

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

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Abstract

There are 18 million children of alcoholics in the United States. Children of Alcoholics have a higher risk of becoming addicted to alcohol because of genetic and neurological predispositions. I will discuss the risk factors for children of alcoholics in depth. The personality and age of onset of drinking alcohol are important factors in the development of an Alcohol Use Disorder. Parental interventions for sober parents to use during their child's adolescence are suggested to lower the risk of an Alcohol Use Disorder founded on the literature of 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, and 11 million of those children are under the age of 18 (NACOA). The heritability of developing an Alcohol Use Disorder (AUD), or how much genetics effect the likelihood of developing an AUD in COAs is estimated to be between 50% and 60% (McGue et al., 1999, p. 109). There are neurological and genetic predispositions that can put COAs in a vulnerable position to develop an AUD, but these predispositions are not completely understood as of yet. It is essential to understand the biological reasons behind this risk so interventions can be put in place to stop COAs from developing an AUD as their parent did. Without interventions, the cycle of passing an AUD from parent to child can continue.

Adolescents are the most critical age group to target, as many studies have found early alcohol intake to be associated with higher risk of developing an AUD later in life, specifically ages 14-19 are the most critical years for alcohol addiction intervention (Nees et al., 2016, p. 5). Neurological Predispositions: The two areas of the brain that are most clearly affected by a family history of AUD are the amygdala and the Mesolimbic Dopamine Pathway (MDP: including the Ventral Tegmental Area and Nucleus Accumbens which is the reward pathway of the brain). These regions (amygdala and MDP) are most well-known for emotion and reward processing, respectively. Those with an AUD tend to have smaller amygdala volumes, which is also seen in individuals with a family history of AUD (Dager et al., 2015, p. 413). Those without a family history, however, do not tend to have smaller amygdala volumes (Dager et al., 2015, p.413). This is important to understand for COAs because this may contribute to an increased risk of alcoholism 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, p. 413). Another brain region commonly associated with AUD is the Mesolimbic Dopamine Pathway: AUD can affect a variety of genes that influence this pathway in different manners, although no conclusive story has yet been found (Morozova et al., 2012, p. 2). It should be noted that 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 conflate the results. There is also a genetic basis to what can affect this pathway in COAs leading to an increased risk of an AUD (Nees et al., 2012, p. 993).

Genetic Predispositions: As previously stated, a large percentage of AUD is heritable. Many genes have been studied that have a basis in alcohol addiction, such as critical signaling molecules including ANKK1 and a protein-coding gene, HOMER1. 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, p. 991). Animal models of addiction have also been useful for identifying genetic predispositions: another gene, AUTS2, has also been identified in humans, mice, and Drosophila as associated with alcohol sensitivity (Nees et al., 2016, p. 5).

Variations in these genes allow for the more likely development of an AUD among COAs.

On a different note, an intriguing genetic model of relapsing and pursuing alcohol involves the epigenetics of how anxiety can impact alcohol abuse. Epigenetics is how gene expression can be modified in a manner other than the alteration of 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 of which genes are expressed, resulting in a decrease in anxiety. This process changes, however as one progresses from acute to chronic use and again once withdrawal occurs. In withdrawal, chromatin is condensed leading to more expression in genes and increased anxiety. This adds to the addiction cycle by causing those with alcohol use disorders to go back to the drug to relieve their anxiety (Pandey et al., 2017, p. 78). Adolescent exposure to alcohol causes higher expression of HDAC2, an enzyme that further condenses chromatin, which maintains this cycle through high stress and drinking behaviors in adulthood (Pandey et al., 2017, p. 78). This is an explanation for how 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.

