Now I've Gotcha!

Addictive chemicals from sugar to meth

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Introduction

What do you experience if you miss your morning coffee, your morning cigarette, or prematurely run out of your pain medication? Why are these chemicals so addictive? Do you have a loved one struggling with an addiction? Are you struggling with an addiction?

Addiction is enslaving and killing our young people, devastating families, overstressing first responders and emergency room staff, and financially draining limited healthcare dollars.

Addiction is a monster that needs to be understood and contained.

Only when you know what it looks like and how it behaves will you have a chance of getting it into an appropriate cage.

Addiction is a disease of comfort-seeking. If I had the power to remove one phrase from the medical vocabulary, it would be the phrase, “drug-seeking behavior,” this phrase should be replaced with the more accurate, “comfort-seeking behavior.” The addict seeks comfort as a cure for the discomfort of their pain and anguish.

Scientists in the field of neurobiology have made significant recent advances. This short book provides a brief description of the science of addiction and why some of

these chemicals, like sugar, caffeine, nicotine, alcohol, marijuana, opioids, cocaine, Ecstasy, Kratom, and methamphetamine, are so addictive.

I hope that an awareness of the power that these chemicals possess will increase the reader's respect for them. These chemicals can rob you of all that you value, and before you realize it, destroy your life.

CHAPTER 1 - Addiction

An addictive substance is a non-essential element, meaning it is not necessary to maintain or sustain life. These chemicals can impact the nervous system by producing a sensation of satisfaction as it creates the desire (craving) and need (physiological dependency) for more of the element. Using the element repeatedly will result in a changed nervous system that now defines the element as being necessary for life, as essential as oxygen, food, water, shelter, and relationships. Eventually, the value of this element surpasses all the other ingredients, and the addicted individual will be willing to sacrifice any of the other elements to get more of the addictive substance.

The Merriam-Webster definition of addiction is "the compulsive need for and use of a habit-forming substance characterized by tolerance and by well-defined physiological symptoms upon withdrawal: broadly: persistent compulsive use of a substance known by the user to be harmful."

Addiction is a strong memory in the reward pathway of the brain.

To understand this memory, we need to understand epigenetics. Genetics is the DNA that we inherit, accounting for our hair color, eye color, etc. Epigenetics it how

individual segments of DNA get expressed in a cell. Every cell has the same DNA, but through epigenetics, cells perform different functions – because of epigenetics, a muscle cell is different than a kidney cell. Epigenetics allows our nervous system to remain adaptable and learn new things. Memory is an epigenetic structural change in the DNA in the neurons in your brain.

Let's take a look at how epigenetics works in memory formation and then in the development of an addiction. A stimulus causes a release of powerful neurotransmitters that directly impact the DNA in the neurons in various ways that will eventually result in the formation of a memory.

Triggered neuroplasticity is the process of stimulus-induced change. Triggered neuroplasticity is the ability of the neurons to change or adjust because of stimuli. Without neuroplasticity, we would not be able to learn or develop new memories or, as we shall see, develop an addiction.

The most common process in triggered neuroplasticity is DNA methylation. DNA methylation leads to DNA exposure. This change in the DNA is similar to the impact a cancer-causing chemical has on a cell that results in it becoming cancer. DNA methylation prepares the cell to change its structure and function.

Also occurring is a process called posttranslational modifications of histones that allows the tightly coiled DNA to uncoil and expose areas of DNA that can then be modified.

The DNA is now, no longer a tightly coiled bundle of DNA. The cell structure and function continue to change through modifications of noncoding RNAs that remodel chromatin and facilitate or suppress gene expression – the cell changes and protein manufacture can be turned on or turned off. The neuron has now changed both its structure and its function. Now you have a newly formed memory.

Let's take a closer look at this process in the context of something we can all relate to – the development of different levels or types of memory.

Short term memory formation is the result of a minimal stimulus. This kind of memory extinguishes quickly. For example, what do you remember about last Wednesday? It was not long ago. The chances are that unless it was a special day, you do not remember much. You might remember if it was your birthday or anniversary, but if it was not, then the details are long gone.

