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Family structure and alcohol use disorder: A register-based cohort study among offspring with and without parental alcohol use disorder

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Abstract

Aims: To assess whether parental alcohol use disorder (AUD) is associated with higher risks of living in a non-intact family and assess whether non-intact family structure is associated with higher risks of AUD in the offspring.

Design: Prospective cohort study.

Setting: Danish nationwide registries.

Participants: 9,948 parental AUD offspring and 98,136 reference offspring from the Danish population.

Measurements: Family structure assessed at birth and at each birthday until age 15 as intact or non-intact (with mother only, father only or neither parent). Years lived in an intact family defined as total number of years lived with both parents from birth until 15th birthday. AUD defined as registration in medical, treatment and cause of death registries. Data was analyzed by Cox regression.

Findings: At birth, 30.9% (95% CI = 29.1–32.6) of parental AUD offspring and 10.7% (95% CI = 10.3–11.0) of reference offspring lived in a non-intact family. At age 15, the numbers were 84.6% (95% CI = 83.9–85.3) and 38.4% (95% CI = 38.1–38.7). Parental AUD was associated with a higher risk of offspring AUD (HR = 1.88, 95% CI = 1.74–2.02). Offspring were at lower risk of AUD if they lived 15 years in an intact family compared with offspring who never lived in an intact family (HR = 0.67, 95% CI = 0.52–0.87 for those with parental AUD, and HR = 0.53, 95% CI = 0.48–0.59 for those whose parents did not have AUD). Findings were inconclusive as to whether or not an association was present between family structure and AUD among offspring with parental AUD and reference offspring.

Conclusions: The prevalence of non-intact family structure appears to be higher in offspring of parents with alcohol use disorder (AUD) than among offspring from the general population. Parental AUD appears to be associated with increased risk of offspring AUD, and non-intact family
structure appears to be associated with increased risk of offspring AUD regardless of parental AUD.

**Key words:** alcohol, alcohol use disorder, family structure, offspring, parents, cohort study, epidemiology

**Introduction**

In most societies, family structures have undergone markedly changes since the 1970s, characterized by lower marriage rates, higher divorce rates and less children being brought up in intact families (i.e. families where both parents are present) (1, 2). Multiple studies have documented that offspring from non-intact families generally fare worse in relation to well-being (3-6), violence, delinquency, self-harm (7-10) and mental health (11-18) compared with offspring from intact families.

Evidence indicates that offspring of parents with alcohol use disorder (AUD) are at high risk of growing up in a non-intact family (19-24). Previous research concerning family structures among offspring of parents with AUD has been based on single or few assessments at specific ages (19-21) or as single measures covering the entire upbringing (22-24). Family structures are, however, dynamic with many possible trajectories over time (7), and a detailed overview is therefore warranted to gain a more comprehensive understanding of whom offspring with parental AUD grow up with. Studies conducted on general population samples have shown that adolescents from non-intact families are at higher risk of harmful alcohol drinking patterns compared with adolescents from intact families (25-29). It is, however, unknown whether these findings apply to offspring with parental AUD. The underlying mechanisms between family structure and development of AUD may differ when a parent is affected by...
AUD because the presence of a parent with AUD in the household is linked with high levels of conflict, aggression and violence (30-32). Studies on the effects of living with a parent with AUD are, however, inconclusive as higher risks of harmful drinking patterns and AUD has been found among offspring who live together with, as opposed to apart from, a parent with AUD in some studies (19, 21, 24), while no evidence for an association has been found in other studies (33, 34).

Based on comprehensive and accurate nationwide information on residential addresses and complete parent-offspring linkage, the present study aimed (1) to test whether parental AUD is associated with higher risks of living in a non-intact family and (2) to test whether and non-intact family structure is associated with higher risks of AUD in the offspring.

