Running Head: CHILDREN OF ALCOHOLICS' PHYSICAL HEALTH OUTCOMES

Children of Alcoholics' Physical Health Outcomes in Early Childhood Avanti Jangalapalli University of Michigan

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Advisors: Dr. Anne Buu and Dr. Robert Zucker

Abstract

Many studies have found that people with alcohol use disorder (AUD) tend to have more physical health problems, such as headaches, gastrointestinal disorders, liver disorders, and cardiovascular problems. Some studies have also found that children of alcoholics (COAs) have a higher risk of developing physical health problems than non-COAs in adolescence and young adulthood. Physical health problems in young adult COAs are consistent with adult alcoholics' health outcomes. However, very few studies have focused on the early development of these physical health problems. This study compares the physical health conditions of COAs with those of non-COAs before their onset of drinking. Analyses were conducted on data regarding childhood health information from 362 child participants (from the Michigan Longitudinal Study). Children who have either parent with a lifetime diagnosis of alcohol use disorder were defined as COAs. We examined physical health problems from birth to ages 9-11. Results show increased sleep problems, more medication usage for various illnesses, and increased reports of inflammatory diseases with COAs in early childhood. Additionally, family environment was associated with parental alcoholism and children's health outcomes. These findings imply that intervention efforts for COAs' physical health problems should start at an age before the onset of drinking.

Children of Alcoholics' Physical Health Outcomes in Early Childhood

Existing literature concerning the effects of alcohol dependence (AD) have largely focused on psychological outcomes in the individual, such as depression, eating disorders, conduct problems, and anxiety (Clark et al., 2007; Diaz et al., 2008; Lee, 2008; Mylant, Ide, Cuevas & Meehan, 2002). Studies examining the subsequent effects of parental alcohol use disorder (AUD) on their children focus on similar psychological health concerns as well as an increased risk of AUD in children, including a telescoping effect, such that the consequences of alcohol use have an accelerated progression (Hussong, Bauer & Chassin, 2008). Physical health outcomes, on the other hand, are mostly reported for adult children of alcoholics (COAs) (Finney & Moos, 1992; Hesselbrock, Segal & Hesselbrock, 2000; Parks, Hesselbrock & Segal, 2001). Studies have found that COAs, at the developmental stages of adolescence and older, have a higher risk of developing physical health problems than non-COAs (Gance-Cleveland, Mays & Steffen, 2008). However, existing research on physical health outcomes in COAs prior to their onset of drinking is minimal.

This may be due to the notion that chronic health conditions surface at an older stage of development. Findings of chronic health conditions arising at childhood could provide implications for the effects of AD on COAs physical health that begin at an early stage and magnify. This study then examines COAs' physical health prior to the onset of drinking in order to understand if the adolescent or adult physical health problems found in existing literature begins at an earlier stage in their lives. Focusing on younger COAs' physical health can be informative in implementing early prevention strategies for this population. Findings of physical health outcomes in adult and adolescent COAs, as well as the existing research on COAs in

childhood is explored to provide a background of physical health problems that arise in COA populations.

Physical health outcomes in adulthood

Physical health outcomes have previously been examined in adult COAs (ages 19 and above). Adult COAs are found to be at an increased risk for hypertension, diabetes, impaired sleep cycles, generalized fatigue or delirium, gastrointestinal diseases, cirrhosis, cancer, hemorrhages, heart problems, headaches, and high blood pressure (Finney & Moos, 1992; Resnick, Perry & Applebaum, 2003; Wakabayashi & Kobab-Wakabayashi, 2002). Studies have also focused on gender differences in adult COAs. Alcohol-related health outcomes that have been reported in these studies include having an increased risk of head injuries, migraines, asthma, abdominal pain, back pain, joint pain, etc. (Hesselbrock et al., 2000; Parks et al., 2001). Male and female participants were comparable in withdrawal symptoms and physical consequences of chronic alcohol use, however, women were more likely than men to report a lower overall health status, more medication use and pain complaints, and were also more prone to stomach and liver diseases.

One study used hospitalization records as indicators of health conditions of participants and found that COAs (beginning from birth to 23 years old) have increased rates of hospitalization and increased costs tied to hospital utilization as compared to non-COAs (Woodside, Coughey & Cohen, 1993). These findings suggest that COAs may have an increased frequency and severity of health problems, and are hospitalized for longer periods of time in comparison with non-COAs. These studies provide valuable information regarding long term physical health outcomes that have been observed in adult COAs and that can be examined in younger COAs.

