Alcohol consumption among the general population has traditionally been a great concern worldwide, especially among adolescents: 26.5% of all 15-19-year-olds usually consume alcohol often in heavy drink sessions (World Health Organization [WHO, 2018]). Prevalence rates for alcohol consumption are higher among the European region (43.8%), followed by the region of the Americas (38.2%) and the Western Pacific region (37.9%). Within the American region, Ecuador has one of the lowest consumption patterns, although is experiencing an increase in both average per capita consumption (reporting 4.4 average liters in 2016) and prevalence of heavy episodic drinking (21.3%) of the adult general population (WHO, 2018).

From a developmental point of view, adolescence is characterized by an increased interest in new experiences with poor inhibitory control, which may promote impulsive actions and, consequently, increase the chances of experimenting with drugs (for example, alcohol), turning adolescents more vulnerable to addiction (Beil-Gawelczyk et al., 2014; Secades-Villa et al., 2013) and other related problems (Green et al., 2016; Silins et al., 2018).

El papel de la edad de inicio en el consumo problemático de alcohol: ¿artefacto o efecto de cohorte?

La edad de inicio se ha señalado como un predictor crucial del consumo de alcohol en la adolescencia. Sin embargo, varios autores han advertido que se trata de un artefacto poco fiable que se explica por la tendencia a retrasar la edad de inicio a medida que aumenta la edad de los encuestados (forward telescoping o sesgo de memoria). Este estudio pretende comprobar si la edad de inicio es una medida aproximativa de la edad o un predictor del consumo de alcohol por sí mismo. Se utilizó un modelo de regresión jerárquica y la Macro Process para SPSS para probar el efecto de la edad de inicio (controlando la edad) en el consumo de alcohol y el papel mediador/moderador de esta edad de inicio, el control parental y el sexo en el consumo de alcohol, respectivamente. Los resultados confirmaron el papel de la edad de inicio como predictor significativo del consumo de alcohol en los adolescentes ecuatorianos en lugar de artefacto. Además, la edad de inicio medió completamente el efecto de la edad en el consumo de alcohol, con frecuencia moderado por el sexo. Se discuten posteriormente las implicaciones prácticas.
Several risk factors are related to alcohol use and abuse among adolescence, such as peer pressure (Duncan et al., 2006), low self-concept (Cornellà-Font et al., 2020), as well as low self-esteem (Fuentes et al., 2020), presence of mental health problems and stress (Anthenelli & Grandison, 2012), or parental substance use (Parolin et al., 2016). It also seems important to take into account low parenting control (e.g., see Lamborn et al., 1991; Steinberg et al., 1994) as a risk factor for problematic alcohol consumption, highlighting the relevance of this key variable to prevent such consumption (Garcia et al., 2019), for example by strengthening communication and warmth between the parents and their children (e.g., Garcia et al., 2020). Sex differences have also been extensively found in research about alcohol consumption (Hermens et al., 2013; Nealis et al., 2016). In general, being a woman is a protective factor, associated with no consumption or low-risk consumption, while being a man is a risk factor, associated with risk consumption. In the Latin American context, higher alcohol consumption among adolescents has been related with lowered parental control (Strunin et al., 2013) and neglectful or dysfunctional parenting style (Conçalves dos Reis & Marques de Oliveira, 2015; Schofield et al., 2015).

