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Impact of the Number of Parents With Alcohol Use Disorder on Alcohol Use Disorder in Offspring: A Population-Based Study

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ABSTRACT

Objective: Although parental alcohol use disorder (AUD) increases risk for alcohol problems in offspring, no studies have evaluated the odds of AUD in offspring based on the number of biological parents with AUD (0, 1, or 2) in a population-based national sample. The purpose of this study was to investigate the relationship between the number of AUD parents and prevalence of AUD in offspring.

Method: This study utilized data from the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions, which assessed AUD using the Alcohol Use Disorder and Associated Disabilities Interview Schedule–*DSM-IV* Version (main outcome variable). We analyzed the sample (n = 40,374) to investigate the effect of the number of AUD parents on lifetime AUD in offspring. In a subgroup analysis, gender differences were examined.

Results: 22% of adults in the United States had at least 1 biological parent with AUD. Compared with offspring of non-AUD parents, offspring of 1 AUD parent had a 2.5-fold increase (AOR = 2.51; 95% CI, 2.38–2.66) and offspring of 2 AUD parents had a 4.4-fold increase (AOR = 4.44; 95% CI, 3.93–5.02) in the odds of lifetime AUD. Each additional AUD parent increased the odds of AUD in offspring in an additive pattern. Female offspring were more vulnerable to the impact of parental AUD than male offspring (OR = 1.17 in offspring of 1 AUD parent; OR = 1.48 in offspring of 2 AUD parents).

Conclusions: Offspring of AUD parents had heightened odds of lifetime AUD, with an additive parental effect. Awareness of this risk can be useful for clinicians to educate individuals with AUD parents about prevention and intervention.

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Alcohol use disorder (AUD) is a serious public health problem in the United States[1] and worldwide.[2] Previous studies have indicated that parental AUD increases risk for AUD in offspring.[3–9] Both genetic[10] and environmental[11] risk factors are associated with higher odds of AUD in offspring. Twin studies have demonstrated that the heritability of alcohol dependence is 50%–60%.[12–14]

Patients and their family members often ask clinicians about the odds and probability of developing AUD when their father, mother, or both parents have alcohol problems. Studies on the number of AUD parents (0, 1, or 2)[15–17] can help us further understand the relationship between parental and offspring AUD. This information would be valuable in providing preventive and clinical interventions. A recent Danish cohort study answered this question and reported that

Clinical Points

- Twenty-two percent of adults in the United States have at least 1 biological parent with alcohol use disorder (AUD).
- The odds of lifetime AUD are 2.5 times higher in offspring of 1 AUD parent and 4.4 times higher in offspring of 2 AUD parents compared to offspring of non-AUD parents.
- Although male offspring have higher prevalence rates of AUD than female offspring, female offspring are more vulnerable to the impact of parental AUD than male offspring.

paternal AUD (odds ratio [OR] = 1.99; 95% CI, 1.54–2.68) and maternal AUD (OR = 1.96; 95% CI, 1.42–2.71) increased the odds of AUD in offspring.[18] However, this study did not examine odds of AUD among offspring of 2 AUD parents. In a community study of adolescents and young adults in Munich, Germany,[19] the odds of alcohol dependence were 2.14 times higher in offspring of 1 AUD parent (OR = 2.14; 95% CI, 1.46–3.14) and 2.75 times higher in offspring of 2 AUD parents (OR = 2.75; 95% CI, 1.42–5.31) compared to offspring of non-AUD parents. The difference was not significant between offspring of 1 AUD parent and those of 2 AUD parents on the 95% CI. This study, however, may not represent all population groups because many individuals develop alcohol dependence long after adolescence and young adulthood. In addition to these 2 important studies, clinical studies have shown that patients with 2 alcoholic parents manifested more alcohol-related symptoms and problems than patients with no alcoholic parents.[20,21] However, clinical samples tend to represent patients with severe or treatment refractory AUD and therefore may not be generalizable to the population at large. Currently, no studies have examined the association between the number of AUD parents (0, 1, or 2) and odds of AUD in offspring in a population-based national sample.