Physical health outcomes in adolescence

Existing literature on COAs covers adolescent and young adult populations (ages 11 to 18 years old) as well. However, these findings largely focus on their development of problem drinking. Adolescent COAs have been found to have an increased risk of developing sleep problems, eczema, epilepsy, diabetes, hearing problems, poor eyesight, hay fever, chronic fatigue, obesity, etc (Clark et al., 2007; Dahl et al., 2003). They also have reported more physical complaints (of stomach aches, headaches, and other pains), and are more at risk for conditions such as, hypertension, abdominal pain, and gastrointestinal disorders (Gance-Cleveland et al., 2008). Thus, the limited findings concerning physical health conditions in young adult and adolescent COAs are consistent with literature focusing on adult COAs' health outcomes. Younger COAs may also present early symptoms of these chronic physical health outcomes that are increasingly reported by adolescent and adult COAs. Additionally, there is a possibility that such physical health problems affecting COA populations could be more prominent due to their own alcohol use in adolescence and adulthood. Therefore, it is essential to examine physical health problems prior to the onset of drinking to understand direct effects of parental AUD without confounding influences of participants' own drinking behaviors.

Physical health outcomes in childhood

One study comparing pre-adolescent children with alcoholic fathers and those without alcoholic fathers found that while COAs did not report being ill more than non-COAs, COAs did tend to have a lower birth weight comparatively. Also, male COAs in non-intact (parents were split up) families were reported to have shorter heights in comparison to non-COAs (Dobkin, Tremblay, Desmarais-Gervais & Dépelteau, 1994). Since height differences were found in COAs

in a single-parent household, this study provides support for theories attributing the causes for health problems in COAs to additional external life stressors.

Theories

There are various theories regarding reasons for the development of physical health problems in COAs that emphasize influences of either the nature or nurture realm. One study examining alcoholism and abnormalities in the functioning of the hypothalamic-pituitary-adrenocortical (HPA) system suggests that the changes in HPA systemic activity evident in alcoholics are evident in COAs with a family history of alcohol use as well. These abnormalities (such as, lower adrenal corticotropin, cortisol, and beta-endorphin levels that slow responses to psychological stress) that appear prior to the onset of drinking in COAs may indicate AD as having an inheritable quality due to its' neurobiological effects (Sher, Cooper, Mann & Oquendo, 2006). Studies examining gender differences in alcohol use find that women are more affected by lower dosages of alcohol than men, and experience more alcohol-related health problems (Deal & Gavaler, 1994; Roman, 1988). Studies supporting inherent gender differences in the effects of alcohol on physical health provide implications for the increased risks faced by female COAs than male COAs.

A study examining chronic back pain in female COAs claimed that alcohol use by parents/caretakers could promote negative family relationships, which in turn, psychologically affect COAs (Pecukonis, 2004). Similar theories have been pursued to explore the possibility of parental alcohol use affecting parenting and parental warmth towards children, although, it should be noted that most of these studies find significant influences with paternal alcohol use (Eiden, Edwards & Leonard, 2004; Keller, Cummings, Davies & Mitchell, 2008). These correlations between parental alcohol use and their subsequent maltreatment of children,

negative parenting methods and a lack of warmth provide implications for the development of psychological issues that can lead to both mental and physical health problems in COAs by causing their vulnerability to translate into somatic symptoms.

A more established understanding of the differences between COAs and non-COAs entails the multiple stressors that are typically involved in the life of families with parents who have a history of alcohol use. Families with parental alcohol use tend to have a lower socioeconomic status (SES) (von Knorring, 1991), are more likely to be single-parent households (Cunningham & Knoester, 2007), and are more disruptive, chaotic and inconsistent (Bennett, Wolin & Reiss, 1988; Conners et al., 2003). These stressors can then combine to negatively affect child development. Lower SES has largely been associated with poorer physical health (Power, Manor & Fox, 1991). Stress levels in an individual have also consistently been associated with lower SES and increased health problems (Cohen, Kaplan & Salonen, 1999). With several theories circulating about alcohol research, there is a need to study biological and environmental indicators that can be identified at an early age and targeted in at-risk youth populations. Existing literature lacks a specific focus of younger COAs and their physical health outcomes as a result of their parental alcohol use. However, early physical indicators of health conditions in COAs can be identified through the examination of the chronic conditions that have frequently been linked to adolescent and adult COAs.

The present study

There is a need to embellish the existing literature concerning the physical health of COAs prior to their onset of drinking. COAs ages 11 and older have consistently been associated with increased risks of physical health problems. Exploring the symptoms and possible development of chronic physical health problems in younger COAs will provide researchers with

more information regarding, (1) the causes behind the relationship between parental AUD and children, (2) indicators of chronic health conditions appearing in early childhood, and (3) guides to implement early prevention strategies that target symptoms of chronic conditions in young COAs.

In this study, we hypothesized that, (1) COAs with parental AUD only before their birth would report more health problems than non-COAs, (2) COAs with exposure to parental AUD would have more health problems than COAs exposed to only paternal AUD, (3) exposure groupings of COAs would have effects on family stress, parenting, family relationships and family SES, which in turn would affect children's health problems, and finally, (4) family density of alcoholism would increase the risk of children's physical health problems.