Methods

Design

This matched cohort study followed offspring with parental AUD and offspring from the general Danish population up for AUD using information from Danish nationwide registers. Since 1968, residents have been assigned a unique personal identification number (PIN) in the Danish Civil Registration System register at birth or when taking permanent residence in Denmark. Linkage across registers is possible as the PIN is used consecutively across registers (35). Information on residential addresses was used to determine offspring’s family structure during upbringing.

The study population was defined as following: Parental AUD was based on registration in the Copenhagen Alcohol Cohort (COPAC), which is a database with information on 30,286
individuals entering alcohol treatment from 1954 to 2009 at five outpatient clinics on somatic hospitals in the greater Copenhagen area. Patients were given a diagnosis of the alcohol use disorder according to the International Classification of Diseases, Revision 8 (ICD-8) (until 1993) or Revision 10 (ICD-10) (since 1994) at treatment start (36). Individuals registered with a diagnosis of AUD (ICD-8: 303.0–303.2, ICD-10: F101–F104, including all sub-levels) were included in this study (n=27,844). Next, offspring to individuals with AUD were identified from the Danish Civil Registration System (n=30,318). Inclusion criteria were: residing in Denmark at 15th birthday, ≥15 years at end of follow-up, at least one parent registered in COPAC prior to the offspring’s 15th birthday and known identities of both parents. Moreover, only offspring born ≥1971 were included, because residential addresses have been registered since then. A reference population was constructed using information from the Danish Civil Registration System with the same inclusion criteria as applied on the offspring with parental AUD. Additionally, reference offspring were not allowed to have a parent registered in COPAC. Next, ten reference offspring were randomly selected per offspring with parental AUD using incidence density sampling with replacement. Matching criteria were sex, birth date (± one year) and living area (municipality) at the offspring’s 15th birthday. Last, we excluded offspring with parental AUD and reference offspring if they were registered with AUD before or on their 15th birthday to be certain that the exposures (parental AUD and family structure) preceded the outcome (offspring AUD).

Measures

Family structure

Since 1971, residential addresses have been recorded in the Danish Civil Registration System with one address recorded at any time for all Danish residents. Addresses are updated without deletion of previous registrations (35). We used information on municipality, street,
house and floor to determine whether offspring lived together with their legal parent. Family structure was assessed at each birthday from birth up to and including the offspring’s 15th birthday. Family structure was categorized as: living with both parents, living with mother only, living with father only, not living with either parent and unknown. Number of years of family intactness (i.e. lived with both parents) was calculated as the cumulated number of years from birth up to and including the offspring’s 15th birthday, and a measure with the following categories was constructed: 0 years, 1–4 years, 5–9 years, 10–14 years and 15 years.

Offspring AUD

AUD in the offspring was defined as one of the following: Registration with a diagnosis of AUD (ICD-8: 291, 303, 571.09, 571.10, 577.0, 577.10, 577.19, ICD-10: F10, G312, G621, G721, I426, K292, K70, K860 – including all sublevels) as a main or secondary diagnosis in the Danish National Patient Register (37) or the Danish Psychiatric Central Research Register (38), or as a cause of death in the Danish Register of Causes of Death (39); Redemption of a prescription of drugs used for alcohol dependence (Anatomical Therapeutic Chemical Classification System (ATC) code: N07BB) in the Danish National Prescription Registry (40); Registration in the National Registry of Alcohol Treatment (41) or Registration in COPAC.

Covariates

Covariates included offspring age at parental AUD treatment, number of parents with AUD, family education level, parental mental disorders, father’s year of birth and mother’s year of birth. Mental disorder among parents was defined as registration in the Danish National Patient Register or the Danish Psychiatric Central Research Register with any non-alcohol-related mental disorder before the offspring’s 15th birthday and was dichotomized as no mental disorder and any mental disorder. Information on family education level was obtained
from the Population's Education Register, which covers all educational achievements since 1981 for almost every person living in Denmark (42). Family education level was defined as the highest achieved educational level by either of the parents before the offspring’s 15th birthday and was categorized as basic or secondary, vocational, short, medium, high and unknown.

