TITLE PAGE

Young adult predictors of alcohol dependence up to age 53: A 44-year prospective cohort study of Danish men

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ABSTRACT

Aims To examine (1) if there is a positive association between drinking volume in young men and lifetime risk of alcohol dependence (AD) and (2) if there are other associations between young adulthood factors and lifetime risk of AD.

Design Prospective cohort study of sons of fathers with alcohol use disorder (AUD) and matched low-risk controls without paternal AUD.

Setting and participants A total of 204 men, who were assessed at baseline in 1979 at age 19-20 years, were followed through record linkage with Danish registers and consecutive psychiatric interviews at the ages of 33, 43 and 53 years.

Measurements AD diagnoses were interview-based according to the Diagnostic and Statistical Manual of Mental Disorders, Third Edition or made by treating clinicians according to the International Classification of Diseases (ICD) Revision 8 (ICD-8) until 1993 and Revision 10 (ICD-10) from 1994. We estimated odds ratios (ORs) with 95% confidence intervals (CI) for the development of AD after adjustment for confounders including smoking, social status, and paternal AUD.

Findings The following variables from the examination at age 19-20 independently predicted lifetime AD: alcohol consumption > 21 beverages/week vs 0-21 (OR 2.46, 95% CI 1.22-4.97), police contact (OR= 2.60, 95% CI 1.28-5.28) and institutionalization related to the individual (OR=2.90, 95% CI 1.39-6.02). Compared with <1 beverages/week, the risk for AD did not increase significantly for drinking volume categories: 1-7, 8-14, or 15-21 beverages/week.

Conclusion Independently of other risk factors in young adulthood, young Danish men’s risk for lifetime alcohol dependence appears to be predicted by a drinking volume at age 19-20 years exceeding 21 beverages per week.
Introduction

Alcohol use disorders (AUD) are common psychiatric disorders affecting approximately 5% of the adult population worldwide, and with a prevalence that is consistently higher in high-income countries (1). A study in the US has recently found a lifetime prevalence of severe AUD around 14% and suggests that the prevalence of AUD may have increased during the last 10 years (2). AUD often goes untreated (2) and is highly comorbid with other psychiatric conditions or substance use disorders (3). Studies have shown that the prevalence of AUD is highest during adolescence, peaks during young adulthood, and then decreases hereafter (4–6).

AUD and alcohol dependence (AD) aggregate in families (7) and involve a significant genetic contribution (8–11). Studies in individuals with a family history of AUD has a long tradition (12–15) beginning with relatively small samples of sons of fathers with AUD and low-risk controls (16,17). Four decades ago, young male participants in The Danish Longitudinal Study on
Alcoholism at high or low risk were, among other things, asked about their alcohol consumption (17) paving the way for a prospective study of the role of alcohol consumption in young adulthood as a potential predictor of AD in midlife. The compulsive use of alcohol in individuals has been hypothesized to be driven by multiple sources of reinforcement that change with an individual’s movement from social use to abuse to alcohol dependence (18). From a longitudinal perspective, relatively little is known about drinking volume in young adulthood and alcohol-related outcomes in the long term. A register-based study observed a positive association between amount of alcohol consumed and risk of alcoholism in middle aged men and women; an effect that was independent of confounders such as smoking (19). Moreover, among middle-aged men, the risk of alcoholism did not increase significantly before 22-41 drinks per week (19). Recent findings among young people indicate that early problematic alcohol use does not necessarily always mature out during the subsequent years (20,21). However, cohort studies restricted to young adults with problematic alcohol use may not capture the full picture, because the incidence of alcohol-related disorder is not confined to the very young adults (22).