Method

Design and Sample

Our sample, from the Michigan Longitudinal Study (MLS), consisted of alcoholic families in a four county area, recruited from a group of men who have had drunk driving convictions. Men either had a first time conviction with a blood alcohol concentration of 0.15% or above, or a previous drinking-related legal issue with a blood alcohol concentration of at least 0.12%. They had to meet the criteria for a probable/definite alcoholism diagnosis in order to be considered for the study. Men were also required to have at least one biological son between the ages of three and five years old for the purposes of the study, and be living with the son and biological mother at the time of recruitment. Mothers did not necessarily meet the criteria for an alcoholism diagnosis. Non-alcoholic families formed a contrast group and were defined as families with neither parent having a history of alcohol use. They were recruited through canvassing door-to-door in neighborhoods of the recruited alcoholic families. Through

canvassing, a group of families with alcoholic fathers who did not have alcohol-related legal problems after their child's birth was also gathered.

MLS families were given a series of questionnaires upon recruitment at baseline (3-5 years old, T1) and subsequently, every three years (T2 and above). This study included a sample of 362 children who have completed T3, with 239 boys and 123 girls, ages ranging from 8.62 to 11.98 years old. Of the sample, 150 (41%) participants were recruited from court alcoholic families, 81 (22%) from community alcoholic families, and 131 (36%) from nonalcoholic families.

Measures

DSM-IV Alcohol Dependence and Abuse Diagnoses. (Robins et al., 2000). To determine parental alcoholism status, a Ph.D. level clinical psychologist conducted alcohol dependence and alcohol abuse diagnoses every three years based on information from the Diagnostic Interview Schedule and the Drinking and Drug History Questionnaire using DSM-IV criteria. When discrepancies were observed between the two instruments, the more severe pattern was taken as the best estimate. Parents were considered as having AUD if they met the diagnosis for either alcohol dependence or alcohol abuse. Participants were divided into four groups according to their exposure to parental AUD; (1) COAs exposed to AUD in both parents, which included children who are exposed to both paternal and maternal AUD in their lifetime, (2) COAs exposed to paternal AUD, which included children with a lifetime exposure to only paternal AUD and not maternal, (3) no lifetime exposure COAs, which included children with parental AUD only prior to their birth, and finally, (4) non-COAs, whose parents do not have a history of alcoholism before or after the child's birth.

Child Health History. This instrument was administered to the parent (either the mother or primary caregiver) for information regarding the child's past and current developmental health. The questionnaire focuses on the following areas: pregnancy, birth of the child, growth and development, child's health, social development, family background information, and school history. Data used in analyses focused on children at T3 (9-11). Specific sleeping troubles assessed in this measure were grouped according to similar problems for the purposes of analyses (i.e., (1) an aggregate measure of restless sleep included questions regarding children tossing in bed, falling out of bed, and having peculiar sleep positions, (2) excessive sleepiness consisted of being easily fatigued and sleeping unusually soundly, (3) insomnia reflected children who had trouble getting to sleep and who have to sleep in their parents' bed, (4) sleep disordered breathing included sweating in sleep and snoring, (5) unusual activity in sleep included talking, laughing or crying in sleep, grinding teeth, sleepwalking/wandering at night, nightmares or night terrors). Additionally, we grouped inflammatory diseases assessed in this measure into one category consisting of measles, meningitis, rubella, polio, pneumonia, mumps, whooping cough, encephalitis, scarlet fever and rheumatic fever. In assessing medication use by the child, we utilized the median values reported of the number of days the participants took particular medications in analyses.

Demographic Questionnaire. This instrument assesses characteristics of the participant and the participant's family background, including, family origin, religion, church attendance, education, and occupation. It uses Duncan's Socioeconomic Index (SEI), an established occupation-based measure of SES based on United States census data, to classify the family's social standing (Mueller & Parcel, 1981). Both parents responded to the questionnaire and a mean of father and mother reports of SES were averaged to reflect overall family SES.

Information collected from this instrument was used in examining if SES has effects on the development of physical health problems in COAs.

Family Crisis List (FCL). (Patterson, 1982). This scale, developed by the Oregon Social Learning Center contained 40-items reflecting family-related stressors occurring in the past six months and the interaction between stressors and patterns of family coerciveness. Seven composites were created of the items to be used in analyses (i.e. crisis in the family, home, family's economy, health, school, social relations, and legal area). Questions provided information regarding family-experienced crises to determine if such stressors were related to the development of physical health problems in COAs.