If you increase the stimulus, the memory will last longer. This type of memory formation is the effect seen when cramming for exams, or with the memory of significant historical events – such as the assassination of President Kennedy or the first moon landing. What do you remember about September 11, 2001? Do you remember that it occurred on a Tuesday? Do you remember where you were? Do you remember what you did that morning or what you had for breakfast?

If you increase the stimulus, even more, the memory will last even longer, to the degree it may have a lasting, lifelong impression. The event changes you. You may also act differently or interpret the world differently because of this overwhelming stimulus caused memory. What do you remember about what happened in Boston on April 15, 2013? We most likely share very different memories of this day. On this cold, windy day on Boylston Street, my wife and I were standing across the street from the second bomb at the Boston Marathon. I can remember a lot about that day. I can easily relive that moment – from what I had for breakfast to the sensation of the cup of hot chocolate in my left hand as we experienced the explosion across the street. The overwhelming stimulus changed me. This level of stimulus causes a profound type of memory that may result in post-traumatic stress disorder.

This process of DNA unfolding and changing both its structure and function is how different degrees of memories are formed and stored.

Addiction develops similarly except the area of the brain being affected is the reward pathway of the limbic system, and the magnitude of the stimulus caused by drugs of addiction is more overwhelming than the stimuli that result in the formation of a profound memory. The structural and functional changes in the reward pathway of the brain have been confirmed with several medical studies. A study on the

effects of cocaine on the brains of rats showed that there were 17 DNA changes in six brain areas. A study on heroin addicts that had been in confirmed recovery for over nine years showed ongoing significant functional MRI changes when exposed to auditory or visual drug-using stimuli. A study where subjects were given Morphine 50 mg daily for 30 days and then followed with monthly functional MRIs showed that the changes in brain function that occurred because of the morphine exposure did not improve six months after the completion of the study. Changes in these areas of the brain have a prolonged and dramatic effect. Some of these changes may be permanent.

Chemicals that can cause addiction present an overwhelming stimulus to the neuron. This stimulus changes the DNA in the neuron. The stimuli cause a flood of dopamine from the nucleus accumbens that sends messages to parts of the brain, saying, "this is good and necessary." The experience or feeling is something to be desired. You now have a powerful memory of desire and craving.

The impact of the stimulus in the hippocampus enhances the memory of the reward experience. It reinforces the memory that: "This is something I do not want to forget – I should remember as much about this reward as possible – where I got it, who I got it from, the people I used it with, the room I used it in – and on and on."

The impact on the amygdala is an emotional one. It produces a feeling of security, confidence, and peace – a level of completeness never experienced before drug exposure.

The impact on the prefrontal cortex is to assign a high level of value to this experience. Nothing else compares to this experience, and the now addicted individual is willing to pay whatever it may cost to maintain this feeling.

As previously described, different levels of stimulus cause different permanence of memory. Varying degrees of activation of the reward system results in varying levels of formed desire. A low-level reward stimulus results in a preference. I like puppies better than kittens is an example of preference, but I don't want to pay for that puppy or pay to take care of it for the next seventeen years. I am free to make rational choices. Preferences can easily be changed, especially if you have an issue with your preference or a better preference presents itself. Wait till that puppy chews your favorite shoes.

Increase the stimulus in the reward pathway, and you get what we call a desire. A desire comes from a felt need for a sense of completeness. This desire area is the realm of the advertising industry. If I can link your felt desire with a product that I sell, then the sale is easy. If I can sell you the fantasy that a new Lamborghini can give you a sense of contentment or acceptance, then you just bought a new

Lamborghini. Unfortunately, the romance quickly fades with the first scratch, the $1,000 oil change, the eventual awakening to the reality that the illusion did not bring the contentment or sense of wellbeing it promised. But we still keep searching and buying.