We used a sub-cohort of The Danish Longitudinal Study on Alcoholism (204 men interviewed at age 19-20) and aimed to examine if there is a positive association between drinking volume in young men and risk of AD until midlife (age 53) independently of other putative risk factors such as high-risk status, proxies of externalizing behaviour (police contact or institutionalization related to the individual) and smoking. Because a previous Danish cohort study (19) found that the risk of male alcoholism did not increase significantly before 22-41 drinks per week, we hypothesized an association between drinking volume > 21 beverages per week and increased risk of subsequently developing AD. In The Danish Longitudinal Study on Alcoholism, young individuals were, besides...
questions about alcohol consumption, asked about upbringing circumstances (including institutionalization related to the individual), smoking, police contact, and general emotional problems (15,17). The goals of this study was to examine (1) if there is a positive association between drinking volume in young men and lifetime risk of alcohol dependence (AD) and (2) if there are other associations between young adulthood factors and lifetime risk of alcohol dependence (AD).
Method

The study sample and its elements

Figure 1: Overview of the most important elements of the study

In 1979, a baseline investigation (N=204) of participants (born 1959-61) aged 19-20 was carried out. Between 1979 and 2013, all 204 participants were followed up through Danish registers and alcohol-related diagnoses were registered. At age 33, 43 and 53, respectively, surviving participants from the original cohort were invited to participate in interview-based follow-ups.

In 1979, a sample of 204 young men aged 19-20 years was comprehensively assessed (17). Here, 2/3 of participants (N=134) were sons of fathers with AUD and 1/3 (N=70) were matched controls without paternal AUD (17,23). Participants were originally drawn from the Copenhagen Perinatal Cohort (CPC) of 9,125 individuals born to 8,949 mothers during 1959-1961 (24). A comprehensive search was conducted in the Danish Psychiatric Register (25) and in alcohol treatment clinics of greater Copenhagen for the biological father of the CPC members (17,23). Male members of the CPC with fathers treated for AUD became eligible as participants in the high-risk group. For every two high-risk boys, a single control participant was selected from the remaining pool of cohort members whose fathers were not treated for AUD. They were matched to the high-risk group according to birthday, mother's age, parity number, father's socioeconomic status, and their parents'
marital status (17). Daughters were not included because the lifetime prevalence of female AUD was substantially lower than the prevalence in males at the time of initiation of the study.

Exposures

In 1979, all 204 participants underwent the baseline assessment, which included questioning individuals about upbringing characteristics and family structure, intake of alcohol and drugs, police contact, institutionalization related to the individual and behavioral problems (26) (27). We focused on the following exposures: (1) Alcohol consumption: the number of self-reported beverages per week were recorded semi-quantitatively as 12 non-overlapping categories (0, 1-7, 8-14, 15-21, 22-28 etc.). A Danish standard drink (beverage) contains 12 g of alcohol, corresponding to one beer, one glass (1/6 of a bottle) of wine or four centiliters of 40% proof spirits. The quantitative alcohol consumption was divided into the following groups: >21 beverages per week (the recommended drinking limits in Denmark) or below (2) Smoking: recorded as yes/no (3) Police contact: recorded as yes/no (4) Institutionalization related to the individual: recorded as yes/no (5) High-risk vs low risk: sons of fathers with AUD vs sons without a father with AUD as described previously (17), and (6) social status: a semi-quantitative variable measured on a 1-8 point scale based on information about breadwinner’s occupation, education, type of income and quality of housing when the cohort member was 1 year old (28). Data on parental social status was missing for 45 cohort members (22%): when parental social status was included in statistical models, the overall mean was substituted for missing values, and a dummy variable indicating missing values was included (28).

Follow-up
The *current sample* is defined as all 204 men, who participated in the 1979 baseline interview (Figure 1). We have subsequently traced vital status and other relevant information on all these individuals by Danish registers. A total of 84.3% of the cohort members were followed-up by interview at least once.