The primary goal of this study was to investigate the relationship between the number of AUD parents and AUD in offspring in nationally representative data. Our hypothesis was that prevalence of AUD in offspring would be higher in offspring of 1 AUD parent and the highest in offspring of 2 AUD parents as compared to offspring of non-AUD parents. We also examined gender differences among offspring and their AUD parents in this association.

METHOD

Study Sample

A survey sample consisted of participants in the 2001–2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC),[22,23] which was designed, sponsored, and conducted by the National Institute on Alcohol Abuse and Alcoholism. The NESARC is a nationally representative household survey of the US civilian noninstitutionalized population. Nationwide face-to-face personal interviews were conducted with 43,093 participants aged ≥ 18 years by the US Census Bureau in 2001–2002. The overall response rate was 81%. The NESARC procedures were reviewed and approved by the US Census Bureau and the US Office of Management and Budget. All NESARC participants provided informed consent. The survey provides data on alcohol and drug use, psychiatric classification of substance use disorders and other psychiatric disorders, alcohol treatment utilization, sociodemographic information, and family history of AUD.

Of 43,093 individuals in the NESARC, 40,374 participants (93.7%) provided data on parental history of AUD for both parents. The current study was based on the subsample of these 40,374 participants. The mean (SD) age of the study sample (n = 40,374) was 46.4 (18.2) years (mode = 40). The majority of this study sample was female (57.0%), white (57.6%), and married/cohabiting (51.8%).

Data Assessment

Parental history of alcohol use disorder. Parental history of AUD was determined as "yes," "no," or "don't know" by face-to-face personal interviews with the following questions: "Has your blood or natural father been an alcoholic or problem drinker at any time in his life?" and "Has your blood or natural mother been an alcoholic or problem drinker at any time in her life?" For these questions, an alcoholic or problem drinker was defined and explained to participants by interviewers as follows: "By alcoholic or problem drinker, I mean a person who has physical or emotional problems because of drinking; problems with a spouse, family, or friends because of drinking; problems at work or school because of drinking; problems with the police because of drinking—like drunk driving; or a person who seems to spend a lot of time drinking or being hungover." The reliability of AUDADIS parental history variables is good to excellent.[24,25]

Alcohol use disorder in offspring. To be classified with lifetime AUD, participants (ie, offspring) were required to have alcohol abuse, alcohol dependence, or both at any time during their lives. Diagnosis of alcohol abuse required at least 1 of the 4 abuse criteria. Diagnosis of alcohol dependence required at least 3 of the 7 dependence criteria in any 12-month period. Diagnoses of alcohol abuse and alcohol dependence were made with the Alcohol Use Disorder and Associated Disabilities Interview Schedule–*DSM-IV* Version (AUDADIS-IV),[26] which was developed based on the *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition (*DSM-IV*).[27] The AUDADIS-IV has shown to be reliable and valid for the diagnosis of alcohol use disorder.[24,25]

Statistical Analysis

Three offspring groups were identified based on the number of biological parents with AUD: non-AUD parents, 1 AUD parent, and 2 AUD parents. The 3 groups were compared for sociodemographic characteristics using χ^2 analysis for categorical variables. The prevalence rates of lifetime AUD in the 3 offspring groups were calculated using cross-tabulations.

We conducted logistic regression and tetrachoric correlations to evaluate the effect of the number of AUD

Table 1. Sociodemographic Characteristics of 3 Offspring Groups Based on the Number of Parents With Alcohol Use Disorder (AUD)