If you increase the stimulus, even more, you have an overwhelming stimulus. This overwhelming stimulus requires significant brain changes to accommodate, and you end up with an addiction. You are now past minor preference and desire, into the world of an intense craving. The chemical has effectively highjacked your neurons. Your brain is telling you that you may die if you do not get the substance that produced the stimulus. Stimulus caused alteration in the function and structure of brain DNA is the disease of addiction.

These addictive elements work in several different ways; they can mimic a natural neurotransmitter and activate a nerve cell directly, they can cause an increased release of a natural neurotransmitter, they can cause a delay in the removal of a natural neurotransmitter, or they can attach to a receptor and block the action of a neurotransmitter. What they all share in common is that to varying degrees, they all cause an increase in dopamine activity in the nucleus accumbens part of the brain. This release of dopamine labels the element as a reward and facilitates memory of the environment associated with that reward. These

characteristics are all shared by the following addictive items: sugar, caffeine, nicotine, alcohol, marijuana, opioids, cocaine, Ecstasy, Kratom, and methamphetamine.

Why is it so hard to quit? The brain is a delicate organ, designed to function with high speed while maintaining a precise balance. If there is just a little too much glutamate released, the cell dies, if too little is released, normal mental function is not possible.

Each addiction has its specific neurological and physiological effects, both during the use of the substance and during abstinence. The alcoholic gets anxious; the narcotic user experiences pain; the tobacco user cannot concentrate. Each of these highly unpleasant symptoms can be enough to encourage continued use, yet there is still another layer of anguish shared by all addictions.

To varying degrees, all addictions cause issues with regulation of glutamate in the nucleus accumbens. The normal function of glutamate in the nucleus accumbens involves the release of glutamate with rapid activation of the next cell and then prompt removal of the glutamate by excitatory amino acid transporter 2 (EAAT2). If the glutamate is not cleared rapidly, it can overexcite the cell causing its death. All addictive compounds decrease the effectiveness of glutamate on the cell membrane and reduce the activity of the excitatory amino acid transporter 2. During times of abstinence, this regulation becomes very unstable,

causing a sense of severe anxiety, stress, sleep disturbance, profound fatigue, poor memory function, and even cell death. Abstinence feels like impending death. The loss of control over mental functions feels like one is losing their mind.

The brain, its delicate balance, and proper function is now the prisoner of the compound. Now I've gotcha! Choosing to consume a known toxic chemical feels like life when, in reality, it is a movement towards a premature death.

Living life with addiction is like trying to swim with lead weights on your ankles – don't get addicted to anything, and if you are addicted, get help immediately – your life depends on it.

CHAPTER 2 – Sugar

Once our taste buds have experienced the taste of sugar, we are trapped. From that time on, all it takes is thinking about the taste and dopamine levels in the nucleus accumbens rise. The brain uses 50% of the body's available sugar energy. This sugar energy is taken directly from the blood because the brain has no effective way to store sugar. The proper balance of sugar is necessary for the manufacture of neurotransmitters and brain functions like attention, cognition, and mood stability. If there is too much sugar in the brain it ages more quickly, accelerating memory loss and dementia.

The amount of sugar in our average diet has increased dramatically in the last 100 years. The amount of sugar eaten in a year by my grandfather when he was seven will be consumed by my seven-year-old grandson in less than three weeks.

This increase in sugar consumption not only causes an increase in the risk of diabetes, but it also causes increased inflammatory stress on the nervous system.

There is a delicate balance in the levels of glucose, insulin, cortisol, and serotonin that must occur for our nervous system to function correctly. A rapid increase in glucose will

potentially be toxic to the nerve cells, so the body responds by increasing levels of insulin to decrease the brain concentration of glucose by sending the excess glucose into the cells – most often the fat cells. Cortisol, the stress hormone that prepares the body for fight or flight, also plays a role in decreasing a rapidly rising brain sugar level by causing an increased movement of blood sugar into the fat cells. If the brain level of sugar drops too quickly, cortisol is released to increase the release of glucose from the liver to ensure the brain has enough sugar to function properly.