**Data sources based on Danish registers**

All participants from the 1979 baseline investigation were followed in Danish registers until October 31st, 2015. Information was obtained from registers with national coverage and linked using each study participant’s personal identification number (PIN). The PIN is a unique ten-digit number assigned to all Danish citizens, and because it is used across all Danish registries, linkage across registers is possible (29). The Danish Civil Registration System provides information on family relations, sex, date of birth, emigration status and vital status since 1968 (30). The Danish National Patient Register provides information on admissions to non-psychiatric hospital departments since 1977 (31) and since 1995, the same information from emergency units and outpatient clinics have been registered. The Danish Psychiatric Central Research Register includes information on psychiatric admissions to mental hospitals and psychiatric departments since 1969 (32). Individuals who sought treatment for alcohol-related problems in alcohol clinics or hospital-settings were identified in these registers and were given an alcohol-related diagnosis by the treating clinician according to the International Classification of Diseases (ICD) Revision 8 (ICD-8) (33) (until 1993) and Revision 10 (ICD-10) (since 1994) (34).

For all deceased individuals in Denmark, death is recorded in the Danish Register of Causes of Death (35), and the death certificate contains information about underlying and contributory causes of death, manner of death, data from any postmortem examination (e.g. autopsy and toxicological
analyses), and other information that could pertain to the circumstances surrounding death, such as an accident while intoxicated. A senior Danish psychiatrist (JK) carefully reviewed and extracted information about, for instance, diagnoses of AD for these individuals.

**Interview-based diagnoses**

Individuals were invited by letter or telephone to a follow-up at age 33 (36,37) and subsequently at age 43 (15) and lastly at age 53 (15). When appropriate, they were reminded of their participation 10 years before. Participants were home-interviewed or interviewed at Copenhagen Municipal Hospital by a senior psychiatrist (PJ or JK). The interview typically lasted 2 hours. The diagnostic evaluation was based on the Psychiatric Diagnostic Interview, fourth edition (PDI-R) (38) and later modified for the DSM-III and-IV editions. The PDI is a descriptive, fully operationalized, criterion-referenced and structural diagnostic interview covering 20 basic DSM psychiatric syndromes, including alcohol abuse and dependence. Also, several self-report tests were administered (15) and the interviewer recorded up to six lifetime DSM-III-R diagnoses to characterize the psychiatric history of the participants. The DSM-III-R criteria were used at 33-, 43- and 53-year follow-ups to ensure diagnostic consistency.

**Register-based diagnosis**

Thirty-two individuals (15.7%) were non-participants in follow-ups. We used Danish registers to identify AD in participants as well as non-participants. The criteria were (1) any registration in Danish registers with ICD-8 code 303.3 (including all sub-levels) or any ICD-10 code F 10.2 (including all sub-levels).
**Definition of the study outcome**

Based on all available information, we were able to identify living or deceased individuals with a history of AD until age 53. We assigned cohort members a diagnosis of AD if they had (1) received an interview-based DSM-III-R diagnosis or (2) an ICD-8 or ICD-10 register-diagnosis of AD according to Danish registers and the individual had not been interviewed at any of the 3 follow-ups (i.e. this was relevant for the 32 non-participants only). If both a DSM-III-R diagnosis and an ICD 8 or ICD 10 diagnosis had been assigned, the interview-based DSM-III-R diagnosis was used.

**Statistical analysis**

All available diagnostic information from the 3 follow-ups was used to measure the proportion of the entire sample that had been assigned a lifetime diagnosis of AD at the end of study. We investigated the OR of developing AD for each separate potential predictor using univariate analyses (model 1). Model 2 investigated multivariable effects of all 6 predictors/risk factors using adjusted logistic regression models using SPSS (IBM, SPSS, version 22.0). Overall, the level of statistical significance was $P < .05$, and tests were 2-sided.

Because the analysis had not been pre-registered on a publicly available platform, the results should be considered exploratory.

The Study was approved by the Danish Scientific Ethics Committee and the Institutional Review Board of the University of Kansas Medical Center.
Results

Interview participation rates and mortality

Out of 172 participants that had been interviewed at least once, 169 had been interviewed at age 33 (135 at age 43 and 111 at age 53). Out of 172 participants that were ever interviewed, 9 (5.2%) had died at a mean age of 50.6 years (range 44-55). Out of 32 non-participants, 3 (9.4%) had died at a mean age of 53.3 years (range 52-54). Out of 204 participants from the baseline investigation, 12 (5.9%) had died by age 53 (12.3% among those having received a diagnosis of AD and 2.9% among the rest of the cohort).