Moos Family Environment Scale. (FES; Moos, 1974; Moos & Moos, 1976). The FES assesses family members' perceptions of their family climate, incongruence, and social environment. It is empirically based on and requires fifth or sixth grade level reading skills. The FES items were divided into ten subscales of family climate (i.e., cohesion, expressiveness, conflict, independence, achievement orientation, intellectual-cultural orientation, active recreational orientation, moral-religious emphasis, organization and control) and questions focused on the differences between groups within the family, and the extent of family incongruence. The instrument's subscales have been significantly associated with substance abuse families in previous studies and found to have good reliability and validity.

Genogram and Family History. A genogram was completed by each biological parent to reflect his/her family history of health conditions. Data were collected through a structured interview and dated back to three generations for each parent at baseline, and then updated every three years to include the most recent information. The genogram displayed a person's family relationships, morbidity, and mortality statuses. For this particular study, we looked at family

history of alcoholism only. Reliability testing in a set of 20 families was conducted for numbers of alcoholic relatives reported, which showed adequate test-retest reliability (.96 over 18 weeks). Family density of alcoholism for participants' parents was categorized into four groups, such that the parent had; (1) no relatives with alcoholism, (2) at least one relative but not parent with alcoholism, (3) a parent with alcoholism but no relatives from previous generations with alcoholism, and (4) a parent and at least one other relative from previous generations with alcoholism.

Statistical Analyses

T-test analyses were conducted to compare the physical health outcomes between nolifetime exposure COAs and non-COAs, as well as differences between COAs exposed to AUD in both parents and COAs exposed to only paternal AUD. We employed a one-way ANOVA to compare the differences in family environments among COAs exposed to both parental AUD, COAs exposed to paternal AUD, and a non-exposure group of participants that consisted of no-lifetime exposure COAs and non-COAs. In order to examine the relationships between family environment and children's physical health outcomes, we then conducted correlation analyses.

Additionally, one-way ANOVA was used to determine the influence of family density of alcoholism on children's physical health outcomes. The test was conducted to assess whether the prevalence of alcoholism in previous generations was positively associated with more health problems in offspring.

Results

No lifetime exposure COAs vs. non-COAs

No significant difference was found in physical health outcomes between children with a parental history of AUD before their birth and non-COAs (Table 1). However, results largely

demonstrated that no-lifetime exposure COAs reported more health problems than non-COAs. We employed *t*-test analyses separately on both male participants and female participants and noted a few differences. Male COAs with no lifetime exposure to parental AUD reported more health problems including excessive sleep, insomnia, restless sleeping, sleep disordered breathing, and unusual sleeping in comparison to male non-COAs. These participants also reported having taken a fewer number of immunizations that are required by the Center for Disease Control, as well as having more days using pain medications, decongestants, multisymptom cold remedies, asthma medications, and antacids. Female COAs with no lifetime exposure reported more sleep disordered breathing and had taken fewer immunizations than female non-COAs. They also reported more days using pain medications, decongestants, antihistamines, antibiotics, allergy medications, and laxatives compared to female non-COAs. *COAs exposed to both parents' AUD vs. COAs exposed to paternal AUD*

COAs with exposure to both parents' AUD were found to have more physical health problems than COAs exposed to only paternal AUD (Table 2). Particularly, male COAs with parental AUD had significantly higher reports of restless sleeping than male COAs with only paternal AUD. Female COAs with exposure to parental AUD had significantly higher occurrences of inflammatory diseases (i.e., measles, meningitis, rubella, polio, pneumonia, mumps, whooping cough, encephalitis, scarlet & rheumatic fevers) than female COAs with only a paternal history of AUD. In general, while most of the results were not significant, male COAs with both parental diagnoses reported more inflammatory diseases and more sleeping troubles such as, excessive sleepiness, insomnia, disordered breathing, and unusual sleeping than male COAs with only paternal AUD. These participants also reported more use of decongestants, multi-symptom cold remedies, allergy medications, vitamins, and antacids. In comparison to

female COAs with only paternal AUD, female COAs with both parents' AUD reported more sleep problems such as, excessive sleepiness, insomnia, disordered breathing while sleeping, and unusual sleeping. These participants also had taken fewer immunizations, and reported using more cough medications, decongestants, multi-symptom cold remedies, asthma and allergy medications, and antacids.

Family environmental influences

Table 3 and Table 4 show correlations between family environment and children's physical health problems in males and females. In general, crises in children's schooling environment and crises in their family economic situation were positively correlated with worse physical health outcomes. Family intellectual-cultural orientation and family moral-religious emphasis were negatively related to children's physical health problems. We also found that male and female COAs with exposure to both parental AUD were more likely to be from families with a worse living environment (Table 5). Male COAs with exposure to both parental AUD had significantly more crises in their family economic situation, more crises in the schooling environment, and less family intellectual-cultural orientation in comparison to male COAs with paternal AUD or males in the non-exposure group. Female COAs with exposure to both parental AUD had significantly more crises in family economical status, less family intellectual-cultural orientation, and less family moral-religious emphasis, in comparison to female COAs exposed to paternal AUD or females in the non-exposure group.