Statistical analyses

The analysis plan was not pre-registered, and results should thus be considered exploratory. Cox Proportional Hazards models with calendar time as the underlying time-axis were used to estimate the associations between family structure and offspring AUD. Follow-up began on the day after the offspring’s 15th birthday and ended at AUD onset (n=5,726), death (n=825), loss to follow-up (n=3,482) or end of study (March 2018) (n=98,051), whichever came first. AUD offspring and reference offspring contributed with a total of 152,128 and 1,548,973 (median 14.0 and 14.7) person-years. To account for the possibility of intragroup correlations, we used robust estimations of standard error. A group was defined as the offspring with parental AUD together with the corresponding reference offspring. The assumption of proportional hazards was examined graphically using log-log plots, and no violations were found. All hazard ratios were adjusted for the matching criteria (sex, age and living area), and adjusted hazard ratios were furthermore adjusted for covariates. Results did not differ noticeable between unadjusted and adjusted analyses; thus, only adjusted analyses are presented. Wald test was used to test for interaction between parental AUD and family structure, and between parental AUD and number of years of family intactness.
Data management was performed using SAS Software 9.4, SAS Institute, Cary, NC, USA and analyses were performed using STATA Version 15, StataCorp TX, USA. The significance level was set to a $P$ value of 0.05 (two-sided).

**Results**

**Cohort**

Table 1 lists characteristics of the 9,948 offspring with parental AUD and 98,136 reference offspring included in the study. AUD in a father (76.4%) was more common than in a mother (19.8%). Only 3.8% of the cohort had two parents with AUD. Offspring with parental AUD were on median 4 years when the parent entered AUD treatment. At parental AUD treatment start, 37.3% among offspring with parental AUD lived in an intact family, while this was the case for 74.2% among reference offspring at the corresponding time. Over the study period, offspring with parental AUD and reference offspring lived on median 2 year and 15 years in an intact family. During the offspring’s first 15 years, basic or secondary school was the highest attained family educational level for 32.8% of offspring with parental AUD and 16.1% of reference offspring. The prevalence of mental disorders in either a father, mother, or both parents was 59.6% among offspring with parental AUD and 14.7% among reference offspring.

[Insert Table 1]

**Family structure**

Figure 1 shows an overview of the family structures during upbringing among offspring with parental AUD and reference offspring. More non-intact families were found among offspring with parental AUD than among reference offspring. At birth, 30.9% of offspring with parental
AUD and 10.7% of reference offspring lived in a non-intact family. At offspring's 15\textsuperscript{th} birthday, the numbers had increased to 84.6% and 38.4%. Offspring from non-intact families, most often lived alone with their mother. At birth, 29.7% among offspring with parental AUD and 9.1% among reference offspring lived alone with their mother, while 0.3% among offspring with parental AUD and 0.2% among reference offspring lived alone with their father at the corresponding ages. At all ages, offspring with parental AUD more often lived apart from both parents compared with the reference offspring. Thus, at age 15, 14.1% among offspring with parental AUD and 1.8% among reference offspring lived apart from both parents (see Table S1 for numbers).

[Insert Figure 1]

Figure 2 presents an overview of family structures among offspring with parental AUD according to the gender of the parent with AUD. The proportion of offspring who grew up in an intact family did not differ considerably between offspring with paternal AUD and offspring with maternal AUD. Among those offspring who lived in a non-intact family, differences were, however, observed between offspring with paternal AUD and offspring with maternal AUD. At age 15, 4.2% among offspring lived alone with their father if he had AUD, while 36.2% among offspring lived alone with their mother if she had AUD. While 12.0% of offspring with paternal AUD did not live with both parents at age 15, this was the case for 25.8% of offspring with maternal AUD (see Table S2 for numbers).

