Addictive Disorders on the Autism Spectrum: A Hidden Comorbidity?

Frankly, this program asks as many questions as it answers. We're in something of uncharted territory in a discussion of autism spectrum disorder (ASD) as it may correlate to addictive disorders. What is ASD? What causes it? Are ASD and addiction related? Can people with ASD become addicts, and is treatment available for them? Do people with ASD have a natural resistance to developing problems with addictive disorders, as has been assumed in the past? Has the comorbidity always been there, hidden from view?

INTRO

Hello, everyone, and welcome to our podcast! We're coming to you from our studio at the Council on Alcoholism and Drug Abuse of Northwest Louisiana! I'm Kent Dean, CADA's Director of Clinical Development. Today, we're discussing the presentation and possible interrelation between autism spectrum disorder and addictive disorders. You'll be able to earn one contact hour of continuing education by completing the post-test after you listen to the program. We'll give you instructions on how to do that at the end of the show.

People with autism and those with addiction employ repetitive behaviors in similar ways to cope with emotional problems. They're similar as well as in their impulsivity and compulsions. The two conditions affect some of the same brain regions, and they may involve some of the same genes as well.

According to a landmark 2016 Swedish literature review on ASD/SUD comorbidity, an autism diagnosis doubles the risk of addiction. Elevated risk is concentrated among those with an IQ of 100 or above. Parents and siblings of people with autism also have a higher risk of addiction.

To get a fuller understanding of possible interactions between ASD and addictive disorders, we need first to review the criteria for each group of disorders. First, here's a somewhat paraphrased version of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) description of autism spectrum disorder:

1. What Is Autism Spectrum Disorder?

A. Persistent deficits in social communication and social interaction, as seen by:

1. Abnormal-to-absent social approach and reciprocity behavior, including paucity of conversation. For example, I say, "Hi, Joseph," and extend my hand, and he fails to lock on to my gaze and hand extension in a coherent way.

2. Deficits in integrated verbal and nonverbal communication and in understanding and using gestures to a total lack of facial expressions and nonverbal communication. For example, Joseph's response to my direct greeting is more oblique, vaguer in that he doesn't fully address me bodily, meet my gaze directly, or respond proportionally to my verbal pleasantries.

3. Deficits in developing, maintaining, and understanding relationships, ranging from difficulties adjusting behavior to suit various social contexts, to difficulties in making friends, to absence of interest in peers. For example, despite my friendly greeting and manifest willingness to engage Joseph in a cordial way, his response remains somewhat flat at best.

B. Restricted, repetitive patterns of behavior, interests, or activities, as seen by:

1. Stereotyped or repetitive motor movements, use of objects, or speech. For example, simple motor stereotypes, lining up toys or flipping objects, echolalia, idiosyncratic phrases.

2. Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior. For example, extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat same food every day.

3. Highly restricted, fixated interests that are abnormal in intensity or focus. For example, strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).

4. Hyper- or hypo-reactivity to sensory input or unusual interest in sensory aspects of the environment. For example, apparent indifference to pain or temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement.

C. Symptoms must be present in early development, but may not become fully manifest until social demands exceed limited capacities. They may be masked by learned strategies in later life.

D. Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.

2. What Are Addictive Disorders?

Now, let's briefly review the DSM-5 criteria for substance-related disorders.

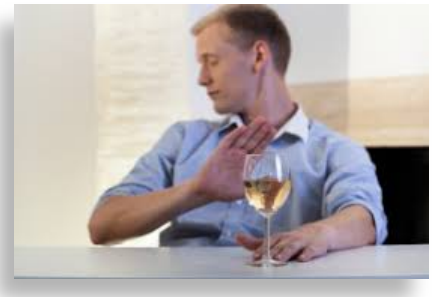
At least two of the following criteria must be manifest for a period of one year:

1. Taking the substance in larger amounts or for longer than intended.

2. Wanting to cut down or stop using the substance but not managing to.

3. Much time spent getting, using, or recovering from use of the substance.

4. Cravings and urges to use the substance. [Note: Interestingly enough, this criterion appears for the first time in DSM-5.]