Family density of alcoholism

Table 6 shows the effects of family density of alcoholism on children's health conditions.

Male children with at least one alcoholic grandparent on their biological father's side were significantly more likely to have sleep disordered breathing, in comparison to male children who

had no alcoholic relatives reported above their father's generation. For female children, family density of alcoholism shows effects on their medication use. Girls reported using significantly more decongestants, antihistamines, allergy medications, and antacids if they had at least one alcoholic grandparent on their biological mother's side. In general, significant effects on boys were only found from father's family density of alcoholism, and significant effects on girls were only from mother's family density of alcoholism.

Discussion

Results do not show significant differences in physical health conditions between non-COAs and COAs with parental AUD prior to their birth. However, our results largely indicate that COAs with no-lifetime exposure to parental AUD have more reported health problems related to sleeping, and more medication use for respiratory conditions than non-COAs. This finding demonstrates the possibility of COAs having an increased risk for the development of health conditions during childhood. Similarly, there were no significant differences in physical health conditions between COAs with only paternal AUD and COAs with parental AUD. These findings are consistent with existing literature which has not found notable differences in the development of physical health problems in COAs prior to adolescence. However, results show a trend in which COAs with parental AUD tended to report more physical health problems than COAs with only a paternal history of AUD. These findings suggest that while COAs may have an increased risk of physical health problems due to a parent with a history of AUD, there is an increased risk placed on children when both parents exhibit excessive alcohol use rather than just one parent.

Findings show an association between groups of COAs and family environment, such that, COAs with parental AUD tended to experience an increased amount of family

environmental stressors than COAs with just paternal AUD or non-COAs. Harsher family environments were also then related to increased reports of physical health problems in children. These results suggest an indirect relation between parental alcoholism status, family environmental stressors and children's subsequent physical health outcomes. This is an expected result due to existing literature supporting the link between negative family environments and the development of physical health problems. Literature shows that environmental stressors such as those examined in this study (e.g., family conflict, family disorder, low SES) are more prevalent in families with a history of alcoholism, and are also factors influencing the development of physical health problems. However, literature is limited in the focus of this association on a younger population. This study's findings provide support for theories regarding the environmental effects on children's development since there is a positive relation between parental AUD and family crises, as well as between family crises and children's health problems. This indirect relation parallels existing literature that focuses on these variables while demonstrating the relationship in COAs at an earlier stage.

Family density of alcoholism was positively associated with physical health conditions in participants. This suggests that physical health outcomes of COAs may be an effect of the increased incidence of alcoholism occurring in their entire family rather than just in their parents' alcoholism status. Interestingly, significant results with male COAs were only found when their biological father had a higher family density of alcoholism. Similarly, significant results with female COAs were only found when their biological mother had a higher family density of alcoholism. That male COAs are more affected by their biological father's family history of alcoholism and female COAs are more affected by their biological mother's family history of alcoholism implies that there may be a gender-related genetic component involved in the

association between parental alcoholism and children's health outcomes. Additionally, a higher number of significant results were found in female COAs, supporting existing literature that shows that females have an increased vulnerability to the effects of alcohol use, and as a result, possibly to the effects of parental alcoholism as well.

While there is substantial research reflecting the increased risk of chronic health conditions in adult COAs, there is a need to focus on a younger population to determine if such conditions arise before the child initiates alcohol use. This study begins to examine this younger population of COAs to determine the existence of risks they face for chronic health conditions as a result of their parents' drinking behaviors. Findings from this study demonstrate that COAs experience more sleep problems and use more medications than non-COAs. Information regarding children's medication use supplemented their reports of health problems and thus demonstrates the strength of this study's findings. Additionally, this study's strength lies in its examination of physical health conditions in a younger population of COAs' while providing an expansive look at the genetic and environmental links between this relation. The results also show that the risks of health problems are greater regardless of COAs' exposure to parental AUD, implying the possibility of a common environmental characteristic experienced by families with alcoholism. This is consistent with theories that suggest that alcoholic families are more likely to live in stressful environments that harm their children's development. Therefore, prevention strategies should target children early on who are exposed to harmful family environments and have a parental history of alcoholism to deter the development of chronic health conditions that are prevalent in adult COAs.

Future studies examining the effects of parental alcoholism on their children's development of physical health conditions should take into consideration a few limitations of this

study. Firstly, the data gathered for this study is based on participant reporting of their personal and family health history. Therefore, parent reports and child reports may not be as truthful as possible and may be biased. Secondly, the method for creating each parent's family history of alcoholism required parents to remember details of their relatives alcohol use for three generations prior to theirs. For this reason, the information gathered could be affected by a memory bias. Thirdly, the MLS recruited men and their families for this study with a few restrictions; men had to have at least one son between the ages of three and five years old and had to be living with their son and his biological mother. Thus, this recruitment procedure reduces the external validity of this study. Finally, this study separated analyses between male and female children; however, comparisons between genders were not focused on. Future studies examining physical health outcomes in this population could compare differences in the development of conditions among genders to examine if there is a gender-related genetic component to COA's health.

