Alcohol-Induced Impairment in Adolescents Admitted to Inpatient Treatment After Heavy Episodic Drinking: Effects of Age and Gender

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ABSTRACT. Objective: In Germany and many other countries, the number of adolescent heavy episodic drinking–induced hospital admissions (HEDHA) in pediatric units markedly increased during the past decade. A low level of response to alcohol in young adults is associated with high risk for later development of alcohol use disorders (AUDs). **Method:** We performed a retrospective chart review of all 1,123 HEDHA cases in adolescents aged 11–17 years who were admitted to one of the pediatric inpatient units covering the cities of Dresden, Pirna, and Rostock, Germany, between 2000 and 2008. Blood alcohol concentration (BAC) and Glasgow Coma Scale (GCS) measures on admission were documented in 846 cases. **Results:** The mean (SD) BAC was 155 (50) mg/100 ml full blood, and *M* (SD) GCS was 12.21 (3.02).

These parameters were negatively correlated with each other ($r = -.256$, $p < .001$), indicating more impairment at higher BACs. To describe a numerical estimate of how severely a subject was compromised relative to his BAC, the GCS scores were inverted (making high scores indicate severe impairment) and divided by BAC. The resulting alcohol-induced impairment index (AIII) was significantly influenced by an interaction between age and gender, decreasing with age in boys but increasing in girls. **Conclusions:** During adolescence, alcohol-induced impairment develops differently in boys and girls, which may be because of the girls' developmental edge. The high variability of observed AIII might help to predict the risk for later AUDs in the emergency department, simply by measuring BAC and GCS. (*J. Stud. Alcohol Drugs*, 76, 493–497, 2015)

THE CONTINUOUSLY INCREASING PREVALENCE of adolescent heavy episodic drinking (HED) with the aim of deliberately getting intoxicated has raised concerns in most countries of the Western world during the last decade. Intoxications resulting in emergency pediatric inpatient treatment (heavy episodic drinking–induced hospital admission [HEDHA]) comprise an extreme variant of drinking and should be expected to have even worse consequences. Although the incidence of this phenomenon has increased considerably over the last years (Kuželová et al., 2009; Schöberl et al., 2008), only one recent study systematically described some biomedical determinants of HEDHA (Van Zanten et al., 2013).

Alcohol use disorders (AUDs) typically begin during adolescence (Paus et al., 2008). Their age of onset shows

a maximum between 16 and 20 years (Brown et al., 2008). Early age at first drink (Buchmann et al., 2009) and high subsequent drinking (Meyers & Dick, 2010) are important determinants for the development of AUDs. A self-evident explanation possibly linking these observations is that the adolescent brain may be particularly vulnerable to alcohol exposure. This notion is one of the reasons why underage drinking is prohibited. Data from animal studies, however, suggest a more differentiated picture. Some alcohol effects, such as motor impairment and sedation, can actually be less pronounced in adolescent compared with adult rodents (Spear, 2000). However, acute alcohol-induced cognitive impairment has been shown to be more pronounced in adolescent than adult rats (Land and Spear, 2004; Markwiese et al., 1998).

A relatively low level of response to alcohol's intoxicating effects has been reported in young adult humans with a genetic risk for alcoholism (Schuckit et al., 2004) and is associated with an increased risk to develop alcohol dependence within the next decade (Trim et al., 2010). For obvious ethical and legal restrictions, alcohol research in human adolescents is almost exclusively observational, and we are aware of only one experimental study (Behar et al., 1983), which investigated behavioral and physiological ethanol effects in children ages 8–15 years after inducing a mean blood alcohol concentration (BAC) of 50 mg/100 ml, which is a relatively low level of intoxication.

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This is why we considered adolescent extreme HED leading to HEDHA as a natural experiment offering the opportunity to study individual differences in the response to acute alcohol effects. In a retrospective chart review, we took advantage of the meticulous documentation of BAC and Glasgow Coma Scale (GCS). The GCS was initially developed to enable physicians, nurses, and other medical staff to give a quick, reliable, and objective assessment concerning consciousness of patients after a head trauma (Teasdale & Jennett, 1974). It rates eye opening, verbal communication, and motor activity in response to commands and, if necessary, to painful stimuli. The sum of these three subscales can vary from 3 (indicating deep coma) to 15 (a fully awake uncompromised person); even a GCS of 13 describes a severely compromised patient. By now, use of the GCS has been extended from head trauma to many other acute medical situations and is validated in adolescents (Borgman et al., 2011; Levin & Eisenberg, 1979). People with life-threatening alcohol intoxication present with progressive inability to open their eyes, communicate verbally, and perform coordinated movements. These symptoms are the same in adults and adolescents and are specifically assessed by the GCS. Consequently, the GCS has previously been used to score consciousness in patients with severe drug and alcohol intoxication (Eizadi Mood et al., 2011; Heard & Bebart, 2004).