[Insert Figure 2]
Family structure and offspring AUD

During follow-up, 1,118 (66.9% male) offspring with parental AUD and 4,608 (66.6% male) reference offspring developed AUD, corresponding to incidence rates of AUD of 7.4 and 3.0 per 1,000 person-years. Relative to reference offspring, offspring with parental AUD had a higher risk of AUD. Lower risk estimates were observed when adjusting for family structure at offspring’s 15th birthday and/or number of years in an intact family. No interaction was found between parental AUD and family structure, or between parental AUD and years of family intactness in relation to offspring AUD (Table 2).

[Insert Table 2]

Lowest risks of AUD were found among offspring who lived wholly or partly in an intact family during upbringing compared with those offspring who never lived in an intact family during upbringing. Thus, offspring with parental AUD who lived 5–9, 10–14 and 15 years in an intact family were at lower risk of AUD compared with offspring with parental AUD who never lived in an intact family. Among reference offspring, lower risks were found for those who lived 1–4, 5–9, 10–14 and 15 years in an intact family (Table 3). Table S3 shows the association between the number of years offspring with parental AUD lived in an intact family and AUD depending on the gender of the parent with AUD. The associations seemed to be independent of the gender of the parent with AUD.

[Insert Table 3]

No association was found between family structure at the time of parental AUD treatment and risk of offspring AUD. Offspring with maternal AUD who lived apart from both parents when the mother entered AUD treatment were, however, at higher risk of AUD compared
with offspring with maternal AUD who lived in an intact family when the mother entered AUD treatment (Table 4).

[Insert Table 4]

Discussion

In this register-based cohort study, we found that offspring with parental AUD grew up in markedly different family structures characterized by more non-intact families compared with offspring from the general population. Parental AUD was associated with a higher risk of AUD in the offspring. Moreover, non-intact family structure was associated with higher risks of offspring AUD, regardless of parental AUD status.

Strengths and limitations of study

Major strengths of this study include; the use of nationwide register data which enabled complete identification of parent-offspring relationships and detailed information on family structure; the large study population which enabled investigations of family structure depending on the gender of the parent with AUD; and up to 15 years of follow-up which allowed for long term investigation of the examined associations.

The results should be interpreted in the context of the study's limitations. The first, and perhaps most important, limitation concerns that the Danish Civil Registration System only allows registration of one residential address per individual at any time. It has become common that offspring spend time in each parent’s home following parental split up (43, 44), and it is therefore likely that offspring lived partly or had contact with the other parent which
they did not share residential address with. Secondly, we did not know whether offspring lived with other adults such as step- or grandparents. The presence of other adults in the household may either have provided compensating resources or may, on the other hand, have affected the offspring negatively. Thirdly, the temporality of parental AUD and parental split-up may be of importance for offspring AUD. However, we did not take this into consideration because individuals may have AUD for several years before appearing with AUD in the registers. Thus, register data may not be the most appropriate data source to study temporality of AUD and family structure. Fourthly, some misclassification of offspring AUD may have occurred, as not all individuals with AUD enter alcohol treatment (45), and among those who do, a higher severity of AUD and mental comorbidity is found relative to untreated individuals (46-51). It is therefore likely that only a subset of offspring with severe AUD were identified. We do, however, have no reason to believe that this misclassification was differential according to the exposure, thus any possible misclassification has, therefore, most likely attenuated the association between family structure and offspring AUD. Fifthly, another weakness was the risk of residual confounding as we, for example, did not have information on non-registered alcohol and other substance use disorders and on family characteristics such as quality of parent-child relationship. Finally, AUD status can vary over the life course as some will recover momentary or completely. We did not take this into consideration. However, we defined parental AUD to have occurred prior to the offspring’s 15th birthday. By doing it this way we were sure that the offspring were exposed to parental AUD during their childhood and not as adults.

Results in relation to other studies

Our finding of a higher risk of AUD among offspring of parents with AUD compared with offspring from the general population corresponds with results from other studies (52, 53).
Genetic factors are suggested to contribute substantially to the elevated risk (54), but parental AUD has also been linked to a lack of parenting (55), less parental resources and parental relationship quality (6) which altogether may add to the risk of offspring AUD.