5. Not managing to do what you should at work, home, or school because of recurrent substance use.

6. Continuing to use, even when it causes problems in relationships.

7. Giving up important social, occupational, or recreational activities because of substance use.

8. Recurrent use, even when it is hazardous to do so.

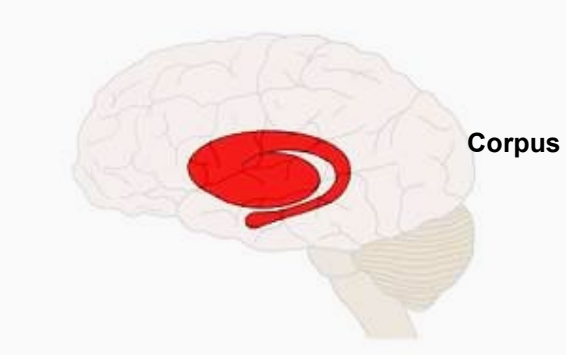
9. Continuing to use, even with the knowledge of having a physical or psychological problem that was probably caused or made worse by the substance.

10. Needing more of the substance to achieve the desired effect (tolerance).

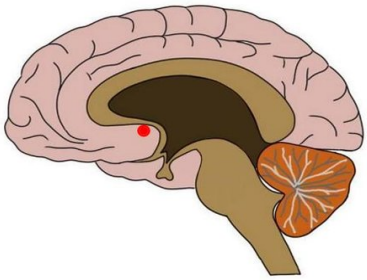
11. Development of withdrawal symptoms, which can be relieved by taking more of the substance.

3. Coetiology Between ASD and Disorders?

If we're looking for answers on the possible etiology of ASD, a fruitful place to start is the striatal area in the middle of the brain. This is the area containing structures known to be implicated in addictive disorders as well as ASD.



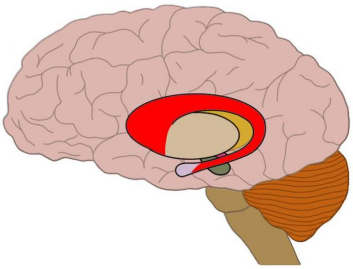
It's been observed that, before substance use progresses to addiction, brain activity as a reaction to chemical use occurs mainly in the ventral area of the striatum, which is involved with motivation and seeking pleasure. At this stage, people take drugs mainly because they provide a good, comfortable feeling. **The ventral region seems also to be involved with impulsive behavior. As the addictive process continues, the dorsal striatum becomes more involved. It is a region involved in rendering behavior automatic to more programmed patterns, set off by environmental triggers. Automation in the dorsal striatum can be useful when it processes a complex skill into a single, voluntary action—but it can also create a powerfully-insistent compulsion. The compulsion could be drug use, but it could also manifest itself as the repetitive behaviors characteristic of autism. In both cases, the striatum drives the persistent behavior.**



Nucleus Accumbens

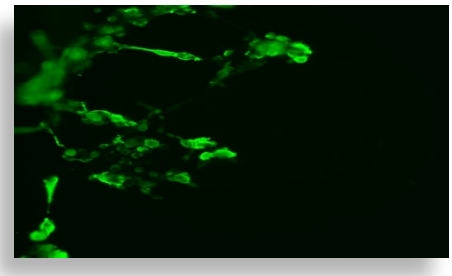
In the ventral section lies the nucleus accumbens (indicated by the small, red dot in the drawing above), which is involved with motivation, aversion, reward, reinforcement learning, and motor control.