If the brain level of glucose rises slightly, it causes an increased release of serotonin – the neurotransmitter that has a mood-calming effect – it reduces anxious thoughts and feelings and may even lessen the experience of physical pain. If the serotonin levels drop, then the result is often increased cravings for sugary or carbohydrate-rich foods that have a high amount of simple sugars.

In small amounts, sugar-containing foods are not harmful or even very addictive, but if there is a repeated intake of high sugar foods in someone genetically sensitive, then a condition can occur where the cells become less sensitive to the effect of the rising levels of insulin. This condition is called insulin resistance and is the cause of type 2 diabetes. People with type 2 diabetes have intense sugar cravings; they have an addiction to sugar. This addiction causes obesity, blood vessel disease, heart disease, kidney

disease, and brain inflammation resulting in memory and mood problems. If there is one primary gateway drug, that drug is sugar.

CHAPTER 3 – Caffeine

Caffeine acts as a stimulant in the nervous system, and it does this by the way it works on adenosine receptors. Adenosine's action on the surface of the nerve causes the nerve activity to slow down; this causes drowsiness, dilates the blood vessels, and aids in falling asleep.

Caffeine acts as an antagonist of adenosine, meaning it attaches to the receptor and does not allow the adenosine to connect to the cell membrane. Caffeine also causes an increased release of an adrenocorticotropic hormone that leads to an increased release of adrenaline that can improve alertness, learning, and memory.

Caffeine can also cause increased anxiety, increased stomach acid, increased blood pressure, fine muscle tremors, and increased urination.

If excessive amounts of caffeine are consumed, in the range of ten to fifteen cups of regular coffee, a condition called caffeinism, can occur. This condition combines caffeine's physical dependence with symptoms of nervousness, insomnia, restlessness, irritability, irregular or rapid heart rate, and headaches.

If used in modest doses, caffeine is not very addicting in that it seems to cause very little tolerance, but it may cause some

physical dependency in 50% of caffeine users who experience headaches, sleepiness, irritability, and nausea if they stop the intake of caffeine.

Dependency on caffeine is the result of the increased levels of dopamine it produces in the nucleus accumbens core.

CHAPTER 4 – Nicotine

Nicotine comes from tobacco products; it can be inhaled, sniffed, chewed, or worn as a patch. Once nicotine enters the body, it goes to work very quickly.

The release of adrenaline from the adrenal glands causes an immediate increase in respiratory rate, blood pressure, and heart rate. If someone has issues with elevated blood pressure, they should not smoke for several hours before having their blood pressure evaluated.

Nicotine imitates the naturally occurring neurotransmitter, acetylcholine. It binds to acetylcholine receptors throughout the body. The three significant activation areas are in the adrenal gland, where it causes an increased release of adrenaline, the cerebral cortex where it mimics acetylcholine, and in the ventral tegmental area where it causes a release of dopamine in the nucleus accumbens shell.

The increased release of dopamine in the shell of the nucleus accumbens causes a powerful and lasting memory of reward. This action in the nucleus accumbens shell is the primary cause of the source of cravings that can occur years after someone has quit smoking. In February 1985, I was attending a patient with a very challenging labor. It was a

time of much stress and anguish. The laboring mom was exhausted, the labor had progressed very slowly, and it took the assistance of carefully applied forceps to deliver a healthy baby girl. Exhaustion turned to excitement; anguish turned to joy – everyone could take a deep breath. As I walked the half-mile home in the sub minus 20-degree winter morning, the air was brisk, the snow crunched beneath my feet, and the green and magenta northern lights danced in the sky. All was well with the world as I puffed on a cigar that I had just received from the new father. I am not a smoker, and it has now been over 30 years, yet on a cold, dark, Thursday morning, when I take my garbage can to the street, I want a cigar. The memories of reward from the nucleus accumbens shell are robust and long-lasting.

It is the action of nicotine on the acetylcholine receptor that creates the need for that next cigarette. The acetylcholine receptor activated by the nicotine briefly becomes unresponsive to any neurotransmitter. These neurotransmitter receptor naps become extended with regular exposure to nicotine. It is like the receptors of acetylcholine go to sleep.