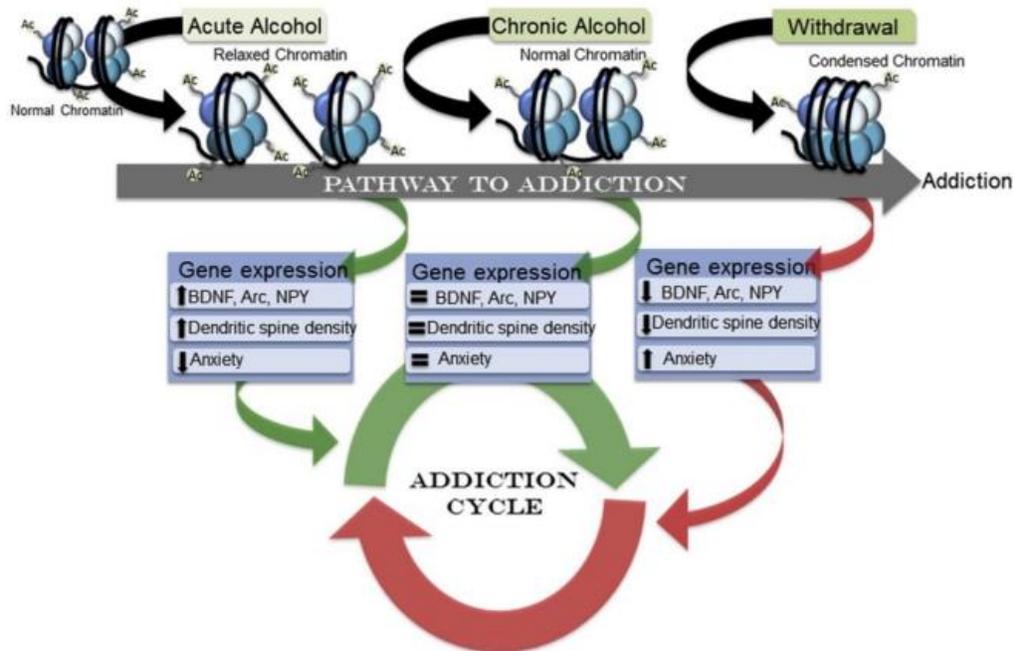


Figure One adapted from Pandey, S. C., Kyzar, E. J., & Zhang, H. (2017). Epigenetic basis of the dark side of alcohol addiction. *Neuropharmacology*, 122, 74–84, with permission from Elsevier.

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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 (Nees et al., 2016, p. 5). Additionally, there is a relationship between 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 p. 4, Ayer et al., 2011, p. 1302). 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 slower than those with a later age of onset. This difference between early adolescence and late adolescence 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 abstain to avoid the risk, but this is not a solution that is practical for all 18 million COAs in the United States. The majority of recommendations for COAs involve parental interventions. Interventions in the literature are largely based on parental interventions or community and school based intervention efforts. However, parental interventions have proven to be the most beneficial in reducing the prevalence of development of an AUD, as school based interventions are often too short in duration (Kumpfer 1999, p.46). School based efforts as well tend to focus on COAs whose parents are abusing alcohol. It should be noted; the suggestions described here are for parents who are not currently struggling with AUD, are sober, and who can put the majority of their focus on deterring their children from alcohol addiction. A parent presently working towards their sobriety would need to focus on their behavior and other traumas their children would be experiencing, but that is not the focus of these suggestions.

The parental interventions mentioned here are all grounded on a strong relationship with one's child and being aware of the child's actions during adolescence. The easiest solution involves a parent talking to their child about their addiction and the aforementioned risks that follow the child. It is best if an open dialogue can evolve from these conversations so a COA is aware of their increased risk and what could happen if they start drinking. The literature on which parental interventions serve the best outcomes is conflicting. Sharmin et al. found that when parents set rules regarding alcohol use with children, they are less likely to develop risky

drinking behaviors and other alcohol-related problems (Sharmin et al., 2017, p. 11). The authors, however, stated there was chance of confounding bias and limitations in their study design. If possible, delaying experimentation with alcohol 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 (progressing from use to an AUD is faster in ages closer to 16.45 than 14.37). Nees et al. recommends secondary prevention by parents at the stage after their child's onset of alcohol use but before the transition to an AUD, because it is not possible to dissuade all children from 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, p. 609). It is essential during a COA's childhood to create a strong bond with their parents. This is called indirect control because it can actively deter a child from drug and alcohol use later in life, as the child does not want to disappoint their parents (Oxford et al., 2001, p.601). Overall there has not been enough longitudinal research on interventions with COAs to explicitly state the most beneficial intervention technique, however the research on parental interventions have been promising thus far.

Conclusion

Children of Alcoholics are at an increased risk of becoming alcoholics, but there are specific precautions that parents can utilize 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. With the help of parental intervention, these risks can be lowered.

These are interventions that last throughout childhood and adolescence and require parents who want to be very involved in their child's social life.