While prior studies have indicated that offspring with parental AUD less often grow up in intact families compared with offspring from the general population (19-24, 56), we extended these findings by demonstrating that family structure already differed at birth and throughout the entire upbringing. We showed that 84.6% among offspring with parental AUD lived in a non-intact family at age 15, which is somewhat higher than in two large studies using Finnish (56) and Swedish (19) register data. These studies did, however, differentiate between the number of parents with AUD and results showed that considerably more offspring lived in a non-intact family if both parents were affected by AUD or alcohol/drug abuse. This may explain the divergent findings in relation to our results as we did not differentiate between the number of parents with AUD. We observed that offspring with parental AUD more often lived with their mother than father even though she was the one with AUD. Similar results have been noted earlier by others (19, 56). This finding may contribute to the understanding of why maternal AUD has been associated with higher risk of offspring AUD in a range of studies (57-59). Possible mechanisms that might explain why offspring with parental AUD more often grow up in a non-intact family include a family environment with high levels of conflicts, aggression and violence (30-32, 60), which consequently may end up in family dissolution.

We found an inverse association between number of years of family intactness and offspring AUD risk. To the best of our knowledge, no previous studies have investigated how family structure influence the risk of AUD among offspring with parental AUD, as done in this study.
Previous research has found a higher risk of substance use among adolescents of fathers with a drinking problem who live in a single-parent family relative to adolescents from an intact family (61). An intact marriage between parents has also been associated with a lower risk of AUD among offspring with parental AUD (62), while the combination of parental AUD and parental separation has been associated with higher risk of offspring early substance involvement and AUD than the risk incurred by either of the factors separately (63-66). Among adolescents from the general population, non-intact family structure during upbringing has been associated with more harmful drinking patterns (25-29). Different mechanisms may contribute to a higher risk of AUD among offspring from non-intact families compared with intact families. First, growing up in a non-intact family is associated with other outcomes, such as offspring mental health problems (67), which may independently increase the risk of AUD. Secondly, parental AUD is likely more severe and with more consequences to the family among those families that split up compared with intact families. Last, other family environment characteristics, such as parent-child relationship and parenting skills, may be of importance in the underlying relationship between family structure and offspring AUD. The present study does, unfortunately, not include such information due to the register-based study design.

We found no evidence for an association between family structure at time of parental AUD treatment and offspring AUD. Some studies have indicated that children of substance abusing parents are at higher risk of AUD, health and behavior problems if they live with the affected parent than if they live apart from the parent (19, 68). One study found that maternal substance use disorder (including AUD) was associated with higher risk of AUD (24). This contrasts with other studies which have found no evidence for a difference in AUD risk between offspring exposed to paternal AUD and offspring not exposed to paternal AUD (24,
Inconsistent findings were found among young people where living with a parent with alcohol problems was associated with some, but not all, aspects of harmful drinking patterns (21).

**Implications**

Although AUD runs within families (19-24), not all offspring of parents with AUD develop AUD. Identification of individuals at high risk for AUD who may benefit from preventive initiatives could contribute to hinder transmission of AUD across generations. Our results highlight the importance of parental AUD in conjunction with family structure when assessing for offspring AUD risk. The possible influence of family structure on risk of AUD in the offspring might be related to complex interplays between family characteristics, such as parenting skills, quality of parent-child relationship and family cohesion. Future cohort studies are needed to investigate these possible underlying mechanisms to gain a better understanding of the etiology of AUD.

**Conclusions**

We found that family structures differed considerably between offspring with parental AUD and offspring from the general population, as more offspring with parental AUD grew up in a non-intact family compared with offspring from the general population. Parental AUD was associated with a higher risk of offspring AUD, and non-intact family structure was associated with increased risk of offspring AUD, regardless of parental AUD.

**Acknowledgements**

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Data sharing
No additional data available.

Article information

Author contributions
Holst had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Holst is the guarantor.