This condition of nicotine tolerance can develop quickly, so now, when the level of nicotine drops a little, those acetylcholine receptors throughout the brain begin to wake up, and they are not happy. It takes about twenty cigarettes per day to keep those receptors quiet.

Without the nicotine, the receptors are now hypersensitive to the activity of acetylcholine. This hypersensitivity results in the experience of irritability, attention difficulties, increased appetite, and powerful cravings. The smoker's experience of that mad and desperate rush for that first cigarette in the morning functions to put the receptors back to sleep and recreate a sense of calm.

CHAPTER 5 – Alcohol

Alcohol absorbs very rapidly and enters the bloodstream quickly, reaching all parts of the body. In the brain, alcohol binds to receptors for acetylcholine, serotonin, GABA, and the NMDA glutamate receptors.

This binding to multiple receptors has a profound effect on mood, memory, personality, alertness, and physical coordination. The most significant impact of alcohol in the brain is on the GABA receptors and the NMDA glutamate receptors. Glutamate is the most prevalent excitatory neurotransmitter in the brain.

Activation of the GABA receptors causes most of the effects experienced by alcohol, such as sedation, relaxation, less anxiety, and a greater sense of calm with some decrease in inhibitions. Alcohol imitates GABA and inhibits nerve activity more powerfully than GABA, and it also decreases glutamate activity at the NMDA receptor.

Alcohol decreases glutamate's ability to bind to the NMDA receptor causing inhibition of NMDA receptor and glutamate activity. This effect on the NMDA receptor can result in memory blanks. During times of abstinence in chronic alcoholics, the NMDA receptors become upregulated,

resulting in enough glutamate activity to kill neurons and cause seizures and hallucinations.

Over time these receptors accommodate the presence of alcohol, causing the NMDA receptors to become more sensitive to glutamate and the GABA receptors to become less sensitive to sedating effects of the alcohol. The accommodated state, or new normal in the presence of alcohol, results in a hyperexcitable state during times of abstinence.

The addictive effect of alcohol occurs at the nucleus accumbens, where it increases levels of dopamine by slowing down its reuptake.

CHAPTER 6 – Marijuana

The active ingredient in marijuana is delta-0-tetrahydrocannabinol (THC). THC mimics the action of the natural neurotransmitter N-arachidonoyl ethanolamide (anandamide). Anandamide binds to CB1 cannabinoid receptors throughout the brain and CB2 receptors in the body. CB2 receptors have a role in regulating immune function. CB1 receptors impacts mood, memory, appetite, pain, and cognition.

CB1 receptors activated by either anandamide or THC causes a reduction of neurotransmitter to be released from the neuron, causing a general decrease in brain excitability. This action causes relaxation and change in mood, altered sense of time, impaired memory, difficulty with problem-solving, altered body movements, and an altered sense of hearing and sight.

There are no CB1 receptors on the dopaminergic neurons in the reward pathway of the brain. THC and anandamide enhance the dopamine activity in the reward pathway by inhibiting the GABAergic neurons that would normally cause a reduction in dopamine released, with inhibition of the GAGAergic neurons more dopamine is released.

With chronic use of THC and an overstimulation of the receptors, the brain compensates by reducing the number of CB1 receptors. Reduced CB1 receptors in the arteries of the brain cause vasoconstriction resulting in a sustained reduction of blood flow. This reduction in blood flow results in oxygen deprivation in the brain, causing problems with memory, attention, learning ability, and possibly an increased risk of stroke.

CHAPTER 7 – Opioids

Narcotics mimic the action of the body's naturally occurring opiate-like substances called endorphins, enkephalins, and dynorphin. There are three types of opioid receptors present throughout the brain; mu receptors, delta receptors, and kappa receptors. Narcotics have most of their effect on the mu receptors, of which there are seven subtypes. The body's natural opioids attach to one or more of these receptor types to regulate hunger, thirst, mood, immune response, and awareness of painful stimuli. In the brain stem, the opioid receptors are active in regulating blood pressure, alertness, and breathing.