Offspring Characteristic	No. of Parents With AUD: None (n = 31,470), n (%)	1 (n = 7,639), n (%)	2 (n = 1,265), n (%)	χ^2	*df*	*P*
AUD Parent						
AUD father	0 (0)	6,784 (88.8)	1,265 (100)			
AUD mother	0 (0)	855 (11.2)	1,265 (100)			
Gender				71.6	2	< .001
Male	13,885 (44.1)	3,002 (39.3)	481 (38.0)			
Female	17,585 (55.9)	4,637 (60.7)	784 (62.0)			
Age, y				600.5	6	< .001
18–29	6,106 (19.4)	1,705 (22.3)	300 (23.7)			
30–44	9,411 (29.9)	2,661 (34.8)	471 (37.2)			
45–64	9,177 (29.2)	2,437 (31.9)	420 (33.2)			
≥ 65	6,776 (21.5)	836 (10.9)	74 (5.8)			
Race/ethnicity				281.0	8	< .001
White	17,983 (57.1)	4,423 (57.9)	830 (65.6)			
Hispanic/Latino	6,079 (19.3)	1,588 (20.8)	159 (12.6)			
Black	5,713 (18.2)	1,318 (17.3)	211 (16.7)			
Native American	496 (1.6)	209 (2.7)	54 (4.3)			
Asian/Pacific Islander	1,195 (3.8)	101 (1.3)	11 (0.9)			
Education, y				129.4	6	< .001
0–11	5,565 (17.7)	1,339 (17.5)	254 (20.1)			
12	9,010 (28.6)	2,301 (30.1)	383 (30.3)			
13–15	9,027 (28.7)	2,472 (32.4)	409 (32.3)			
≥ 16	7,868 (25.0)	1,527 (20.0)	219 (17.3)			
Personal income ($)				38.1	6	< .001
0–19,999	15,137 (48.1)	3,771 (49.4)	668 (52.8)			
20,000–34,999	7,244 (23.0)	1,823 (23.9)	280 (22.1)			
35,000–59,999	5,731 (18.2)	1,369 (17.9)	219 (17.3)			
≥ 60,000	3,358 (10.7)	676 (8.8)	98 (7.7)			
Marital status				5.1	4	.277
Married/cohabitation	16,356 (52.0)	3,921 (51.3)	649 (51.3)			
Widowed/divorced/separated	8,085 (25.7)	1,923 (25.2)	332 (26.2)			
Never married	7,029 (22.3)	1,795 (23.5)	284 (22.5)			

Table 2. Prevalence Rate and Adjusted Odds Ratios (AORs) of Lifetime Alcohol Use Disorder (AUD) in 3 Offspring Groups Based on the Number of Parents With AUD

No. of AUD Parents	Prevalence of AUD, % (n)	OR (95% CI)	AOR[a] (95% CI)
None (n = 31,470)	23.1 (7,268)	1	1
1 (n = 7,639)	40.4 (3,085)	2.26 (2.14–2.38)*	2.51 (2.38–2.66)*
2 (n = 1,265)	54.3 (687)	3.96 (3.53–4.43)*	4.44 (3.93–5.02)*

[a]Adjusted for gender, age, race, education, and personal income.
*$P < .001$.

parents on lifetime AUD in offspring (main outcome variable). First, unadjusted ORs and 95% CIs were calculated. Second, the 5 sociodemographic variables found to be statistically significant on the initial χ^2 analyses (gender, age, race, education, and personal income) were entered into the logistic regression analysis to estimate adjusted odds ratios (AORs) and 95% CIs.

For subgroup analysis stratified by gender, we first split the sample by gender and repeated logistic regression to examine the effect of the number of AUD parents on offspring with lifetime AUD in the 3 offspring groups. Male and female offspring were compared with same gender offspring in the 3 groups. Second, without splitting the sample by gender, we calculated interaction effects between gender and number of AUD parents in logistic regression models to examine if the gender variable modified the effect of the number of AUD parents on AUD in offspring. Statistical analyses were performed with IBM SPSS Statistics, version 19 (IBM SPSS; Armonk, New York).

RESULTS

Sample Characteristics

Table 1 presents sociodemographic characteristics of 3 offspring groups based on the number of biological parents with AUD. Offspring of non-AUD parents (77.9%) comprised the largest group, followed by offspring of 1 AUD parent (18.9%) and offspring of 2 AUD parents (3.1%). When the 3 groups were compared, all sociodemographic variables (gender, age, race, education, and personal income) showed a significant difference except marital status. Offspring of 2 AUD parents were more likely than those of non-AUD parents to be associated with female gender, younger age, Native American, lower education, and lower personal income. Offspring of 2 AUD parents were less likely than those of non-AUD parents to be associated with Asian/Pacific Islander and Hispanic/Latino.

