Intergenerational Effects of Alcoholism, Children of Sober Alcoholics: Brain and Behavioral Risks, Interventions, and Implications

Julia Benville, *University of Pennsylvania Center for Neuroscience and Society*

Abstract

There are 18 million children of alcoholics in the United States. Children of Alcoholics have a higher risk of becoming addicted to alcohol based on genetic and neurological predispositions. The personality and age of onset of drinking alcohol are important factors in the development of an Alcohol Use Disorder. I suggest multiple parental interventions, for sober parents, to use during adolescence to lower the risk of an Alcohol Use Disorder, all based on the literature on intergenerational effects of alcoholism.

Introduction

According to the National Association for Children of Alcoholics, there are 18 million children of alcoholics (COA) in the United States, with 11 million of those children under the age of 18 (NACOA). The heritability of developing an Alcohol Use Disorder (AUD), is estimated to be between 50-60% (McGue et al., 1999). There are neurological and genetic predispositions that can put COAs in a vulnerable position to develop an AUD. However, there is still a lack of complete understanding of what puts COAs at this increased risk. It is essential to understand the biological reasons behind this risk, so interventions can be put in place to stop COAs from following their parent in having an AUD. Children and adolescents are the most critical age group to target as many studies have found early alcohol intake to be associated with risk of developing an AUD later in life. Ages 14-19 are the most critical intervention years in terms of alcohol addiction (Nees et al. 2016).

Neurological Predispositions: The two areas of the brain that are most clearly affected by a family history of AUD is the amygdala and the Mesolimbic Dopamine Pathway (including the Ventral Tegmental Area and Nucleus Accumbens which is the reward pathway of the brain). These regions are most well known for emotion and reward processing, respectively. Those with an AUD, tend to have smaller amygdala volumes, and this is also seen in individuals with a family history of AUD, as opposed to those without a family history (Dager et al., 2015.). This is important to understand for COAs because this may contribute to an increased alcoholism risk by creating a faster link to alcohol-related cue-response learning, which is dependent on amygdala volume (Hill et al. in Dager et al., 2015). Another brain region commonly associated with AUD is the Mesolimbic Dopamine Pathway; AUD can affect a variety of genes that can play into this pathway in different ways (Morozova et al., 2012). It is very complicated, and there is no conclusive story. It should be noted there is hypersensitivity of this pathway during adolescence. This could lead to conflicting results because, if the pathway is already activated during adolescence it could appear to be conflating the results. However, there is a genetic basis to what can affect this pathway in COAs leading to an increased risk of an AUD (Nees et al., 2012).

Genetic Predispositions: As previously stated, a large percentage of AUD is heritable. There have been many genes studied looking at alcohol addiction, such as critical signaling molecules including *ANKK1* and a protein-coding gene, *HOMER*1. Variations in these genes seem to be as important as personality in later adolescent years in terms of the likelihood of developing an AUD (Nees et al., 2012). Animal models of addiction have also been useful for identifying genetic predispositions, another gene *AUTS2*, has also been identified as humans, mice, and Drosophila as associated with alcohol sensitivity (Nees et al., 2016). Variations in these genes are making it so that COA's are more likely to develop an AUD.

On a different note, an intriguing genetic model of relapsing and seeking out alcohol involves the epigenetics of how anxiety can impact alcohol abuse. Epigenetics is how gene expression can be modified instead of altering the genetic code. As seen in the figure below adopted from Pandey

et al., the acute use of alcohol relaxes chromatin (the proteins around which DNA is bound), leading to a change in which genes are expressed and a decrease in anxiety. However, this process changes as one moves from acute to chronic use and again once withdrawal occurs. In withdrawal, chromatin is condensed leading to expression in genes and increased anxiety. This adds to the addiction cycle which causes them to go back to the drug to relieve their anxiety (Pandey et al., 2017). Adolescent exposure to alcohol causes higher expression of HDAC2, further condensing chromatin, which maintains this cycle with high stress and drinking behaviors in adulthood (Pandey et al., 2017). This is why alcohol use at a young age can quickly change into an AUD. This, on top of other risk factors for COAs, puts adolescent COAs at high risk of developing an addiction if they experiment with alcohol.