When an opiate attaches to a mu or delta receptor in the brain, it reduces the excitability of that neuron, producing a sensation of calm and euphoria. When the opiate attaches to a mu receptor in the ventral tegmental area of the brain, it acts to reduce the excitability of the GABAergic neurons causing less inhibition in the release of dopamine in the nucleus accumbens, thus activating the reward pathway. Ongoing receptor activation by opiates results in several adaptations in the neurons. When this occurs, more opioids are required to get a previous effect; the neurons have developed a condition called tolerance, where the cell is less

sensitive to the action of opioids. The cells adapt by changing their structure to make them less sensitive to opioids, or they can internalize the receptors to make less of them available on the surface of the cell membrane. The internalization occurs by the cell membrane covering the receptor and pulling it into the body of the cell. The cell is now more resistant to the action of the opioid.

A condition of increased sensitivity to painful stimuli can also occur with the ongoing use of opiates. This condition is called opioid-induced hyperalgesia and can present as either hyperesthesia: a condition where there is a dramatic increase in sensitivity to painful stimuli, or allodynia: a condition where the pain is brought about by stimuli that would normally not cause pain. This condition of increased pain is caused by the opiates increasing the activation of glutamate NMDA receptors in the spinal neurons that make them more sensitive to painful stimuli and by increasing spinal cholecystokinin and dynorphin, which cause amplification of the painful stimuli presented to the spinal cord.

CHAPTER 8 – Cocaine

Cocaine works by blocking the channels that typically remove the neurotransmitters dopamine, norepinephrine, and serotonin, with the result being an amplified effect of all of these neurotransmitters.

The increased dopamine action in the nucleus accumbens results in more dependency - the increase in the level of norepinephrine increases energy - the increased serotonin levels result in an increased feeling of confidence.

The artificial increase in the activity of these three neurotransmitters produces a powerful sense of pleasure in the reward center of the brain.

Chronic cocaine use results in an extensive adaptation by the neurons, especially in the reward pathway where the high levels of cocaine produced dopamine results in the cell membrane manufacturing new dopamine receptors, so it now takes more dopamine to produce the previously felt level of satisfaction. If the dopamine level falls to the level of an ordinary, happy, content cocaine naive adult, the cocaine adapted brain will feel a deep depression with intense cravings.

CHAPTER 9 – Ecstasy

Ecstasy (MDMA) is the synthetic drug 3,4-methylenedioxymethamphetamine that acts as both a stimulant and a hallucinogen similar to both amphetamines and LSD.

Ecstasy blocks the reuptake pumps for the neurotransmitters norepinephrine, dopamine, and serotonin. The effect on dopamine and norepinephrine is minor; its primary impact is on serotonin, where it causes both an increased release of serotonin and inhibits the reuptake of serotonin. The increase in norepinephrine causes an increased heart rate and blood pressure. The rise in dopamine causes an increase in energy, activity, and euphoria. The increased flood of serotonin caused by Ecstasy causes an elevation of mood and a surge of oxytocin release in the hypothalamus. This surge of oxytocin causes an increased sense of wellbeing, trust, and closeness.

If the rise in serotonin is too high, then a condition known as serotonin syndrome develops that causes agitation, elevated body temperature, increased reflexes, tremor, sweating, dilated pupils, diarrhea, seizures, liver damage, muscle breakdown and possibly death. Serotonin syndrome is easily mistakenly diagnosed as opioid withdrawal.

The surge of serotonin lasts three to six hours, followed by a decrease in serotonin that lasts about a week. The reduction in serotonin and oxytocin causes irritability, aggression, sleep problems, depression, decreased appetite, problems with memory and attention, anxiety, reduced trust, and closeness. The ecstasy user is left feeling isolated and anxious for a prolonged period.