Lifetime Alcohol Use Disorder in Offspring

Table 2 summarizes prevalence, unadjusted ORs, AORs, and 95% CIs of AUD in the 3 offspring groups. Prevalence of lifetime AUD in offspring gradually increased from 23.1%

Table 3. Prevalence Rate and Adjusted Odds Ratios (AORs) of Lifetime Alcohol Use Disorder (AUD) in Male and Female Offspring Groups Based on the Number of Parents With AUD

	Male Offspring (n = 17,368)		Female Offspring (n = 23,006)	
No. of AUD Parents	AUD, % (n)	AOR[a] (95% CI)	AUD, % (n)	AOR[a] (95% CI)
None (n = 31,470)				
Male offspring (n = 13,885)	35.6 (4,947)	1	…	…
Female offspring (n = 17,585)	…	…	13.2 (2,321)	1
1 (n = 7,639)				
AUD father only (n = 6,784)				
Male offspring (n = 2,694)	56.6 (1,525)	2.37 (2.18–2.59)*	…	…
Female offspring (n = 4,090)	…	…	28.7 (1,173)	2.57 (2.36–2.80)*
AUD mother only (n = 855)				
Male offspring (n = 308)	59.4 (183)	2.30 (1.81–2.90)*	…	…
Female offspring (n = 547)	…	…	37.3 (204)	3.30 (2.73–3.80)*
2 (n = 1,265)				
Male offspring (n = 481)	67.8 (326)	3.51 (2.88–4.27)*	…	…
Female offspring (n = 784)	…	…	46.0 (361)	5.26 (4.50–6.15)*

[a]Adjusted for gender, age, race, education, and personal income.
*$P < .001$.

Table 4. Interaction Between Offspring Gender and Number of Parents With Alcohol Use Disorder (AUD) on Offspring AUD Using Logistic Regression Models

No. of Parents With AUD	OR	95% CI
None (n = 31,470) vs 1 (n = 7,639)		
Male offspring (n = 16,887)	1	Reference
Female offspring (n = 22,222)	1.17*	1.04–1.30
None (n = 31,470) vs 2 (n = 1,265)		
Male offspring (n = 14,366)	1	Reference
Female offspring (n = 18,369)	1.48*	1.16–1.89

*$P < .01$.

in offspring of non-AUD parents to 40.4% in offspring of 1 AUD parent and 54.3% in offspring of 2 AUD parents. Compared to offspring of non-AUD parents, offspring of 1 AUD parent had 2.51-fold increased odds of lifetime AUD (AOR = 2.51). Offspring of 2 AUD parents had 4.44-fold increased odds of lifetime AUD (AOR = 4.44) compared with offspring of non-AUD parents. In addition, tetrachoric correlations showed a significant positive relationship between number of AUD parents and AUD in offspring of 1 AUD parent ($r = 0.278$, $P < .001$) and in offspring of 2 AUD parents ($r = 0.368$, $P < .001$).

Gender Differences

As shown in Table 3, the study data were stratified by gender. Compared to male offspring of non-AUD parents, male offspring of AUD-fathers-only had 2.37-fold increased odds of lifetime AUD (AOR = 2.37). The increased odds of AUD was similar to male offspring of AUD-mothers-only (AOR = 2.30). Male offspring of 2 AUD parents had 3.51-fold increased odds of lifetime AUD (AOR = 3.51) compared to male offspring of non-AUD parents.

Parental AUD increased the odds of offspring AUD even greater in female offspring. Female offspring of AUD fathers only (AOR = 2.57), female offspring of AUD mothers only (AOR = 3.30), and female offspring of 2 AUD parents (AOR = 5.26) had higher odds of lifetime AUD than female offspring of non-AUD parents. Compared to offspring of non-AUD parents in each gender, the odds of lifetime AUD were substantially higher in female offspring of 2 AUD parents (AOR = 5.26; 95% CI, 4.50–6.15) than in male offspring of 2 AUD parents (AOR = 3.51; 95% CI, 2.88–4.27). Table 4 describes the interaction effects between gender and number of AUD parents on AUD in offspring. The interaction was significant. Compared to offspring male gender, offspring female gender increased the odds of AUD in offspring of 1 AUD parent (OR = 1.17; 95% CI, 1.04–1.30) and in offspring of 2 AUD parents (OR = 1.48; 95% CI, 1.16–1.89).