Development of AD among interviewed and non-interviewed

Out of 172 interviewed, 58 (33.7%) had been assigned an interview-based DSM-III-R AD diagnosis during lifetime and almost all (98%) of those with any interview-based AD diagnosis had already been interviewed at age 33. Out of 58 with any DSM-III-R AD diagnosis during lifetime, 41.4% had already received this interview-based DSM-III-R AD diagnosis by age 33. Out of 172 ever interviewed, 24 (14.0%) had received a register-based alcohol related diagnosis according to either ICD-8 or ICD-10 (the mean age at first alcohol registration was 37.4 years).

Among the 32 non-participants, 7 persons (21.9%) had received a register-based alcohol-related diagnosis according to either ICD-8 or ICD-10 (mean age at first alcohol registration 29.0 years).

In total, there were 65 individuals (31.9%) out of the entire sample of 204 with a lifetime diagnosis of AD according to our outcome definition (58 were diagnosed according to DSM-III-R and 7 according to ICD-8 or ICD-10).
Predictors of AD during lifetime

Findings presented in Table 1 show that drinking volume, smoking, institutionalization related to the individual and police contact univariably predicted lifetime risk of AD. Parental AUD (high-risk status) and social status were not significant predictors in this study. Drinking > 21 beverages per week, when compared to lower alcohol intake, was associated with a 2.4 fold (95% 1.44-4.11) higher odds of developing AD (model 1). We found that 37.3% drank > 21 beverages per week. Additional multivariable analyses (Table 1, model 2) showed that the mutually adjusted odds associated with drinking > 21 beverages per week was 2.4-2.5 fold higher when compared to the reference drinking category. Findings indicate that neither smoking, paternal AUD, social status, institutionalization of the individual or proxies of externalizing behavior such as police contact explained this association between drinking volume and risk of AD.

Neither paternal predisposition (high-risk background) nor current smoking were significant independent predictors of AD in this sample. Almost 45% in the sample had earlier police contact at age 19-20 and this variable emerged as a robust independent predictor of AD with a more than twofold higher risk estimate, after mutual adjustment for other risk factors. About 30% of individuals in the sample had a history institutionalization at some time in their life and this variable robustly predicted a more than twofold elevated risk of AD.

Further analyses of alcohol consumption and risk of AD

Table 2 shows that the risk of AD varied according to the semi-quantitative categories of weekly beverage intake (<1, 1-7, 8-14, 15-21, 22-28 and >28). Using <1 beverages/week as reference, the risk did not increase significantly in any of the categories: 1-7, 8-14, and 15-21. For the categories
22-28 and >28, the risk increased significantly in some, but not all, of the adjusted analyses. For instance, adjustment for police contact and history of institutionalization attenuated the effect of drinking > 28 beverages/week. Further analyses using alcohol intake as a semi-quantitative variable showed that an incremental change in alcohol-intake category was associated with ORs of 1.39 (95% CI 1.13-1.57) in unadjusted analyses and 1.25 (95% CI 1.05-1.50) in mutually adjusted analyses.
Discussion

Main findings

This prospective cohort study of 204 sons of fathers with AUD and matched low-risk controls examined whether there is a positive association between drinking volume in young men and risk of AD during lifetime and if there are other associations between young adulthood factors and lifetime risk of alcohol dependence (AD). Young men had a 2.4-fold higher lifetime risk of AD if they self-reported > 21 beverages a week of alcohol consumption when compared to those with lower consumption extending and corroborating previous research (19). A drinking volume > 21 beverages a week predicted AD independently of other risk factors (contact with police, institutionalization related to the individual, smoking and paternal AUD).

Current Danish drinking limit recommendations (sensible drinking limits) for alcohol intake are 14 and 21 drinks for women and men, respectively. However, at the time (1979) when participants of this cohort were interviewed at baseline, public campaigns to inform about sensible drinking limits (39) were not yet started. Seen in the light of AD, the present study confirms the fact that these limits may be relevant for young men with respect to risk of developing AD.

