods of time, even when they are not drinking. These folks are probably clogged up with excitotoxins and need to address that underlying toxicity.

Hypoglycemic

Alcohol is a liquid sugar. It does not even need to be digested as the sugar alcohol passes straight from the stomach to the bloodstream. If a person is experiencing low blood sugar, the craving is of course for sugar and even more potently, alcohol. You can spot these folks often because without alcohol they gravitate toward sugar and coffee, which is the same addictive biochemistry. The drinking pattern is usually a person who drinks coffee first thing in the morning, and if they eat in the morning at all, it is a likely sugar based. Then, at some point in the afternoon, the blood sugar plummets and they drink. They often present as the all day, every day drinker, that keeps a steady state alcohol level. Unfortunately, the AA programs support this addictive biochemistry by serving donuts and coffee at meetings. The philosophical aspects of AA are fabulous, their attention to the addictive biochemistry is not.

To break this pattern, it is going to take several months of dedicated effort. The person should eat protein based breakfast every morning, and eat at least 3 times per day, avoiding sugar. Coffee should be avoided for the first several months, and if added, which is not recommended, should only be consumed after eating breakfast. The power of a blood sugar drop to create cravings for alcohol is a brain survival mechanism that will overpower even the

best of them. The vast majority of people who do *not* put attention on their diet will often “white-knuckle” their recovery until they eventually relapse. If you really want to break the hypoglycemic addiction to alcohol you will break the addiction to sugar by addressing hypoglycemia. Hypoglycemic dispositions can occur in combination with any of the other dispositions and any person suffering from alcohol should respect these basic tenants.

Allergic Addicted

When a person is allergic to alcohol, the inflammatory reaction releases endorphins and the person gets addicted to their own endorphins. You can spot this type of person because they have a very low alcohol tolerance before their mood turns. Any nationality can have this disposition, but is more common in the Native American and Asian populations. Binge drinkers also often fall into this category. The fixes for this are the same recommendations given in the endorphin-opiate section.

Endorphin Deficient (THIQ)

Technically, being allergic addicted is an endorphin deficient disposition. What is unique about the THIQ disposition is the drinking pattern and the mechanism. THIQ is an opiate like compound that is produced in small amounts during the breakdown of alcohol. But, you have to drink a lot to get there. These folks have a fantastic liver, and can convert alcohol extremely well. You can spot these folks as they are the ones that can drink ridiculous amounts of alcohol and still be standing. The THIQ as an opiate can lower the social anxiety and these folks are generally the life of the party. Eventually, the party stops and the body breaks down and they find themselves stuck. The correction for this type of drinker is the same as those given in the endorphin-opiate section.

To Address Alcohol Issues:

Supplements: Address Endorphin issues as mentioned in the endorphin section. Add B vitamins, specifically B1, B2, B5 and niacin as alcohol burns up B vitamins. You can take 500 mg of niacin every time there is a craving.

Toxic Impairment: Stop smoking if possible, as cigarettes are often cured with sugar. At least switch to American Spirits which have less additives than more popular, commercial brands.

Diet: Eliminate sugar and caffeine. Include protein based foods. Follow the recommendations regarding hypoglycemia, this is *the* most important point to take in. Eat and or drink water if there is a craving. Thirst and hunger are often confused with alcohol cravings.

Environment: Get into a supportive community. That might be AA, church, yoga, or any other community a person is attracted to. It is not good to get lonely or bored in early recovery.

Closing Comments

If a person is low in one inhibitory neurochemical, then they are likely low in others, as other neurochemicals try to compensate. The same goes for excitatory chemicals as well. It is best to feed the entire spectrum of inhibitory chemicals when a person is too revved up. If a person is too flat they may want to utilize DLPA and tyrosine and they could even do some of the inhibitory protocols as well. You do not however, want to do a stimulatory protocol with someone who is anxious or having any kind of delusions.

The supplement recommendations here are very basic and mostly involve the precursors to the neurotransmitters being addressed. The Alternative to Meds Center uses many hundreds of different products tailored to individual situations. We keep it simple in this book for those who cannot come to the center and wish to bring this information to their at-home doctors.

The Alternative to Meds Center has a very strong science component, but is ultimately a place of love and compassion. We find that a true caring environment, with staff that relate to the emotions a person is experiencing, is where the true healing begins. Really, how else can a person heal, but in a place where they feel loved?



Disclaimer

Much of what has said in this book is outside of conventional wisdom. Generally speaking, there is no conventional wisdom regarding medication withdrawal available. Therefore, we have postulated theories based upon our clinical observations and we avail ourselves to change and deeper understandings as we collectively evolve our understanding.



None of what has been said should be considered medical fact or medical recommendations, but rather practical guidelines, knowing that each person is an individual and responds uniquely to treatment. If you are considering any recommendations given in this book, consult with a medical professional who is actually present with you through your process. If you do not have such a doctor, you can find one in your area using the ACAM.org website.

Thank you and good luck in your journey.

¹ Always consult with your doctor before taking supplements or changing any significant aspect of your health regimen. The following is not intended as a medical recommendation but rather as reference material you can discuss with your doctor.