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References

- Ayer, L., Rettew, D., Althoff, R. R., Willemsen, G., Ligthart, L., Hudziak, J. J., & Boomsma, D. (2011). Adolescent personality profiles, neighborhood income, and young adult alcohol use: A longitudinal study. *Addictive Behaviors*, 36(12), 1301–1304. <https://doi.org/10.1016/j.addbeh.2011.07.004> (<https://doi.org/10.1016/j.addbeh.2011.07.004>)
- Behrendt, S., Wittchen, H.-U., Höfler, M., Lieb, R., & Beesdo, K. (2009). Transitions from first substance use to substance use disorders in adolescence: Is early onset associated with a rapid escalation? *Drug and Alcohol Dependence*, 99(1–3), 68–78. <https://doi.org/10.1016/j.drugalcdep.2008.06.014> (<https://doi.org/10.1016/j.drugalcdep.2008.06.014>)
- Dager, A. D., McKay, D. R., Kent, J. W., Curran, J. E., Knowles, E., Sprooten, E., Glahn, D. (2015). Shared Genetic Factors Influence Amygdala Volumes and Risk for Alcoholism. *Neuropsychopharmacology*, 40(2), 412–420. <https://doi.org/10.1038/npp.2014.187> (<https://doi.org/10.1038/npp.2014.187>)
- Home. (n.d.). Retrieved April 19, 2019, from NACOA website: <https://nacoa.org/>
- How to talk to your kids about family addiction. (n.d.). Retrieved April 19, 2019, from *Washington Post* website: <https://www.washingtonpost.com/news/parenting/wp/2017/02/03/how-to-talk-to-yourkids-about-family-addiction/> (<https://www.washingtonpost.com/news/parenting/wp/2017/02/03/how-to-talk-to-yourkids-about-family-addiction/>)
- Kranzler, H. R., Zhou, H., Kember, R. L., Vickers Smith, R., Justice, A. C., Damrauer, S., Gelernter, J. (2019). Genome-wide association study of alcohol consumption and use disorder in 74,424 individuals from multiple populations. *Nature Communications*, 10(1). <https://doi.org/10.1038/s41467-019-09480-8> (<https://doi.org/10.1038/s41467-019-09480-8>)
- Kumpfer, K. L. (1999). Outcome Measures of Interventions in the Study of Children of Substance-abusing Parents. *Pediatrics*, 103(Supplement 2), 1128–1128.
- McGue, M. (1999). The Behavioral Genetics of Alcoholism. *Current Directions in Psychological Science*, 8(4), 109–115. <https://doi.org/10.1111/1467-8721.00026> (<https://doi.org/10.1111/1467-8721.00026>)
- Morozova, T. V., Goldman, D., Mackay, T. F., & Anholt, R. R. (2012). The genetic basis of alcoholism: multiple phenotypes, many genes, complex networks. *Genome Biology*, 13(2), 239. <https://doi.org/10.1186/gb-2012-13-2-239> (<https://doi.org/10.1186/gb-2012-13-2-239>)
- Nees, F., Tzschoppe, J., Patrick, C. J., Vollstädt-Klein, S., Steiner, S., Poustka, L., Flor, H. (2012). Determinants of Early Alcohol Use In Healthy Adolescents: The Differential Contribution of Neuroimaging and Psychological Factors. *Neuropsychopharmacology*, 37(4), 986–995. <https://doi.org/10.1038/npp.2011.282> (<https://doi.org/10.1038/npp.2011.282>)
- Nees, F et al. (2016). Prediction of alcohol drinking in adolescents: Personality-traits, behavior, brain responses, and genetic variations in the context of reward sensitivity | Elsevier Enhanced Reader. (n.d.). <https://doi.org/10.1016/j.biopsycho.2016.05.002> (<https://doi.org/10.1016/j.biopsycho.2016.05.002>)
- Oxford, M., Oxford, M. L., Harachi, T. W., Catalano, R. F., & Abbott, R. D. (2001). Preadolescent Predictors of Substance Initiation: A Test of Both the Direct and Mediated Effect of Family Social Control Factors on Deviant Peer Associations and Substance Initiation. *The American Journal of Drug and Alcohol Abuse*, 27(4), 599–616. <https://doi.org/10.1081/ADA-100107658> (<https://doi.org/10.1081/ADA-100107658>)
- 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> (<https://doi.org/10.1016/j.neuropharm.2017.02.002>)
- Sharmin, S., Kypri, K., Khanam, M., Wadolowski, M., Bruno, R., Attia, J., Mattick, R. P. (2017). Effects of parental alcohol rules on risky drinking and related problems in adolescence: Systematic review and meta-analysis. *Drug and Alcohol*