Caudate Nucleus



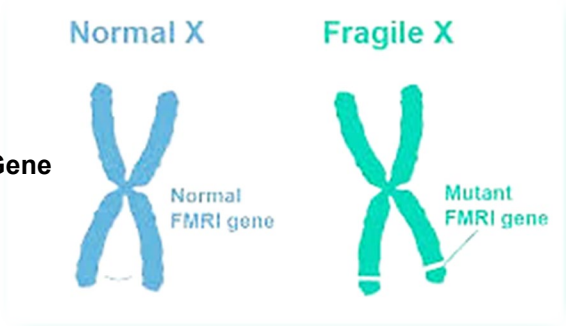
In the dorsal section, we have the caudate nucleus, which is involved in motor processes, procedural learning, inhibitory control, and pleasure and reward. Also in the dorsal section is the putamen, which is involved with movement planning, preparation, range, sequence, and repetition.

NLGN3 Gene



Addiction and autism may also share genetic connections. One theory posits problems with the Neuroligin 3 gene (NLGN3), a candidate gene for autism, which is active at both GABA and glutamate synapses. GABA is a major inhibitory transmitter in the central nervous system (the "brakes," if you will), and glutamate is one of most important excitatory transmitters, involved with learning, memory, and synaptogenesis (the creation of new synapses between neurons). *NLGN3* is highly active in the nucleus accumbens, a region in the ventral striatum that is linked to desire and drug use; a major player in addiction. Research continues in the role of mutations of the gene in ASD and addiction.

Fragile X Gene



Still another piece of the puzzle is something called "fragile X syndrome (FXS)," which affects both removal and addition of synapses, important both in ASD and addiction. The gene, which resides on the X chromosome, is responsible for fragile X is called FMR1. Everyone has the FMR1 gene on their X chromosome. FMR1 makes a protein, the unfortunately-named fragile X mental retardation protein (FMRP), which is important in brain development, but people with FXS have a deficiency of this protein.

Fragile X creates disruptive changes for many types of learning:

1. Forming new memories.

2. Revising older memories

3. Dependence on addictive substances

ASD is likely caused by connectivity issues brought about by ineffective synaptic pruning in development, which may cause:

1. Sensory hypersensitivity

2. Social overstimulation

3. Problems with reward processing

(Think of a radio or TV that's been tuned somehow to several different channels at once; what you'll hear is the gibberish caused by coexisting, competing, poorly-attenuated signals.)

FMRP helps transport materials to the synapse and controls which synapses are stabilized and which are eliminated. Loss of FMRP leads to Fragile X Syndrome, a common cause of ASD. FMRP may be involved in drug addiction, but what's not clear is whether it's a risk factor or a protective factor. The defect, or mutation, on the FMR1 gene interferes with protein synthesis.

Arnevik and Helverschou note that screening for SUD in people with ASD is not yet a part of routine clinical assessment. They further assert that, "little is known about successful interventions for individuals with ASD and SUD; in fact, typical interventions for SUD may be particularly unsuitable for people with ASD. Thus, although group treatment is often the main focus of substance abuse interventions in residential or community-based facilities, '12-Step' programs and self-help groups, forced involvement in group sessions may be anxiety and anger provoking for ASD patients and may precipitate drop out or rejection from the group.

“The failure of individuals with ASD to socialize and participate in group activities may be interpreted as a lack of motivation or cooperation, while their social aloofness may induce frustration and helplessness both in other patients and in clinicians, which can result in a failure to receive, or respond to, treatment among individuals with ASD and may induce feelings of failure and exclusion that risk worsening the patients’ symptoms and their drug abuse.”

4. Adapting Addiction Treatment for ASD

It’s understood that interaction with people with ASD has to be accomplished carefully, respecting the inherent instability they present. The first step may well be increasing ASD awareness among counseling staff. Interestingly enough, this same fragility is also characteristic of people with SUD who aren’t on the autism spectrum. The philosophy, mindset, and style of evidence-based addiction treatment, with its emphasis on trauma-informed care, fits right in with our enhanced appreciation for the fragility of people in early addiction recovery.

1. Cognitive behavioral therapy remains useful for this population, but some adjustments may be necessary, including aligning cognitive components to each patient’s language level and style.