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Author's Note

Avanti Jangalapalli, Department of Psychology, University of Michigan, Ann Arbor.

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Questions or comments concerning this research paper should be directed to Dr. Anne Buu, Research Assistant Professor, University of Michigan, Department of Psychiatry. She can be reached at 'buu@med.umich.edu'.

Table 1 Comparison between No Lifetime Exposure COAs and Non-COAs

	N	Iale Children		Female Children			
Variables	Non-COAs No exposure,		<i>t</i> -value	Non-COAs	No exposure,	<i>t</i> -value	
	with parental				with parental		
	AUD before				AUD before		
	birth				birth		
Inflammatory diseases	0.24 (0.57)	0.21 (0.60)	0.20	0.21 (0.41)	0.14 (0.44)	0.69	
Excessive sleepiness	0.61 (0.56)	0.73 (0.72)	-0.86	0.80 (0.57)	0.66 (0.60)	0.98	
Insomnia	0.49 (0.39)	0.58 (0.44)	-0.96	0.59 (0.59)	0.45 (0.52)	1.05	
Restless sleeping	0.35 (0.42)	0.43 (0.44)	-0.91	0.43 (0.39)	0.33 (0.37)	1.05	
Sleep disordered breathing	0.48 (0.56)	0.65 (0.63)	-1.32	0.39 (0.44)	0.40 (0.54)	-0.02	
Unusual sleeping	0.34 (0.35)	0.38 (0.34)	-0.48	0.30 (0.32)	0.29 (0.25)	0.20	
Number of missing immunizations	2.52 (4.24)	3.12 (4.17)	-0.65	2.03 (3.50)	2.62 (3.76)	-0.66	
Days taking pain medications	2.85 (1.90)	2.88 (2.09)	-0.07	2.68 (1.76)	3.10 (1.76)	-0.97	
Days taking cough medications	1.74 (1.92)	1.61 (1.94)	0.33	1.97 (1.79)	1.76 (1.82)	0.48	
Days taking decongestants	1.54 (2.14)	1.58 (2.42)	-0.07	1.18 (2.08)	1.55 (2.13)	-0.71	
Days taking antihistamines	1.34 (2.15)	0.76 (1.71)	1.33	0.29 (1.23)	0.96 (1.97)	-1.62	
Days taking multi-symptom cold remedies	0.49 (1.16)	0.54 (1.77)	-0.16	0.47 (0.98)	0.41 (1.27)	0.22	
Days taking antibiotics	2.36 (2.36)	1.39 (2.09)	1.95	1.63 (2.07)	1.79 (2.21)	-0.31	
Days taking asthma medications	0.25 (1.03)	0.45 (1.35)	-0.80	0.23 (1.46)	0.17 (0.93)	0.22	
Days taking allergy medications	0.86 (2.16)	0.79 (2.25)	0.16	0.42 (1.26)	0.52 (1.57)	-0.28	
Days taking vitamins	4.39 (4.10)	3.67 (4.20)	0.80	3.66 (3.96)	1.93 (3.33)	1.89	
Days taking laxatives	0.08 (0.53)	0.06 (0.35)	0.26	0.00 (0.00)	0.34 (1.32)	-1.41	
Days taking antacids	0.29 (0.89)	0.33 (0.85)	-0.24	0.63 (1.32)	0.55 (1.18)	0.26	
Median of days taking medications	10.61 (9.76)	20.23 (37.48)	-1.38	8.71 (10.96)	12.17 (10.70)	-1.28	

Note. Mean and (Standard Deviation) values are provided in the columns.