DISCUSSION

This study is the first to use a population-based national sample to investigate the odds of AUD in offspring based on the number of AUD parents. The findings indicated that (1) 22% of adults in the United States had at least 1 biological parent with AUD, (2) the odds of lifetime AUD were 2.51 times higher in offspring of 1 AUD parent and 4.44 times higher in offspring of 2 AUD parents compared to offspring of non-AUD parents, (3) each additional AUD parent increased the odds of offspring AUD in an additive pattern, and (4) female offspring were more vulnerable to the impact of parental AUD than male offspring.

The finding that 22% of adults had AUD parents was similar to an earlier report estimating that 28.6% of children in the United States are exposed to AUD in their family.[28] Potentially, our rate of 22% could have been higher if the NESARC had included individuals in institutional settings, such as hospitals and jails, where many adults with AUD may be found. Among participants with 1 AUD parent, the number of those with AUD fathers (n = 6,784) was 8 times higher than the number of those with AUD mothers (n = 855). This finding is consistent with the high prevalence of AUD among men as compared to women.[29,30]

The 3 offspring groups differed in sociodemographic characteristics. The overall age distribution suggests that survival past age 65 decreases progressively with more AUD parents. Native Americans were more negatively impacted by parental AUD, perhaps due to greater poverty among Native Americans.[31] Conversely, Asians were less affected by parental

AUD, perhaps due to genetic protection related to alcohol-metabolizing genes.[32] Offspring with any AUD parent were less apt to complete college, and those in the highest income range ($60,000 and higher) had fewer AUD parents.

The main findings of the study were the increased odds of lifetime AUD among offspring of 1 AUD parent (AOR = 2.51; 95% CI, 2.38–2.66) and those of 2 AUD parents (AOR = 4.44; 95% CI, 3.93–5.02). Our findings were stronger than those in the Munich community study[19] in which the odds of offspring alcohol dependence were increased in offspring with 1 AUD parent (OR = 2.14; 95% CI, 1.46–3.14) and those with 2 AUD parents (OR = 2.75; 95% CI, 1.42–5.31) in a threshold pattern. In the community study, the odds of offspring alcohol dependence did not differ statistically between 1 AUD parent and 2 AUD parents. One possible explanation could be younger age of the community study sample (aged 14–24 years) compared to our sample (mean age of 46.4 years), suggesting that young offspring of AUD parents may develop AUD later in their lives more often than other young offspring of non-AUD parents who do not carry a genetic risk of AUD. Two AUD parents increase offspring AUD because of higher genetic risk, environmental sequelae, and parental assortative mating.[33,34] When parental assortative mating occurs, high-risk genes are transmitted from 2 AUD parents to their offspring. Another possibility could be the propensity of a sober partner to become alcoholic as a result of exposure to the spouse's drinking, which in turn could worsen the home environment in which the offspring grows up. Regarding offspring of 1 AUD parent, our results were similar to that of the Danish study[18] in which offspring with an AUD parent had 2-fold increased odds of developing AUD. Regardless, these studies demonstrated higher odds of AUD in offspring of AUD parents.

Our data also showed the additive parental effect on offspring AUD, indicating that each addition of an AUD parent increased the odds of offspring AUD. Compared to 1 AUD parent (AOR = 2.51), 2 AUD parents (AOR = 4.44) significantly increased the odds of offspring AUD. This finding suggests that doubling the number of AUD parents almost doubled the number of offspring with AUD. Alternatives would be the threshold effect as shown in the Munich community study,[19] in which the increase would occur from non-AUD parents to 1 AUD parent, and then level off from 1 AUD parent to 2 AUD parents. This threshold pattern would indicate that the influence for offspring AUD would be more environmental, with the 1-parent condition and the 2-parent condition having the same or similar effect. A geometric increase from non-AUD parents to 1 AUD parent and then a much larger increase from 1 AUD parent to 2 AUD parents might suggest a combination of both genetic and environmental factors creating a very great likelihood of AUD in the offspring. The additive parental effect that we observed seems most consistent with genetic effects accounting for the increase but with minimal environmental pathogenicity from 1 AUD parent to 2 AUD parents.