2. Treatment is highly individualized.

3. Sessions are more structured

4. Sessions are distinctly directive.

5. Sessions are of longer duration

6. Treatment term is longer

7. Sessions emphasize emotional education and social training, anxiety reduction, and stress management.

8. Visual strategies, such as drawing and cartoons, are helpful.

9. The therapist often helps secure housing and employment

10. Coach the patient in creating drug-free social contacts and leisure activities

11. Individual therapy is often preferred over group therapy.

The therapeutic environment can immediately set the tone contributing to the level of success with working with those with both ASD and SUD. It will benefit from attention to the following, occasionally-overlooked characteristics to which both these populations are known to be exquisitely sensitive:

1. Calmness and quietude

2. Neat, simple office layout

3. Relaxing sound design

4. Predictable routines, activities, and interactions

It can often be difficult for the therapist to avoid getting caught up in the intensity manifested by someone with ASD. Awareness of the panoply of problems and challenges faced by people with ASD and SUD behooves therapists to guard against becoming too emotionally solicitous of their patients. A good rule of thumb to follow: At the end of the workday, “leave it at the office.” It’s not at all callous to remind therapists to go home and have a life!

As clinicians, we can’t go too far wrong if we remember that those we serve begin their recovery in what might be thought of as a near-feral state, with impaired ability to process and integrate the myriad sources of sensory information intruding on their consciousness. Nonetheless, one size doesn’t fit all. Individualized treatment planning and interaction will always win the day, and progress is where you find it.

OUTRO

That’s our podcast for today. If you’d like one hour of CE credit for just $5.00, you can go to the School’s website, cadaschool.com, click on “online courses,” and just follow the instructions. Once you pass the post-test, which includes evaluation questions, you’ll be able to download and print your certificate of completion. Be sure and stay in touch with us on Facebook! See you next time!

References

Books

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. Arlington, VA: American Psychiatric Association, 2013.

Kunreuther E and Palmer A: Drinking, Drug Use, and Addiction in the Autism Community. London: Jessica Kingsley, 2017.

Tinsley M: Asperger Syndrome and Alcohol: Drinking to Cope? London: Jessica Kingsley, 2008.

Websites

31 Shareable Fragile X Facts. National Fragile X Foundation. 2019.
https://fragilex.org/understanding-fragile-x/fragile-x-101/31-shareable-fragile-x-facts/

Arnevic EA and Helverschou SB. Autism spectrum disorder and co-occurring substance use disorder: a systematic review. 2016.
https://www.ncbi.nlm.nih.gov/pubmed/27559296

Asperger's and Addiction. Brentwood, TN: Foundations Recovery Network. 2019.
https://www.dualdiagnosis.org/aspergers-addiction/

Helverschou SB, Brunvold AR and Arnevik EA. Treating patients with co-occurring autism spectrum disorder and substance use disorder: a clinical explorative study. 2019.
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6472168/

Sumners C. Can studying autism also yield clues about addiction? Texas A&M Health Sciences Center Vital Record. 2018.
https://vitalrecord.tamhsc.edu/can-studying-autism-also-yield-clues-about-addiction/

Szalavitz M. The Hidden Link between Autism and Addiction. 2017.
https://www.theatlantic.com/health/archive/2017/03/autism-and-addiction/518289/

Videos

Aspergersfrominside: Asperger Syndrome: Rejection Is the Story of My Life (2016)
https://www.youtube.com/watch?v=Bs63f-uFIc0

AspieAnswersAll: The Hidden Link between Autism and Addiction (2017)
https://www.youtube.com/watch?v=7z86KmVy73g

(Videos)

Dailor D: My Brain Works Differently: Autism and Addiction (2018)
https://www.youtube.com/watch?v=FU5nUEfCDdI

Derrat M: The Truth about Asperger's Syndrome – Drugs and Alcohol (2019)
https://www.youtube.com/watch?v=BFQ2VX_U5tY

Soluna M: Asperger's and Substance Abuse. (2016)
https://www.youtube.com/watch?v=I-Mt3VNl8RY