Table 2 COAs Exposed to both Parents' AUD vs. COAs Exposed to Paternal AUD

	M	ale Children	Female Children			
Variables	COAs with	COAs with	<i>t</i> -value	COAs with	COAs with	<i>t</i> -value
	Paternal AUD	Parental AUD		Paternal AUD	Parental AUD	
Inflammatory diseases	0.34 (1.23)	0.34 (0.69)	-0.02	0.10 (0.40)	0.44 (0.51)	-2.59*
Excessive sleepiness	0.72 (0.63)	0.76 (0.62)	-0.35	0.75 (0.58)	0.92 (0.55)	-0.98
Insomnia	0.41 (0.38)	0.52 (0.51)	-1.26	0.55 (0.38)	0.58 (0.60)	-0.21
Restless sleeping	0.22 (0.36)	0.44 (0.44)	-3.13*	0.42 (0.35)	0.39 (0.40)	0.30
Sleep disordered breathing	0.48 (0.48)	0.50 (0.49)	-0.20	0.32 (0.40)	0.56 (0.56)	-1.70
Unusual sleeping	0.29 (0.30)	0.37 (0.33)	-1.35	0.37 (0.39)	0.42 (0.30)	-0.52
Number of missing immunizations	3.08 (4.42)	3.02 (4.85)	0.07	1.83 (3.95)	2.28 (4.08)	-0.38
Days taking pain medications	3.17 (1.94)	2.72 (1.65)	1.38	3.03 (2.02)	2.39 (1.82)	1.11
Days taking cough medications	2.40 (1.88)	1.90 (1.80)	1.54	1.57 (1.96)	1.83 (1.86)	-0.47
Days taking decongestants	1.37 (2.10)	1.54 (1.96)	-0.47	0.90 (1.49)	1.39 (1.91)	-0.99
Days taking antihistamines	0.85 (1.68)	0.82 (1.73)	0.11	0.60 (1.30)	0.44 (1.34)	0.40
Days taking multi-symptom cold remedies	0.44 (1.17)	0.58 (1.25)	-0.67	0.50 (1.25)	1.11 (1.53)	-1.51
Days taking antibiotics	1.91 (2.32)	1.74 (2.33)	0.41	1.90 (2.56)	1.39 (2.14)	0.71
Days taking asthma medications	0.43 (1.66)	0.36 (1.27)	0.27	0.17 (0.91)	0.28 (1.18)	-0.37
Days taking allergy medications	0.66 (2.01)	0.90 (2.19)	-0.65	0.40 (1.52)	0.67 (1.57)	-0.58
Days taking vitamins	3.54 (4.19)	3.66 (4.11)	-0.16	3.43 (4.11)	2.56 (3.81)	0.74
Days taking laxatives	0.11 (0.86)	0.04 (0.28)	0.73	0.10 (0.55)	0.00 (0.00)	1.00
Days taking antacids	0.46 (1.18)	0.80 (1.96)	-1.12	0.53 (1.10)	0.67 (1.28)	-0.38
Median of days taking medications	14.69 (19.93)	10.52 (12.99)	0.25	10.50 (11.72)	8.59 (6.44)	0.68

Note. Mean and (Standard Deviation) values are provided in the columns.

^{*} *p*< .05

Table 3 Correlations between Family Environment and Male Children's Physical Health Problems

Children's	Family	Crisis in	Crisis in	Family Intellectual-	Family Moral-
Health Problems	SES	Family Economy	School	Cultural Orientation	Religious Emphasis
Inflammatory Diseases					
	0.10	0.01	-0.07	0.07	0.00
Excessive Sleepiness					
	-0.12	0.01	0.01	-0.18*	-0.13*
Insomnia					
	0.01	-0.03	0.01	-0.05	0.04
Restless Sleep					
•	-0.04	0.12	0.15*	-0.08	-0.09
Unusual Sleeping					
	-0.10	-0.08	0.01	0.00	0.12
Number of Missing Immunizations					
- · · · · · · · · · · · · · · · · · · ·	-0.18*	0.01	0.11	-0.01	0.05
Days Taking Pain Medications					
2 ujo 1 mm 1/10 uzo uzo no	0.15*	0.12	0.08	0.02	-0.05
Days Taking Decongestants	0.15	0.12	0.00	0.02	0.05
Buys Tuking Decongestants	0.21*	0.01	-0.03	-0.02	0.01
Days Taking Multi-Symptom Cold Ren		0.01	0.03	0.02	0.01
Days Taking Waiti Symptom Cold Rei	0.02	0.00	0.04	-0.07	-0.10
Days Taking Antibiotics	0.02	0.00	0.04	-0.07	-0.10
Days Taking Andibiodes	0.03	0.00	-0.04	0.06	0.11
Days Taking Laxatives	0.03	0.00	-0.04	0.00	0.11
Days Taking Laxatives	0.00	0.02	0.02	0.04	0.00
Davis Talina Antosida	0.08	-0.03	0.02	0.04	0.08
Days Taking Antacids	0.05	0.15*	0.16*	0.12	0.00
	-0.05	0.15*	0.16*	-0.12	0.00

Note. N=233.