A history of any police contact was associated with significantly higher risk of subsequently developing AD. This association may reflect underlying conditions including accident proneness and our results seem broadly consistent with findings of other longitudinal studies of predictors of AUD among young adults suggesting a link between externalizing behavior in youth and later alcohol-related diagnosis. (40–44) Previous investigations in this high-risk cohort suggest ADHD
and conduct disorders in childhood as factors predicting AD (44). Obviously, a police contact does not *per se* say much about a person’s possible criminal history (i.e. both very mild and serious offences would be captured into this non-specific variable). We also found that institutionalization related to the individual predicted AD independently of e.g. individual’s own alcohol consumption or of paternal AUD. This corroborates recent findings from a Danish register-based study suggesting that children from non-intact family structures have an increased risk of developing alcohol problems regardless of parental AUD (45). We found that 30% of participants had reported institutionalization at the individual level and the association with developing AD later in life may reflect a wide range of underlying factors. Sometimes institutionalization is reserved as an option to shelter young individuals from their exposure to consequences of substance abuse, childhood adversity, disrupted caregiving, violence etc. However, children may also, themselves, have manifested early behavioural deviance that brought them into early contact with the social service system in Denmark and in turn into contact with a family institution.

Our multivariable analyses indicated non-significant associations between familial high-risk status and AD. We found an association that was somewhat weaker than reported in the literature (28,46,47) and this may reflect a combination of low statistical power and the fact that 24.3 % of our “low risk” sample developed AD during lifetime. Individuals at “low risk” could have had fathers (or mothers) with unregistered AUD or diagnosed before the national health registers were computerized in 1969 and 1977, respectively. Also, strict matching on sociodemographic background characteristics to families in which the father had AUD could have implied that low-risk controls were exposed to environmental factors such as family disruption, which appear to increase the risk of developing AUD independently of parental history of AUD (28)(45).
Strengths and limitations

Strengths of our study include the multidisciplinary etiologic approach and the attractive design features that made it possible to focus on the “premorbid” assessment of AUD (17). The long period of follow-up, the complete register-based follow-up and the ability to follow a relatively large proportion of the cohort are among other study strengths (84.2% cohort members and 89.2% of all cases with AD were interviewed at least once). The same two psychiatrists (JK and PJ) completed the structured interviews to assess AD or AUD diagnoses and to characterize the psychiatric history of the cohort members.

The study has several limitations including a potential for residual confounding. We explored 6 potential predictor variables in a relatively small sample, and we recommend that findings are sought replicated in larger samples drawn from the general population. Despite having gathered information from all death certificates, some misclassification of the outcome of AD could have occurred among those who had died prematurely. However, in a conservative sensitivity analysis assuming that all death individuals (including individuals whose death certificate did not contain an alcohol-related diagnosis) would have developed AD if they had lived longer), our risk estimates remained largely unchanged. There remains a concern that alcohol dependent persons could have both refused to participate in interviews and not have been captured in Danish registers, but this potential bias could hardly have turned our general results in the direction of no-effect. Also, detailed lists of coding practices for the 1979-interview were not available and the original baseline information does not contain details. There are also limitations due to the study design which disproportionally oversampled only sons of men with AUD. We know today that both sons and daughters of parents with AUD are at higher risk of AUD than the general population (28,46), but
we cannot conclude that the significant predictors found in this all-male sample can be generalized to youth of both sexes. Also, limited sample size meant that some of the non-significant findings (e.g. for smoking) could be subject to type-II error. Moreover, some of the young adult males might have had milder alcohol problems at baseline, although in a 1986 paper by Schulsinger et al. on the 204 participants, none were reported to meet research criteria for alcoholism (48) or ICD-8 criteria or DSM-III for alcoholism at the time (17).