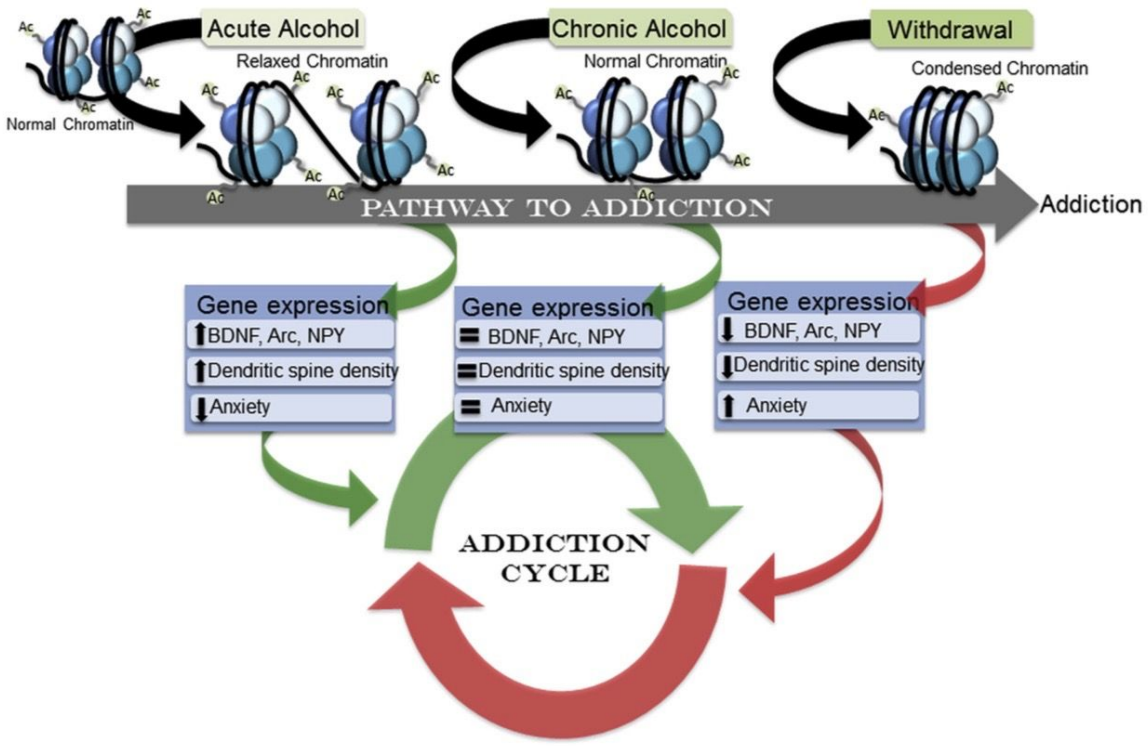

Figure One adapted from Pandey, S. C., Kyzar, E. J., & Zhang, H. (2017). Epigenetic basis of the dark side of alcohol addiction. *Neuropharmacology*, *122*, 74–84. https://doi.org/10.1016/j.neuropharm.2017.02.002

While there are many findings of genetic and neurological predispositions for AUD, there is no cohesive theory of all risk factors for COAs.

Role of Personality: Early adolescent alcohol use is associated with an increased risk of developing an AUD. However, there is a difference in the age of first use of alcohol and the rate of transition to alcohol abuse. Personality traits (such as sensation seeking, impulsivity, extraversion, etc.) are the most crucial factors in explaining early adolescent drinking (with a mean age of 14.37). Variations in the genes mentioned above (*ANKK1* and *HOMER1*) seem to be equally as important as these personality traits in later adolescent drinking developing into an AUD (with a mean age of 16.45) (Nees et al. 2016, Ayer et al., 2011). A later age of onset is associated with a faster transition to an AUD. However, those with an early onset also had an overall higher risk of AUD, but this transition happened more slowly than those with a later age of onset. This difference between early adolescence and late adolescent is partially due to genetics, but might also be due to different drinking patterns, less parental control, and increased peer influence.

Recommendations

Unfortunately, advocating for adolescents to completely abstain from alcohol use during the ages of 14-19 is not a feasible solution. Some children of alcoholics do decide to do this to avoid the risk, but this is not a solution that is working for all 18 million COAs in the United States. The majority of recommendations for COAs are parental interventions. It should be noted; however, these suggestions are for parents who are not currently struggling with AUD and are sober and who can put the majority of their focus on deterring their children from alcohol addiction. A parent presently working towards their soberly would need to focus on their behavior first and other traumas their children would be experiencing, but that is not the focus of these suggestions.

The parental interventions mentioned here are all based on a strong relationship with one's child and being aware of a child's actions during adolescence. The easiest solution can be for a parent to talk to their child about their addiction, and the risks that follow the child, that I have laid out above. It is best if an open dialogue can come from these conversations, so a COA is aware of their increased risk and what could happen if they start drinking. If possible delaying past ages 14-19 would be the best solution. But, merely trying to delay alcohol use in adolescence is not necessarily useful, because children can develop alcohol addictions at a faster rate (differences between 14.37 and 16.45). Nees et al., recommends secondary prevention by parents at the stage after alcohol use onset but before the transition to an AUD, because it is not possible to dissuade all children from ever drinking alcohol during adolescence. This can be achieved with parental monitoring, to decrease substance use after initiation if and when it occurs. (Oxford et al., 2001). It is essential during a COAs childhood to create a strong bond, which is called indirect control because it can actively deter children from drug and alcohol use later in life because the child does not want to let down their parents (Oxford et al., 2001).

Conclusion

Children of Alcoholics are at an increased risk of becoming an alcoholic, so there are specific precautions that parents can use to give their child the best chance of not becoming addicted to alcohol. Both neuroscientific and behavioral factors play into the risk of first use of alcohol and transition into an AUD. But, with the help of parental intervention, these risks can be lowered. However, they are interventions that last throughout all childhood and adolescent years and can require parents who want to be very involved in their child's social life.

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