^{*} *p*< .05

Table 4
Correlations between Family Environment and Female Children's Physical Health Problems

Children's	Family	Crisis in	Crisis in	Family Intellectual-	Family Moral-
Health Problems	SES	Family Economy	School	Cultural Orientation	Religious Emphasis
Inflammatory Diseases					
	-0.05	0.02	0.10	-0.21*	0.06
Excessive Sleepiness					
	0.03	-0.07	0.05	-0.11	-0.16
Insomnia					
	0.14	-0.02	0.22*	-0.10	-0.09
Restless Sleep					
	-0.04	0.23*	0.22*	-0.21*	0.02
Unusual Sleeping					
	-0.09	-0.05	-0.02	-0.23*	0.02
Number of Missing Immunizations					
	-0.09	0.24*	-0.07	0.11	0.00
Days Taking Pain Medications					
	0.04	0.03	-0.06	0.11	0.16
Days Taking Decongestants					
	0.34*	-0.06	-0.02	0.14	0.18
Days Taking Multi-Symptom Cold Medi	cations				
	-0.12	-0.06	0.01	-0.05	-0.19*
Days Taking Antibiotics					
	0.10	0.00	-0.13	0.07	0.17
Days Taking Laxatives					
	0.04	0.01	0.04	-0.04	-0.02
Days Taking Antacids					
	0.14	0.03	-0.09	-0.02	0.09

Note. N=233.

^{*} *p*< .05

Table 5
Family Environmental Conditions of Exposure Groups

		Male Childre	n	Female Children			
	Non-	Exposed to	Exposed to	Non-	Exposed to paternal	Exposed to	
	exposure	paternal	both parental	exposure		both parental	
	group	AUD	AUD	group	AUD	AUD	
	(N=90)	(N=87)	(N=50)	(N=67)	(N=30)	(N=18)	
Family SES	376.51	315.96*	281.90*	373.98	329.00	261.03*	
	(136.59)	(123.45)	(96.86)	(142.35)	(133.37)	(70.13)	
Crisis in family economy	0.59	0.73	1.14*†	0.66	0.89	1.33*	
	(0.87)	(0.92)	(1.09)	(1.00)	(1.06)	(0.97)	
Crisis in school	0.78	0.62	1.12†	0.55	0.57	0.61	
	(0.97)	(0.87)	(1.38)	(0.64)	(0.74)	(0.85)	
Family intellectual-cultural orientation	5.83	5.22	4.02*†	5.59	5.59	3.83*†	
	(1.90)	(2.02)	(2.29)	(2.08)	(2.14)	(2.09)	
Family moral-religious emphasis	6.64	5.69*	5.28*	6.38	5.67	4.94*	
	(2.10)	(2.08)	(1.91)	(2.19)	(2.34)	(1.51)	

Note. Mean and (Standard Deviation) values are provided in the columns.

^{*} The mean of particular group is significantly different from the mean of non-exposure group (p<0.05)

 $[\]dagger$ The means of children exposed to both parental AUD group and children exposed to paternal AUD group are significantly different (p<0.05)

Table 6 Effects of Family Density of Alcoholism on Children's Physical Health Problems

	Male Children				Female Children			
	Biological Father Bio			al Mother	Biological Father		Biologic	al Mother
	Sum of		Sum of		Sum of		Sum of	
Variables	Squares	F Value	Squares	F Value	Squares	F Value	Squares	F Value
Inflammatory diseases	1.80	0.68	3.29	1.30	0.20	0.30	0.22	0.36
Excessive sleepiness	1.33	1.17	1.58	1.35	2.15	2.43	1.48	1.54
Insomnia	0.15	0.27	0.69	1.33	0.86	1.05	0.38	0.52
Restless sleeping	0.42	0.80	0.13	0.25	0.06	0.15	0.26	0.61
Sleep disordered breathing	2.48	2.87*	1.49	1.71	1.07	1.45	1.16	1.56
Unusual sleeping	0.42	1.31	0.26	0.78	0.24	0.74	0.13	0.41
Number of missing immunizations	95.85	1.65	149.98	2.69	4.08	0.10	88.83	2.16
Days taking pain medications	1.93	0.18	14.77	1.34	11.60	1.15	0.46	0.04
Days taking cough medications	4.95	0.44	9.80	0.88	15.66	1.51	14.47	4.82
Days taking decongestants	28.09	2.04	15.36	1.09	0.66	0.06	28.72	2.74*
Days taking antihistamines	6.55	0.60	9.05	0.85	2.76	0.35	46.66	7.84*
Days taking multi-symptom cold remedies	7.54	1.57	1.74	0.38	6.49	1.61	1.81	0.39
Days taking antibiotics	16.38	1.03	8.19	0.52	7.85	0.51	16.77	1.11
Days taking asthma medications	5.19	1.02	4.64	0.78	5.31	1.34	1.12	0.57
Days taking allergy medications	6.52	0.48	0.12	0.01	18.02	2.52	21.27	3.38*
Days taking vitamins	20.38	0.40	101.40	2.01	42.56	1.00	4.27	0.10
Days taking laxatives	0.80	0.63	0.64	0.52	1.38	0.87	0.92	0.57
Days taking antacids	1.86	0.47	7.81	1.91	0.37	0.09	12.10	2.86*
Median of days taking medications	2247.33	1.73	1037.50	0.80	589.23	1.59	317.85	0.95

Note. Degrees of freedom=3.

^{*} *p*< .05