Although male offspring had higher prevalence rates of AUD than female offspring in all groups, this study identified that female offspring were more vulnerable to the impact of parental alcohol problems. Female offspring of 2 AUD parents had higher odds of AUD (AOR = 5.26; 95% CI, 4.50–6.15) than male offspring of 2 AUD parents (AOR = 3.51; 95% CI, 2.88–4.27). This finding was consistent with previous studies demonstrating that parental AUD increased the risk of AUD more in female than male offspring,[35–37] although other studies reported no gender differences.[19,38] Several risk factors associated with female offspring AUD have been identified, such as negative affectivity, severe physical punishment, and childhood stressors.[39] Offspring AUD was not affected by alcoholic parental gender with regard to the 95% CI, but AUD mother (AOR = 3.30; 95% CI, 2.73–3.80) tended to increase AUD more than AUD father (AOR = 2.57; 95% CI, 2.36–2.80) in female offspring. There are several studies demonstrating that maternal AUD causes higher rates of offspring AUD than paternal AUD.[40–42] The increased risk from maternal AUD could be due to intrauterine effects of alcohol on the fetus,[43–45] maternal AUD-related anxiety and depressive symptoms in offspring,[46] or other familial environmental influences. Other studies, however, reported that both maternal and paternal AUD have a similar risk of offspring AUD.[4,38] More research is needed to assess the effect of alcoholic parental gender on offspring AUD.

There are several limitations in the study. First, information on parental AUD was obtained from the offspring rather than from the parents. Although family history data on AUD is highly specific, it tends to be less sensitive.[47] Given the reduced sensitivity, some individuals classified with non-AUD parents may have had AUD parents. Also, the term *parental AUD* was used in this article to save space although the term *parental AUD reported by offspring* would have been correct. Second, cross-sectional data limit the ability to distinguish between genetic, conception, intrauterine, and environmental factors. Third, the NESARC sample excluded individuals in hospitals and correctional settings. If these individuals were assessed, our data could have encompassed more severe cases of AUD.

In conclusion, our study showed that offspring with 1 AUD parent are at 2.51-fold higher odds and offspring with 2 AUD parents are at 4.44-fold higher odds for lifetime AUD. Offspring of AUD parents, however, can be resilient.[48] By utilizing these findings, clinicians may provide relevant education and administer screenings for AUD, which has been found to be a prevalent disorder affecting approximately 18%[49] to 30%[50] of the population. Asking patients about parental AUD is a highly efficient way of screening for AUD. Also, most parents prefer physicians to initiate discussion on parental alcohol use.[51] Clinicians can play an active role in educating individuals with parental AUD and their families, providing resources, and discussing prevention[52] and intervention[53] to mitigate other risk factors of AUD.

Disclosure of off-label usage: The authors have determined that, to the best of their knowledge, no investigational information about pharmaceutical agents that is outside US Food and Drug Administration–approved labeling has been presented in this article.

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Posttest

To obtain credit, go to PSYCHIATRIST.COM (Keyword: August) to take this Posttest and complete the Evaluation online.

1. According to the results of this study, 22% of noninstitutionalized US adults reported that they had at least 1 biological parent with a lifetime alcohol use disorder (AUD). Among the 19% with 1 parent with a lifetime AUD, what was the difference between paternal and maternal AUD?

a. No difference existed; as many mothers as fathers had an AUD
b. The number of mothers with AUD was 2 times the number of fathers with AUD
c. The number of fathers with AUD was 4 times the number of mothers with AUD
d. The number of fathers with AUD was 8 times the number of mothers with AUD

2. Neither of Mr A's parents had a lifetime AUD, but Mr B's father did, and Mr C reports that both of his parents had an AUD. With age, race, education, and personal income being equal, how likely is Mr B to have a lifetime AUD compared with Mr A?

a. The likelihood is the same
b. More than twice as likely
c. 3.5 times as likely
d. More than 4 times as likely

3. With age, race, education, and personal income being equal, how likely is Mr C to have a lifetime AUD compared with Mr A (above)?

a. The likelihood is the same
b. More than twice as likely
c. 3.5 times as likely
d. More than 4 times as likely

4. When you are evaluating Ms D, she mentions drinking wine after putting her kids to bed each evening. As part of AUD screening, you ask whether either of her parents were alcoholics or problem drinkers, and she says that both were. Compared with a woman who has no parental history of AUD, what are the odds of Ms D having an AUD?

a. More than 5 times as likely
b. More than 3 times as likely
c. More than twice as likely
d. The odds are the same