All authors approved the final manuscript as submitted. The corresponding author attests that all listed authors meet authorship criteria and that no others meeting the criteria have been omitted.

Concept and design: All authors.
Acquisition, analysis, or interpretation of data: Holst, Becker, Tolstrup.
Drafting of the manuscript: Holst.
Critical revision of the manuscript for important intellectual content: All authors.
Obtained funding: Becker.

Ethics approval
The study was approved by the Danish Data Protection Agency (2012-58-0004; AHH-2015-018, I-Suite no: 03676). Because the study did not require any contact with the participants, no written informed consent was required according to Danish law.
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41. Danish Health Authority. Fællesindhold for registrering af alkoholikere i behandling [Shared content for registration of alcoholics in treatment]. København: Danish Health Authority; 2016.
Figure 1. Family structure during upbringing among (a) offspring with parental AUD (b) reference offspring.\footnote{At each age, only offspring of parents who are registered with AUD at that particular time are included.}
Figure 2. Family structure during upbringing among (a) offspring with paternal AUD and (b) offspring with maternal AUD. 

At each age, only offspring of parents who are registered with AUD at that particular time are included.
Table 1. Characteristics of offspring with parental AUD and reference offspring

<table>
<thead>
<tr>
<th></th>
<th>Offspring with parental AUD&lt;sup&gt;1&lt;/sup&gt; (N=9,948)</th>
<th>Reference offspring (N=98,136)</th>
</tr>
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<tbody>
<tr>
<td><strong>Men, n (%)</strong></td>
<td>5,139 (51.7)</td>
<td>50,680 (51.6)</td>
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<tr>
<td><strong>Year of birth, median n (5&lt;sup&gt;th&lt;/sup&gt;-95&lt;sup&gt;th&lt;/sup&gt; percentile)</strong></td>
<td>1985 (1972-2000)</td>
<td>1985 (1972-2000)</td>
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<tr>
<td><strong>Parent(s) with AUD, n (%)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father</td>
<td>7,602 (76.4)</td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td>1,972 (19.8)</td>
<td></td>
</tr>
<tr>
<td>Both</td>
<td>374 (3.8)</td>
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<tr>
<td><strong>Age (years) at time of parental AUD, median (5&lt;sup&gt;th&lt;/sup&gt;-95&lt;sup&gt;th&lt;/sup&gt; percentile)</strong></td>
<td>4 (0-13)</td>
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<td><strong>Family structure at parental AUD treatment, n (%)</strong></td>
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<tr>
<td>Living with both parents</td>
<td>3,743 (37.3)</td>
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<tr>
<td>Living with mother only</td>
<td>5,272 (53.0)</td>
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<td>Living with father only</td>
<td>4687 (4.7)</td>
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<td>Living with neither parent</td>
<td>403 (4.1)</td>
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<tr>
<td>Unknown</td>
<td>63 (0.6)</td>
<td>2,585 (2.6)</td>
</tr>
<tr>
<td><strong>Number of years lived with both parents, median (5&lt;sup&gt;th&lt;/sup&gt;-95&lt;sup&gt;th&lt;/sup&gt; percentile)</strong></td>
<td>2 (0-15)</td>
<td>15 (0-15)</td>
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<td><strong>Family education level&lt;sup&gt;1&lt;/sup&gt;, n (%)</strong></td>
<td></td>
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<td>Basic or secondary school</td>
<td>3,262 (32.8)</td>
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<td>3,858 (38.8)</td>
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<td>1953 (1938-1969)</td>
<td>1954 (1939-1970)</td>
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<td>1957 (1943-1972)</td>
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<td>Parental AUD offspring</td>
<td>Reference offspring</td>
<td>Risk of offspring AUD</td>
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<td>Person years</td>
<td>Offspring AUD incidence</td>
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<td>1,548,973</td>
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<tr>
<td>1</td>
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<td>1.74–2.02</td>
<td>1.47–1.72</td>
<td>1.48–1.72</td>
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<td>Basic + family structure 2</td>
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<tr>
<td>3</td>
<td>Basic + years of family intactness 3</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Basic + family structure + years of family intactness 4</td>
<td></td>
</tr>
<tr>
<td>22,761</td>
<td>121</td>
<td>970,545</td>
</tr>
<tr>
<td>5</td>
<td>Years in intact family 5</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>15th birthday 5th</td>
<td></td>
</tr>
<tr>
<td>0–4</td>
<td>94,953</td>
<td>793</td>
</tr>
<tr>
<td>1</td>
<td>Adjusted for sex, date of birth, living area, age at parental AUD treatment, number of parents with AUD, family educational level, parental mental disorders, father’s year of birth and mother’s year of birth.</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Adjusted for 1 and further adjusted for family structure at offspring’s 15th birthday</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Adjusted for 1 and further adjusted for number of years in intact family</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Adjusted for 1 and further adjusted for family structure at offspring’s 15th birthday and number of years in intact family</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Family structure at offspring’s 15th birthday*parental AUD</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Number of years in intact family*parental AUD</td>
<td></td>
</tr>
<tr>
<td>5–9</td>
<td>26,099</td>
<td>160</td>
</tr>
<tr>
<td>10–15</td>
<td>31,076</td>
<td>165</td>
</tr>
</tbody>
</table>