Age of onset represents another age-related factor frequently associated in the literature with alcohol use during adolescence. That is, an earlier age of drinking onset has turned out to be a strong predictor of increased risk for alcohol problem severity and alcohol use disorders (Hingson et al., 2006; Ohanessian et al., 2015). Moreover, those who start drinking earlier are more likely to get involved in dangerous practices and use other licit or illicit substances (Pilatti et al., 2016; Rial et al., 2020). Nonetheless, while some studies indicate a clear relationship between both variables (Haug et al., 2014; Hingson et al., 2006), other studies have claimed lack of reliability for self-reported measures of age of onset due to factors like forward telescoping (i.e., the tendency to delay age of onset as respondents’ age increases; see Golub et al., 2000; Shillington et al., 2012), or its low impact on alcohol use (Kuntsche et al., 2015). However, other studies have highlighted that the existence of forward telescoping does not seem to affect the association between age of onset and subsequent alcohol use (Rogers & Jackson, 2017). Two studies have claimed to have found forward telescoping effects using large longitudinal data (Golub et al., 2000; Shillington et al., 2012), but there are some issues with these studies: (1) the estimations for forward telescoping effects are quite different in both studies; (2) Golub et al. (2000) found that biases in the estimation of age of onset for successive cohorts of respondents could appear in both directions, with forward telescoping being a little more frequent than backward telescoping. It is possible that the existence of both forward and backward telescoping, also found by Rogers & Jackson (2017), only reflects the imprecision of subjective recall, being forward telescoping slightly more frequent just because it is more conservative, but with negligible effects on the predictive power of age of onset; (3) Shillington et al. (2012) have issues with the estimation of age of onset, as ages of 11 or less were all coded as “11”, and was unable to clearly separate forward telescoping effects from attrition, where participants involved in substance use at an early age become less likely to participate in successive surveys (Golub et al., 2000); finally (4), these longitudinal studies, while focusing in differences between successive waves of respondents, fail to consider the cohort effect underlying alcohol consumption, which are transversal, not longitudinal.

With the purpose of overcoming this controversy, the present study aims to investigate whether age of onset is just a proxy for age (consistent with the hypothesis of forward telescoping) or if age of onset represents a different predictor of alcohol consumption of its own. In addition, we explored, first, if age of onset and parental control mediate the relationship between age and alcohol consumption and, second, if sex moderates that effect.

**Method**

**Participants**

The original sample consisted of 1,950 students from the cities of Loja (N = 1,367) and Zamora (N = 583) aged between 12 and 18 years old, and studying in public (N = 582), subsidized (N = 1,056), and private (N = 312) centers; 52.9% of the sample were males (N = 1031) and 47.1% were females (N = 919). However, participants who failed to report age of onset, either because they had never tried alcohol (N = 888), because they did not remember (N = 28), or due to missing data (N = 24) were excluded. Additionally, in order to avoid having potential drinkers labelled as abstainers, only those participants older than the highest age of onset (14.5) were considered. The final sample was composed of 759 participants between 14 and 18 years old (431 males, 56.8% and 328 females, 43.2%). Mean age was 15.67 ± 1.26 years old, and mean age of onset was 13.50 ± 1.35 years old.

**Measures**

A sociodemographic questionnaire was provided to the students. Collected information included participants’ age, age of onset of alcohol consumption, and degree of parental control. Age of onset was presented in the questionnaire as an ordinal variable, with a two-year interval (8–9, 10–11, 12–13, and 14–15). This was devised in order to facilitate participants to estimate the moment they first had tried alcohol using a wider time frame, instead of asking for a specific year. For analysis purposes, each of the categories shown was scored as the midpoint of the interval (e.g., for the interval 8-9, the score assigned would be 8.5). Parental control was also presented as an ordinal variable with four possible answers to the question: “How often do your parents or legal tutor know your whereabouts?” (they never know, they almost never know, they sometimes don’t know, or they always know).

The Spanish version of the Alcohol Use Disorders Identification Test (AUDIT, Self-report version; Babor et al., 2001) was used for the measurement of alcohol consumption. It consists on a 10-item questionnaire designed to screen hazardous alcohol intake. Participants respond to each question by indicating the frequency of alcohol consumption and/or symptoms related to problematic drinking, from 0 = never to 4 = 4 or more times a week, giving a maximum possible score of 40. Higher scores indicate a higher risk of problematic alcohol consumption and a score of 8 or above is considered as a cut-off point for hazardous consumption in males, being 7 or above for females (Conigrave et al., 1995). For our original sample, internal consistency of AUDIT test was .746.