BAC alone does not provide us with enough information to be able to rate alcohol-induced impairment because identical BACs can lead to various stages of adverse effects in humans. In contrast, GCS scores facilitate a clear picture of impairment. The aim of this exploratory study was to provide an easy description of HEDHA based on BAC and GCS.

Method

Patient sample

The study protocol was approved by the Ethical Review Board of the Technische Universität Dresden. Cases were recruited at pediatric inpatient units in four hospitals in two states in Germany: Saxony (University Hospital Dresden, Municipal Hospital Dresden-Neustadt, and Klinikum Pirna GmbH) and Mecklenburg-West Pomerania (University Hospital Rostock). We identified 1,123 cases of patients up to age 17 whose diagnoses on discharge included "acute alcohol intoxication" or "external cause for morbidity and mortality: toxic effects of ethyl alcohol" between January 1, 2000, and December 31, 2008. Because these hospitals cover the entire pediatric inpatient care of the regional residents below age 18, we are confident that we included all alcohol-related admissions occurring during this time interval. Exclusion criteria were the following: lacking documentation of BAC ($n = 24$) or GCS ($n = 157$) and any documented

concurrent intoxication with other illegal substances ($n = 117$).

Data acquisition

We conducted a systematic retrospective chart review, extracting data on patient characteristics (gender, age, GCS, BAC). To describe a numerical estimate of how severely a patient was compromised relative to his BAC, the GCS scores were inverted (e.g., 15 converted to 3, 14 to 4, 13 to 5, etc., making high scores indicative of strong impairment) and divided by BAC. Therefore, high numbers of the resulting alcohol-induced impairment index (AIII) reflect strong impairment relative to BAC. Alcohol concentrations in full venous blood were determined by enzymatic assays from samples obtained immediately after admission and are expressed in mg/100 ml full blood (e.g., 80 mg/100 ml is equivalent to .08% or .8‰).

Statistical analysis

AIII was the primary dependent variable and was analyzed for effects of gender, age, and their interaction by an analysis of covariance (ANCOVA). As a nominal level of statistical significance, $p = .05$ was accepted.

Results

Effects of age and gender on alcohol tolerance

The mean (SD) of AIII was 4.29 (3.31). We excluded the 13 cases whose AIII differed more than 3 SD from the mean, leaving 846 cases (312 females) for analysis. Their mean BAC was 155 (50) mg/100 ml (range: 31–345 mg/100 ml). Mean (SD) of individual GCS scores was 12.21 (3.02) (range: 3–15). The Pearson's correlation coefficient for the relation between BAC and GCS was $r = -.256$, $p < .001$, explaining 6.55% of the variance. A bar chart showing the influence of age and gender on AIII is displayed in Figure 1.

ANCOVA revealed significant effects of the between-subject variable gender, $F(1, 845) = 8.31$, $p = .004$, and its interaction with age, $F(1, 845) = 8.6$, $p = .003$, but no main effect of age on AIII. Inspection of Figure 1 led us to assume that the gender effect on AIII inverted at a critical age of 16. We therefore ran an ANOVA with age as a dichotomous variable (≤ 15 vs. ≥ 16) and gender, which detected no main effects but reproduced the Age $\times$ Gender interaction, $F(1, 845) = 11.1$, $p = .001$. Bonferroni-corrected, one-sided post hoc tests with contrasts revealed that AIII was marginally lower in girls than boys ages up to 15, $M (SD) = 3.78 (2.30)$ versus 4.29 (2.03), $p < .1$, but significantly higher in girls than boys age 16 or older, 4.34 (2.49) versus 3.79 (2.09), $p < .05$. Among the boys, AIII was significantly higher in the younger compared with the older group, $M (SD) = 4.29 (2.03)$ versus

FIGURE 1. Effects of age and gender on alcohol-induced impairment index (AIII) scores in 846 adolescents. Data are mean and *SD*.

3.79 (2.09), $p < .05$, whereas this age difference was only marginal in girls, 3.78 (2.30) versus 4.34 (2.49), $p < .07$).

Because cases from this sample were treated in four hospitals in two states of Germany, we included these variables as additional covariates into the above-described ANCOVA model. Neither state nor hospital had a significant effect on AIII, whereas gender and Gender $\times$ Age interaction still significantly influenced AIII.

Subjects who were excluded from the study were significantly younger than the included cases, $M(SD) = 15.6(1.24)$ years versus 15.5 (1.32) years; $t(1,307) = 2.19$, $p = .029$. There was no gender difference between groups.