**Conclusion**

Our findings demonstrate the utility of including drinking volume in alcohol-epidemiologic studies aimed at identifying predictors of AD throughout lifetime. In this all-male cohort study, drinking > 21 beverages/week at age 19-20 independently predicted alcohol dependence thus providing further support to the already available evidence suggesting that a cut-off around 21 beverages is relevant in Danish men (19). Other predictors of AD were contact with police and having been reared in family institutions. Findings may have implications for public campaigns to inform about potential health implications of drinking habits in young adulthood.
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Table 1: Variables at age 19-20 predicting lifetime risk of AD

<table>
<thead>
<tr>
<th>Variable</th>
<th>N (% exposed)</th>
<th>Lifetime risk (%) of AD</th>
<th>Model 1 (unadjusted)</th>
<th>Model 2 (mutually adjusted)</th>
</tr>
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<tr>
<td>High-risk status at baseline</td>
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<tr>
<td>No</td>
<td>70 (34.3)</td>
<td>24.3</td>
<td>1.00 (ref.)</td>
<td>1.00</td>
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<td>Yes</td>
<td>134 (65.7)</td>
<td>35.8</td>
<td>1.74 (0.91-3.34)</td>
<td>1.58 (0.74-3.40)</td>
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<td>N. of beverages per week</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-21</td>
<td>127 (62.3)</td>
<td>22.8</td>
<td>1.00 (ref.)</td>
<td>1.00</td>
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<td>&gt;21</td>
<td>77 (37.3)</td>
<td>46.8</td>
<td>2.44 (1.44-4.11)</td>
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<td>Smoking</td>
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<td>No</td>
<td>94 (46.1)</td>
<td>24.5</td>
<td>1.00</td>
<td>1.00</td>
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<td>Yes</td>
<td>110 (53.9)</td>
<td>38.2</td>
<td>1.91 (1.04-3.50)</td>
<td>1.11 (0.54-2.28)</td>
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<td>Police contact</td>
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<td></td>
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<tr>
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<td>19.3</td>
<td>1.00</td>
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<td>Yes</td>
<td>91 (44.6)</td>
<td>45.1</td>
<td>3.44 (1.83-6.45)</td>
<td>2.60 (1.28-5.28)</td>
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<td>Family institution, ever</td>
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<td>22.4</td>
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<td>Yes</td>
<td>61 (29.9)</td>
<td>54.1</td>
<td>4.09 (2.16-7.75)</td>
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<td>---</td>
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<td>---</td>
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</tr>
<tr>
<td>Social status, linear effect</td>
<td></td>
<td></td>
<td>0.89 (0.78-1.02)</td>
<td>0.98 (0.84-1.14)</td>
</tr>
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</table>

### Table 2: Weekly alcohol intake at baseline and odds ratio (95% CI) for developing AD during lifetime.

<table>
<thead>
<tr>
<th>N of beverages per week</th>
<th>Unadjusted</th>
<th>Adjusted for smoking only</th>
<th>Adj. for smoking, risk status and social status</th>
<th>Adj. for smoking, risk status, social status, institutionalization and police contact</th>
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<tr>
<td>&lt;1</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>1-7</td>
<td>0.38 (0.12-1.26)</td>
<td>0.38 (0.11-1.24)</td>
<td>0.35 (0.10-1.16)</td>
<td>0.28 (0.08-1.00)</td>
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<tr>
<td>8-14</td>
<td>1.18 (0.45-3.08)</td>
<td>1.11 (0.42-2.94)</td>
<td>1.06 (0.39-2.83)</td>
<td>0.97 (0.33-2.80)</td>
</tr>
<tr>
<td>15-21</td>
<td>1.77 (0.62-5.05)</td>
<td>1.75 (0.61-5.04)</td>
<td>1.99 (0.68-5.88)</td>
<td>1.97 (0.62-6.30)</td>
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<td>21-28</td>
<td>2.64 (0.97-4.14)</td>
<td>2.93 (1.06-6.09)</td>
<td>3.22 (1.13-9.19)</td>
<td>2.92 (0.94-9.09)</td>
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<td>&gt;28</td>
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<td>2.59 (1.04-6.43)</td>
<td>2.28 (1.00-5.79)</td>
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