**Design and Procedure**

A multi-center cross-sectional study was conducted. Centers were contacted before the study was carried out, and the purpose of the study was exposed to the headmaster for both administrative and parental consent. All procedures were performed in accordance with the ethical standards of the institutional research committees and with the 1975 Helsinki Declaration and its later amendments in 2013. Data gathering took place at the classroom and both the AUDIT scale and the questionnaire took the students between 30 and 40 minutes to complete.

**Data Analysis**

All data analyses were performed using the Statistical Package for the Social Sciences (SPSS), version 21 for Mac (IBM Spain, Madrid, Spain). The descriptive analysis of the sample included...
the means and standard deviations ($M \pm SD$) for the quantitative variables, while frequencies and percentages were used for the nominal variables. Both parametric and non-parametric tests were conducted to examine sex differences in alcohol consumption, age of onset, and degree of parental control, and non-parametric correlations were later calculated between the dependent variable (alcohol consumption), the independent variable (age), and the mediators (age of onset and parental control). Then, a hierarchical multiple regression was conducted to examine the effect of age and age of onset on AUDIT scores. Detection of multicollinearity was performed using the variance inflation factor (VIF), with VIF > 5 as cut-off point for diagnosis of collinearity (Sheather, 2009). For the final sample ($N = 759$), the distribution according to age after the removal of younger participants remained similar for both sexes ($M_{males} = 15.67, M_{females} = 15.66$), with no significant differences ($t_{1948} = -1.949, p = .051$). As expected, there were clear and significant differences between both groups (Mann-Whitney's $z = -4.432, p < .001$) with respect to alcohol consumption ($M_{males} = 4.05, SD = 5.83; M_{females} = 2.66, SD = 4.33$). With respect of age of onset, no significant differences were found between males ($M = 13.61$) and females ($M = 13.72$). Finally, there were clear and significant differences between both sexes (Mann-Whitney's $z = -7.252, p < .001$) in parental control ($M_{males} = 3.47, M_{females} = 3.68$).

Table 1 shows the descriptive statistics for both the original and final samples in the variables of interest. It can be appreciated that the final sample has an increased mean age, due to suppression of participants younger than 14 years old, and increased mean alcohol consumption scores due to removal of abstainers.

### Results

#### Comparison between the Original ($N = 1,950$) and Final ($N = 759$) Samples in the Variables of Interest

In general, the percentage of abstainers in the original sample was 51.8%, slightly below the 53.5% reported by the WHO (2018) for Ecuador in 2016; a binomial test showed no significant differences between both percentages. Correspondingly, the prevalence of alcohol consumption (48.2%) was very high and significantly superior to previous results registered in Ecuador throughout adolescent samples (44.8%; Consejo Nacional de Control de Sustancias Estupefacientes y Psicotrópicos [CONSEP, 2012]) and college students (46.09%; Mejía et al., 2011).

Several tests were performed for both samples. Due to the nonnormality of AUDIT scores and the ordinal nature of both age of onset and parental control, nonparametric tests (Mann-Whitney's $U$) were performed for these variables.

For the original sample ($N = 1,950$), the distribution according to age was fairly similar both for males ($M = 14.84, SD = 1.76$) and females ($M = 14.68, SD = 1.79$), with no significant differences between sexes ($t_{1949} = -1.949, p = .051$). As expected, there were clear and significant differences between both groups (Mann-Whitney's $z = -4.432, p < .001$) with respect to alcohol consumption ($M_{males} = 4.05, SD = 5.83; M_{females} = 2.66, SD = 4.33$). With respect of age of onset, no significant differences were found between males ($M = 13.61$) and females ($M = 13.72$). Finally, there were clear and significant differences between both sexes (Mann-Whitney's $z = -7.252, p < .001$) in parental control ($M_{males} = 3.47, M_{females} = 3.68$).