Discussion

Our main purpose was to explore how the level of response to alcohol is influenced by age and gender in adolescents after extreme HED. The main results were that (a) AIII, defined as the ratio of inverted GCS scores and BAC, varied considerably between subjects, and (b) AIII decreased with

age in boys but increased in girls, defining an Age $\times$ Gender interaction. We observed a broad distribution of both BAC (31–345 mg/100 ml) and GCS scores (15–3). The correlation between BAC and GCS was rather weak, explaining less than 10% of the variance. This implies that impairment because of alcohol use was highly variable among the subjects of our sample, with AIII scores varying between 1.07 and 13.33. Observing such variation is consistent with the concept described by Schuckit et al. (2004), who demonstrated in numerous studies, that a low level of response to ethanol (i.e., low AIII scores) is related to heavier drinking, more alcohol-related problems, and an increased risk of future alcoholism (Trim et al., 2010).

AIII was influenced by age and gender. The two interacted significantly because AIII scores decreased with age in boys but increased in girls. Age- and gender-dependent factors such as body height and weight or liver volume cannot account for the observed variability of AIII because we measured BAC, which already accounts for all of these influences. There are, however, two factors that may cause

alcohol-induced impairment to change with aging, namely that early onset of repeated drinking results in acquisition of tolerance, and that specific brain maturation processes are in progress.

Developing tolerance to alcohol can easily explain why AIII decreased with age in boys. However, German epidemiological surveys suggest that the frequency of risky drinking is the same in boys and girls up to age 15, and that girls do not cut down their drinking during the age period of 16 to 17, which would weaken previously acquired tolerance (Lampert & Kuntz, 2014). Therefore, drinking habits offer no good explanation why AIII was higher in girls at older (≥ 16) than younger (≤ 15) ages. On the other hand, the higher AIII in older compared with younger girls may be in line with the above-described observations in rodents: rats tested at peak puberty show fewer signs of alcohol intoxication compared with young adult animals, which suggests that AIII should increase with age during the transition from late adolescence to young adulthood. Peak puberty occurs around age 15 in German girls (Kahl et al., 2007), and the associated brain maturation processes take place about 1–4 years later in boys than girls (Lenroot et al., 2007). Owing to our age range covering only 12–17 years, we may not have been able to observe decreasing AIII in boys because it may occur later, i.e., during early adulthood. We acknowledge that relating the above-described rodent findings to our results is highly speculative because it is unclear whether the age ranges correspond to each other. However, we are not aware of any animal or human data showing how alcohol-induced impairment changes during the entire transition from late childhood over early, mid, and late puberty into young adulthood.

The major limitation of this study is certainly missing data. Because this is a retrospective review of pediatric chart notes, not all parameters had been documented in all cases and were not of sufficient quality to use. Another limitation is the lack of longitudinal data to assess whether the AIII score is actually able to predict long-term outcomes in adolescent heavy episodic drinkers. Future research should therefore include prospective studies and follow-up assessments. As mentioned before, we excluded cases with any documented concurrent intoxication with other illegal substances. Unfortunately, this information was not available in all cases. The likelihood that some of the highly impaired cases included still had used other drugs before hospital admission is quite high. However, because this study mainly focuses on the importance of low impairment despite high BAC, which might possibly be a predictive factor for the development of AUD later in life, we accepted this limitation.

The minor age difference of 0.1 years between included and excluded cases cannot be considered as clinically significant or explain the observed age effects on AIII. Our results bear two implications concerning health care providers who are faced with severely intoxicated adolescents. First, after

HEDHA, clinicians have to deal with the difficult question of whether a bedside brief intervention is sufficient in a given patient (Carey et al., 2007) or whether he or she should be referred to specialized services offering psychosocial aftercare. Given the limited resources of prevention services, the AIII score might be an easily obtained telltale sign helping to identify adolescents who are at particularly high risk for AUDs, at least in settings where the emergency rescue services apply the GCS and BAC as routinely measured on admission.

Second, low impairment despite high doses of alcohol was revealed to be affected by age and gender rather than acquired tolerance at young ages, which implies that these traits cannot be modulated by affected patients. Therefore, it is debatable whether communicating this fact should be part of the counseling. Simply telling patients that they are at increased risk for AUDs but cannot do anything about the cause actually might dishearten them and thereby thwart the therapeutic goal of enhancing self-efficacy (Atwell et al., 2011). On the other hand, patients may not know that their ability to drink more than their peers is a mixed blessing, and educating them about how this fact may lead to higher consumption, more brain damage, and ultimately higher risk for AUDs may be new and interesting to them. Conveying this information can also be used for motivational interviewing by guiding the patient's reflections toward the self-evident conclusion that drinking more carefully is important and an effective approach to contain their risk.

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