*Adjusted for sex, date of birth, living area, age at parental AUD treatment, number of parents with AUD, family educational level, parental mental disorders, father’s year of birth and mother’s year of birth.  
*Adjusted for 1 and further adjusted for family structure at offspring’s 15th birthday  
*Adjusted for 1 and further adjusted for number of years in intact family  
*Adjusted for 1 and further adjusted for family structure at offspring’s 15th birthday and number of years in intact family  
*Family structure at offspring’s 15th birthday*parental AUD  
*Number of years in intact family*parental AUD
Table 3: Person years, incidences and hazards ratios (95% CI) for offspring AUD by years of family intactness from birth to 15th birthday among offspring with parental AUD and reference offspring

<table>
<thead>
<tr>
<th>Years in intact family</th>
<th>Parental AUD offspring</th>
<th>Reference offspring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Offspring AUD incidence</td>
<td>Risk of offspring AUD</td>
</tr>
<tr>
<td>0</td>
<td>42,81 373</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>52,14 420</td>
<td>0.98 (0.85 to 1.13)</td>
</tr>
<tr>
<td>1–4</td>
<td>26,09 160</td>
<td>0.79 (0.66 to 0.96)</td>
</tr>
<tr>
<td>5–9</td>
<td>15,77 90</td>
<td>0.75 (0.59 to 0.94)</td>
</tr>
<tr>
<td>10–14</td>
<td>15,30 75</td>
<td>0.67 (0.52 to 0.87)</td>
</tr>
</tbody>
</table>

1Adjusted for sex, date of birth, living area, age at parental AUD treatment, number of parents with AUD, family educational level, parental mental disorders, father’s year of birth and mother’s year of birth.
Table 4. Person years, incidences and hazards ratios (95% CI) for offspring AUD by family structure at time of parental AUD treatment among offspring with paternal AUD and offspring with maternal AUD

<table>
<thead>
<tr>
<th>Parental AUD offspring</th>
<th>Reference offspring</th>
</tr>
</thead>
<tbody>
<tr>
<td>Person years</td>
<td>Offspring AUD incidence</td>
</tr>
<tr>
<td>Not living with either parent</td>
<td>3,738</td>
</tr>
<tr>
<td>Living with father only</td>
<td>2,376</td>
</tr>
<tr>
<td>Living with mother only</td>
<td>69,132</td>
</tr>
<tr>
<td>Living with both parents</td>
<td>46,425</td>
</tr>
</tbody>
</table>

¹Adjusted for sex, date of birth, living area, age at parental AUD treatment, number of parents with AUD, family educational level, parental mental disorders, father’s year of birth and mother’s year of birth.