### Relationships between Alcohol Consumption, Age, Age of Onset, and Parental Control

In order to test for relationships between the quantitative and ordinal variables of our model, non-parametric correlations were computed between the dependent variable (self-reported alcohol consumption), the independent variable (age), and mediators (age of onset and parental control). Table 2 shows the Spearman's correlation coefficients between these variables.

#### Table 2. Non-parametric Correlations between Alcohol Consumption (AUDIT score), Age, Age of Onset, and Parental Control

<table>
<thead>
<tr>
<th>Variable</th>
<th>AUDIT score</th>
<th>Age</th>
<th>Age of onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>$r$</td>
<td>.111**</td>
<td></td>
</tr>
<tr>
<td>Age of onset</td>
<td>$r$</td>
<td>-.112*</td>
<td>.324***</td>
</tr>
<tr>
<td>Parental control</td>
<td>$r$</td>
<td>-.253**</td>
<td>.074*</td>
</tr>
</tbody>
</table>

$p < .05,*p < .01,**p < .001.$

All variables are significantly correlated with each other. There is a clear positive relationship between age and age of onset; however, correlation is not as strong as to expect problems of multicollinearity. At the same time, there are also weaker, but significant, relationships between age and alcohol consumption (positive), and between age of onset and alcohol consumption (negative). Finally, parental control is significantly related to both age of onset (positive) and alcohol consumption (negative), and less so with age (positive). Table 3 summarizes the two main independent variables of our
study for the final sample: age and age of onset, according to sex. As the correlation coefficient suggested in Table 2, there is a clear positive relationship between both variables, as younger participants tended to start drinking earlier than older participants, who started later. The “forward telescoping” hypothesis attributes this to a mere tendency to adjust age of onset to present age. Using the PROCESS macro, we tested if age of onset mediates the effect of age on alcohol consumption or, in other words, if there is a tendency towards precocity, suggesting that the relationship between age and alcohol consumption in fact reflects a cohort effect.

### Table 3. Cross-tabulation of the Participants according to Sex, Age, and Age of Onset

<table>
<thead>
<tr>
<th>Sex</th>
<th>Age of onset</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>14</td>
<td>15</td>
</tr>
<tr>
<td>Female</td>
<td>8.50</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>10.50</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>12.50</td>
<td>45</td>
</tr>
<tr>
<td></td>
<td>14.50</td>
<td>16</td>
</tr>
<tr>
<td>Total females</td>
<td>73</td>
<td>87</td>
</tr>
<tr>
<td>Male</td>
<td>8.50</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>10.50</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>12.50</td>
<td>64</td>
</tr>
<tr>
<td></td>
<td>14.50</td>
<td>27</td>
</tr>
<tr>
<td>Total males</td>
<td>103</td>
<td>96</td>
</tr>
<tr>
<td>Total Age of onset</td>
<td>8.50</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>10.50</td>
<td>21</td>
</tr>
<tr>
<td></td>
<td>12.50</td>
<td>109</td>
</tr>
<tr>
<td></td>
<td>14.50</td>
<td>43</td>
</tr>
<tr>
<td>Total</td>
<td>176</td>
<td>183</td>
</tr>
</tbody>
</table>

### Sex Differences on Alcohol Consumption by Age of Onset, Parental Control, and Age

In a second step, interactions between sex and the variables in the model were graphically tested. Figure 1 shows the mean age of onset for male and female participants for three different levels of age corresponding, respectively, to the mean age minus 1 SD (14 years old), the mean age (15.5 years old), and the mean age plus 1 SD (17 years old). Apart from mean differences between sexes in age of onset, there seems to exist a slight moderator effect of sex in the relationship between age and age of onset, as initial differences between males and females for older participants (right) with respect to their age of onset tend to disappear for younger participants (left). This would indicate not only a tendency towards precocity for the whole sample (a cohort effect), but also that this tendency apparently gets accelerated in the case of female participants, until mean differences between sexes disappear for the youngest subjects. 