CHAPTER 10 – Kratom

Kratom comes from the tropical evergreen tree Mitragyna speciose. Mitragyna speciose is from the coffee family and is native to Myanmar, Malaysia, Thailand, Indonesia, and Papua New Guinea. Kratom consumption is by capsule, pill, or tea. The active ingredients in Kratom are mitragynine and 7-α-hydroxymitragynine. With the use of small amounts of Kratom, the result is a stimulant effect. In larger doses, it affects the brain's opioid receptors causing sedation, a sense of pleasure, and a reduction in pain. In very large doses, Kratom can produce effects similar to psychedelics. Kratom has recently become very popular for chronic pain patients who are weaning from narcotics. Kratom is currently legal for purchase, but it is highly addictive. Patients have reported that it has been harder to get off Kratom than it was to get off heroin.

CHAPTER 11 – Methamphetamine

Amphetamines cause their overwhelming effect through their action on dopamine. Amphetamines cause a dopamine flood, and that is what makes them so highly addictive. Methamphetamine can increase the level and activity of dopamine by every known mechanism. Amphetamines cause this flood by mimicking dopamine, entering the nerve ending by way of the dopamine transporters, causing an increased release of dopamine, blocking its reuptake, reversing the directional flow of the dopamine transporters, and inhibiting the action of monoamine oxidase A that is responsible for the breakdown of dopamine. This increase in dopamine results in increased alertness, increased physical activity, a decrease in appetite, increased heart rate, and breathing rate, increased blood pressure, altered judgment, and altered decision-making, often leading to increased risky behaviors.

Amphetamines can also increase the excitability of dopaminergic neurons by its action on glutamate neurons. The long-term damage caused by methamphetamine use can continue for years after abstinence.

With long term use, the dopamine-sensitive cells accommodate like described with long-term cocaine use with

the result of depression and intense cravings in the midst of what would be typical levels of dopamine.

CHAPTER 12 – Now I've Gotcha!

All of these chemicals produce a form of immediate pleasure. This pleasure sensation is a fleeting chemical reaction. With repeated use, there is diminishing immediate pleasure, and now the chemical is needed to maintain a new equilibrium. Attempting to stop an addicting substance causes increased pain and discomfort. An addiction may be initiated out of experimentation, wanting to fit in, as a self-treatment for pain or despair, or as an act of defiance, but discomfort is why the addiction is maintained. The discomfort can come from many sources, and at times the cause of the pain experience is confusing. Pain is a frequent, universal human experience. You will experience some pain or anguish today, it may be mild, or it may be catastrophic. It may be physical, or it may be emotional. Be on your guard, and do not move to an addictive substance to control the discomfort.

An addiction will create discomfort and steal your control. Lack of control and discomfort is a powerful force that robs you of your ability to be productive, compassionate, and active. When we are distracted by the chaos and pain of life, we are not free to grow and develop. We feel stuck because, at that point, we are stuck. Our survival depends

on finding a path through the chaos and discomfort that will build us up and not tear us apart. Do all you can to avoid these chemicals that cause addiction. Live as if you are genetically prone to developing an addiction. Living with addiction is like trying to swim with lead weights you have placed on your ankles. If you are struggling with an addiction, then get help. Recovery programs are successful. Living a life of recovery where you are daily doing the little things that move you closer to becoming the person you desire is a rewarding and fulfilling life. It is your life; spend it wisely.

References

If you are interested in reading more about this topic, a list of references is available at fracturedresilience.com.

Fractured Resilience Podcast Channel
https://open.spotify.com/show/4icf8jdtbCtbX5oj0OIxmP

Neurobiologic Advances from the Brain Disease Model of Addiction: **Nora D. Volkow, M.D., George F. Koob, Ph.D., and A. Thomas McLellan, Ph.D.**
https://nejm.org/doi/full/10.1056/NEJMra1511480

McGill University in Montreal, Canada has an excellent web page on drug effects on the brain:
http://thebrain.mcgill.ca/

Best text book on the disease of addiction is:
ASAM's Principles of Addiction Medicine 5th edition

Excellent source of information on the US government web site:

SAMHSA – Substance Abuse and Mental Health Services Administration: https://www.samhsa.gov/

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