QUESTIONS and ANSWERS from
The Neurobiology of Addiction and Pharmacological Concepts

webinar presented by

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Q: how does pregnancy affects the neurobiological response?

A: In addition to the hormonal effects on the nervous system, in pregnancy, there is an increase in the area of distribution of the drugs (the drugs also are distributed to the fetus, so there is less in the brain, causing a cyclical craving for more drugs.

Q: Does any research support the idea that sugar produces a similar response in the brain for a portion of the population?

Answered during the presentation

Q: I have heard that cravings are shown to last only about 60-90 seconds. Is that true? I also describe cravings to my patients as, "the cravings 'hijack' your brain"

Answered during the presentation

Q: what is reference for slide 1 statistics (number of Americans affected, percentage in workforce, etc.)

A: It is from the SAMHSA website.

Q: Is "PAWS" accepted as a diagnosable syndrome by mainstream addiction scientists/researchers? Is PAWS measurable through brain scans or other methods that would lend credibility that PAWS is a Neurobiological syndrome?

Answered during the presentation

Q: Considering the role of dopamine receptors, do antidepressants have a role in recovery phase for those addicted to opiates?

Answered during the presentation

Q: In the Change Point Model, if we have an internal thermastat to gauge what happens to the person with depression?

A: One hypothesis is that a similar dynamic exists with depression as with chemical dependence.

Q: Where can we find a good review in written form of your talk i.e. and article?

A: Although I use many, many references for my presentations, I suggest any recent text on Neurobiology in general, or specific to chemical dependency. Also, there are many recent, good articles on this topic which can be found in a literature search.

Q: How does Vivitrol "resensitize" opiate tolerance? Does the "resensitization" indicate Vivitrol or Naltrexone may be repairing the brain?

A: Naltrexone (Vivitrol) is an antagonist at the dopamine receptor and prevents stimulation of the dopamine receptor. Although the exact mechanism of stabilization is unknown, one putative explanation is the dopamine receptors are not stimulated over time and the dynorphin is no longer manufactured. The receptors will eventually return to normal function. Although, the delta fos B molecules continue to be produced in the addicted brain which explains relapse even after many years of sobriety.

Q: There is an increasing trend of bath salts use, can you explain how that affects those receptors?

A: Bath salts are thought to follow both the 5HT (serotonin) and glutamate pathways. These neurotransmitters, in turn, cause the release of dopamine in the nucleus accumbens.

Q: can people who take simulants at stable dose for ADHD manifest with tolerance ,withdrawal and long term effects from resetting the dopamine receptor system?

A: Obviously, a brain that has been previously addicted to substances of abuse has changed and if stimulants are taken by these individuals (even though prescribed), there is an increase in the delta-fos-B concentrations which will (theoretically) lead to relapse. However, those who are not genetically predisposed to addiction will not have the same effects – although there has been ongoing concern about the other target organs affected by the stimulants – namely the pituitary (and decreased production of growth hormone) and the effect the stimulants have on the hunger centers of the brain (e.g.,the hypothalamus).

Q: Will the synthetic marijuana show as THC on urine testing. On your slide that you show to ED providers.

A: Synthetic cannabis does not give a positive result on the common urine drug screens.

Q: How do you handle the environmental and emotional based cues that induce relapse, when someone is in recovery?

A: The easiest and most effective way is to insure a very strong support system available to the identified patient along with adequate education for both the patient and the support system.

Q: What variables may influence one individual from becoming an addict and another not?

A: The current thinking refers to the Stress-Diathesis Theory (also known as the Two-Hit Hypothesis) which states there must be an internal factor (genetic predisposition) and an external factor (environmental stressor/trauma) and both must be present in order for addiction to be manifested.

Q: Please explain the relationship between the cognitive function (cerebral cortex) and the pleasure center (nucleus accumbans) in addictions.

A: An easy way I like to explain it is through Freudian thought: The cerebral cortex as 'ego' and 'superego' inhibit the impulsive/compulsive wishes of the pleasure center (nucleus accumbens). The cerebral cortex is what causes the need for gratification to be delayed. When the nucleus accumbens is chronically stimulated, there are impulses for immediate gratification. Specifically with chronic opiate addiction, the belief is (as shown on PET scans) that a significant amount of the neuronal fibers connecting the cerebral cortex to the VTA/nucleus accumbens have been destroyed. The good news is that we now know that the brain can either re-route this bridge or repair it if the addiction can be managed early enough.

Q: We know that meditation can change parts of the brain, do you see the use for meditation and other relaxation and stress reduction techniques as useful in treatment of addictions?

A: There is good, replicated research that demonstrates how meditation can change neuronal functioning and improve/re-establish healthy homeostasis (as does psychotherapy) and I am a firm believer of utilizing such measures.

Q: Is there a way to get handouts for this lecture? What other good resources do you recommend?

A: The handout can be found on the IntNSA website. SAMHSA is an excellent source of information and I highly recommend their site.

Q: what research is being done to redirect the response of those with addictive personality

A: There has been much criticism regarding the belief that such a personality exists. ASAM has officially rejected the existence of addictive personality since the traits require a pervasive, stable and rigid behavioral structure and addiction is seen on a continuum, pervasive only in the severely chronic and far from stable.

Q: is there a screening test that uses dynorphin and delta fos B levels to determine addiction?

A: Currently, there are no screening tests available.

Q: In terms of cue based relapse, can watching shows like INTERVENTION, lead to relapse in the recovering addict?

A: This has been hotly debated with proponents on both sides. There has not been any consensus – or valid, reliable data that I've seen one way or the other.

Q: How does the biologic theory of addiction explain individuals who report stopping chronic substance use after years of use and no apparent relapses.

A: Although we are aware of and admire this situation, there is no research available to help explain it. There is anecdotal evidence that in a significant number of these cases there is the presence of a strong support system.

Q: Getting back to cravings, is this something you can tell your patients it will pass in time and to wait it out before it becomes a compulsion to seek out the substance.

A: The current thinking is to give the patient (in addition to psychotherapeutic support) psychopharmacologic support using naltrexone (ReVia or Vivitrol) which helps with cravings.

Q: How do anti-craving medications work? Do they block pleasure from other activities (sex, exercise, etc.) and potentially cause disinterest/anhedonia?

A: A common myth is that dopamine antagonists block the feelings of pleasure and enjoyment in healthy activities – this is not true. These medications block the excessive euphoria that drugs of abuse cause. Remember, though, that the presence of a depressive syndrome co-occurring with the substance use disorder may cause anhedonia and is unrelated to the use of anti-craving medications.

Q: What role does Suboxone play in terms of the neurobiology of addiction?

A: Suboxone is a partial agonist at the mu-opiate receptor and has some very interesting properties. For instance, Suboxone has a very high affinity for the mu-opiate receptor and a very low dissociation coefficient which means it stays on the receptor for a long period of time. These two factors insure that an opiate agonist (oxycodone or heroin, for example) will not be able to stimulate the receptors because they are occupied by suboxone molecules. Suboxone produces a much lower level of stimulation – theoretically enough to prevent cravings.