Figure 2 shows the mean parental control for male and female participants for the same three levels of age. While there are clear differences in parental control between males and females across the whole age spectrum, this difference tends to decrease for successive cohorts. While parental control remains constant for male subjects, it shows a tendency to decrease across female cohorts (right to left). This tendency is more dramatic than Figure 2 seems to suggest, because it shows that 14-year-old females already experience less parental control than their 17-year-old counterparts and, therefore, it is expected that they will experience even less parental control in 3 years’ time, when they are 17. Similarly, the apparent stability of parental control for males is really indicating that 14-year-old males already experience the same degree of parental control than 17-year-old males, and this control will be lowered when they become 17.

Lastly, Figure 3 shows mean AUDIT score for male and female subjects for the same three cohorts. Although mean consumption levels are smaller for younger participants when compared to older subjects (showing the effect of age on alcohol consumption), there is a clear cohort effect in the tendency towards reduced differences between sexes, which are much greater for older cohorts (right), smaller for intermediate cohorts (center), and negligible for the younger cohorts (left).
In summary, our preliminary results show that while there is a clear and significant relationship between age (cohort) and alcohol consumption, this relationship could be masking two different effects: one related specifically with age (i.e., the tendency towards greater alcohol consumption as age increases; see Figure 3), and another reflecting a cohort effect (i.e., the tendency towards precocity in alcohol consumption for younger generations; see Figure 1). Moreover, both statistical tests and graphical analyses indicate the existence of a moderator effect of sex for both precocity in alcohol consumption (see Figure 1), parental control (see Figure 2), and alcohol consumption (see Figure 3), and this moderator effect seems to operate differently for both sexes, as females become similar to males in all these aspects at once.

Table 4 summarizes the effects of the independent variable (age), mediators (age of onset and parental control), and the moderator variable (sex) on alcohol consumption, measured by the square root of the AUDIT score. We found that both age of onset and parental control completely mediated the relationship between age and alcohol consumption. Furthermore, the interaction between sex and age also affected this relationship.

These results support the notion that once included in the model the cohort effects mediated by age of onset ($b_1 = -0.090, t_{755} = -2.669, p = .008$) and parental control ($b_2 = -0.347, t_{755} = -5.837, p < .001$), and the interaction between sex and age ($X*W = 156, t_{755} = 2.26, p < .05$), there is no direct effect of age on alcohol consumption ($c' = 0.056, t_{755} = 1.038, p = .300$).

The Index of Moderated Mediation suggests that the indirect effect of age of onset on alcohol consumption did not vary as a function of sex (95% CI [-.002, .033]): the conditional indirect effect was similarly negative both for males (95% CI [-.064, -.006]) and for females (95% CI [-.043, -.004]), indicating that an earlier age of onset was associated with higher alcohol consumption, and vice versa, irrespective of sex, and after controlling for age.

The index of moderated mediation also indicated that the effect of parental control on alcohol consumption did vary as a function of sex (95% CI [0.002, .07]). This effect was significant and negative for females (95% CI [-.055, -.009]), but not for males (95% CI [-.021, .025]).

With respect to the direct effects of age on the mediators, and in a similar fashion to the index of moderated mediation, the effect of age on age of onset did not vary as a function of sex ($X*W = -0.139, t_{755} = -1.847, p = .065$), but the effect of age on parental control did vary according to sex ($X*W = -0.218, t_{755} = -2.195, p = .030$). The first effect indicates a tendency towards precocity for both sexes. The second effect can be visualized in Figure 2, were the slope showing the effect of age on parental control reveals a tendency towards lowered parental control for successive cohorts of females ($X*W = 0.868, t_{755} = 2.657, p = .008$), and no tendency at all for males ($X*W = -0.008, t_{755} = -0.281, p = .779$). The complete model and all relevant coefficients can be visualized in Figure 4.

**Analysis of Moderated Mediation**

In order to test if the effects of age of onset, parental control, and sex were involved in the relationship between age and alcohol consumption, a moderated mediation model (model 8) was tested using the PROCESS macro. Specifically, we tested if age of onset and parental control mediated the relationship between age and alcohol consumption and the moderator effect of sex, both for the relationship between age and the mediators, and for the relationship between age and alcohol consumption.

Table 4 presents the results for the moderated mediation model. The table shows the effects of age (X), sex (W), and their interaction (X*W) on the mediators (age of onset and parental control) and alcohol consumption (Y), as well as the moderated mediation effects.

**Table 4.** Moderated Mediation Effects of Age of Onset and Parental Control on the Effect of Age on Alcohol Consumption (AUDIT score), Moderated by Sex

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Coef.</th>
<th>$B$</th>
<th>SE</th>
<th>$T$</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>DV: AUDIT score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>$c'$</td>
<td>0.056</td>
<td>0.054</td>
<td>1.038</td>
<td>.300</td>
</tr>
<tr>
<td>Age of onset</td>
<td>$b_1$</td>
<td>-0.090</td>
<td>0.034</td>
<td>-2.669</td>
<td>.008</td>
</tr>
<tr>
<td>Parental control</td>
<td>$b_2$</td>
<td>-0.347</td>
<td>0.059</td>
<td>-5.837</td>
<td>.000</td>
</tr>
<tr>
<td>Sex</td>
<td>$W$</td>
<td>-2.112</td>
<td>1.082</td>
<td>-1.952</td>
<td>.051</td>
</tr>
<tr>
<td>Age x Sex</td>
<td>$X*W$</td>
<td>0.156</td>
<td>0.070</td>
<td>2.259</td>
<td>.024</td>
</tr>
</tbody>
</table>

Index of moderated mediation

<table>
<thead>
<tr>
<th>Mediator</th>
<th>$W$</th>
<th>Index</th>
<th>Boot SE</th>
<th>Boot LLCI</th>
<th>Boot ULCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>Sex</td>
<td>.125</td>
<td>0.009</td>
<td>-.002</td>
<td>.033</td>
</tr>
<tr>
<td>Parental control</td>
<td>Sex</td>
<td>.032</td>
<td>0.016</td>
<td>.002</td>
<td>.066</td>
</tr>
</tbody>
</table>

Conditional indirect effects

<table>
<thead>
<tr>
<th>Mediator</th>
<th>$W$</th>
<th>Boot indirect effect</th>
<th>Boot SE</th>
<th>Boot LLCI</th>
<th>Boot ULCI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>Female</td>
<td>-.033</td>
<td>0.015</td>
<td>-.064</td>
<td>-.006</td>
</tr>
<tr>
<td>Age of onset</td>
<td>Male</td>
<td>-.021</td>
<td>0.010</td>
<td>-.043</td>
<td>-.025</td>
</tr>
<tr>
<td>Parental control</td>
<td>Female</td>
<td>-.030</td>
<td>0.012</td>
<td>-.055</td>
<td>-.009</td>
</tr>
<tr>
<td>Parental control</td>
<td>Male</td>
<td>.003</td>
<td>0.012</td>
<td>-.021</td>
<td>.025</td>
</tr>
</tbody>
</table>

Note. Confidence intervals are reported with a bootstrap sample size = 5,000. LLCI = lower level of the 95% bootstrap percentile confidence interval; ULCI upper level of the 95% bootstrap percentile confidence interval. Sex was coded such that female = 0 and male = 1. Testing each mediator separately revealed conceptually analogous results.
Discussion

The aim of this study was to examine the relationship between age, age of onset, parental control, and alcohol consumption in both males and females. To our knowledge, this is the first study that examines whether age of onset mediates the relationship between age (which, in a cross-sectional study like this, also indicates generation, or birth cohort) and alcohol consumption among a sample of Ecuadorian adolescents. Several major conclusions can be extracted from our study.

First, our results show prevalence rates for alcohol consumption significantly higher than those reported in the past for adolescents in Ecuador, and the percentage of abstainers is smaller, although not significantly, than the one reported by WHO (2018) for the adult population in Ecuador. This is suggesting that the generations included in our study are already drinking more than those in the past.

Second, results of this study suggest a cohort effect, defined by a tendency towards precocity for alcohol consumption onset in successive generations of both males and females, consistent with previous research (Shillington et al., 2012). Precocity, defined as age of onset in alcohol consumption, predicted higher alcohol consumption rates regardless of sex. This finding is consistent with previous studies (Hingson et al., 2006), and particularly relevant considering that low age of alcohol use onset has traditionally been associated with higher risk of alcohol use disorders (AUDs) (Salamó-Avellaneda et al., 2010), where young adulthood remains a critical period for increased risk in alcohol dependence (Newton et al., 2011). However, considering that younger age of onset is associated with higher rates of dependence during adolescence for males rather than females (Hartford et al., 2005; Palmer et al., 2009), sex differences should be studied further; our initial statistical tests supported this by indicating a slightly, but significantly delayed age of onset for females.

Third, a significant decrease in parental control for successive cohorts of females was found. This effect is more pronounced than it seems, given that the apparent lack of differences between cohorts of male adolescents are indicating that 14-year-olds are experiencing the same level of parental control than 17-year-olds. In the case of females, 14-year-olds are already experiencing less parental control than 17-year-olds. This is important because reduced parental control may facilitate precocity. Therefore, the result is congruent with differences between successive cohorts of females.

Fourth, age of onset and parental control did mediate the relationship between adolescence and alcohol consumption, suggesting that age differences involve differences in both age of onset and parental control, which were moderated by sex. These mediating and moderator effects do not support the hypothesis of forward telescoping as the explanation for the relationship consistently found in the literature between age and age of onset. Rather, results are consistent with cohort effects implicit in generational changes affecting alcohol consumption for both men and women. Forward telescoping might bias subjective recall of age of onset, but it fails to fully account for the relationship found between age and age of onset. As both Golub et al. (2000) and Rogers and Jackson (2017) recognized, forward telescoping bias (more frequent than backward telescoping) is disorganized and chaotic, inconsistent with the systematic and positive relationship existing between age and age of onset. Furthermore, the effects of age, age of onset, parental control, and sex found in our study indicate a systematic, more general, cohort effect, whose effects are manifest in the form of changes in parental control and age of onset for alcohol consumption for men and women of successive generations.

In sum, our results support the notion that first contact with alcohol happens earlier in new generations as perception of parental control decrease, especially for females. Age of onset and parental control did mediate the effect of age on alcohol consumption. Finally, the hypothesis of “forward telescoping” fails to account for the results of this study. If age of onset was merely a proxy for age, only partial mediation or absence of it should be expected between age and alcohol consumption. Overall, the results of this research support the role of age of onset on alcohol consumption, not as an artefact, but as a significant predictor of such behavior. Furthermore, these conclusions highlight the importance of age of onset and parental control in future interventions aimed at reducing alcohol-related problems among Ecuadorian adolescents.

Results of this study should be taken with caution. The cohort effect may be affected by populations where alcohol consumption rates between men and women have been similar for extended periods of time, such as European countries.

Conflict of Interest

The authors of this article declare no conflict of interest.

References

Alcoholism, Clinical and Experimental Research, 29(5), 810-828. 
https://doi.org/10.1007/s10587-016-0772-3

https://doi.org/10.1186/1471-2458-14-1202


https://doi.org/10.1080/08897077.2014.989153

https://doi.org/10.1016/j.drugalcdep.2009.01.012

https://doi.org/10.3389/fpsyg.2016.00887

https://doi.org/10.3389/fpsyg.2017.01452

https://doi.org/10.4978/adicciones

https://doi.org/10.1080/1067828X.2017.1305928


https://doi.org/10.1016/j.drugalcdep.2015.10.020

https://doi.org/10.20882/adicciones


https://doi.org/10.1080/1067828X.2012.710026

https://doi.org/10.1111/add.14263

https://doi.org/10.2307/113416

https://doi.org/10.1016/j.addbeh.2013.06.011
