

Individual psychological vulnerability factors related to the initiation or increase in alcohol use in early adolescence: a longitudinal study

Vulnerability factors in alcohol use adolescence

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Funding statement: The study was supported by a grant from the Spanish Ministry of Health, Social, Services and Equality (National Plan of Drugs 2017/039).

Acknowledgments

We are grateful to the individuals who kindly participated in the study.

Authors' contribution

CMP, JJFM, EVM, and LMGM specifically developed the conceptualization and methodology for this study. PMG, RGG, and CPT carried out the evaluations and data collection. CMP and JJFM conducted data curation and formal analysis. CMP, JJFM, EVM, and LMGM contributed to the interpretation of the data and writing the original draft. LMGM contributed to funding acquisition. LMGM and EVM contributed to the project administration and supervision. All the authors contributed to review and editing the final manuscript.

Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this manuscript.

Abstract

Objective: This longitudinal study aims to explore several individual psychological vulnerability factors that could predict the onset or escalation of alcohol use in early adolescents. **Method:** A total of 792 adolescents participated in all stages of this study. Participants were divided into two groups based on their alcohol use patterns: no drinking group (ND) and light drinking group (LD). Cognitive, personality, and neuropsychological measures were assessed using questionnaires, and alcohol use was evaluated at baseline (T1). After two years, alcohol use was reassessed (T2) and participants were categorized into three groups: no drinking group (ND), light drinking group (LD) and heavy episodic drinking group (HED). **Results:** Results indicated that low inhibition, high sensitivity to reward, high openness to experience and high motor impulsivity were primary predictors of the transition from no alcohol use (T1) to alcohol use (T2), regardless of subsequent drinking trajectory (LD or HED). The transition from LD (T1) to HED (T2) was primarily predicted by low inhibition, high cognitive impulsivity, high sensitivity to reward, and sex. **Conclusions:** This longitudinal study provides a novel approach to the study of HED initiation in adolescence. The findings expand our understanding of the risk of psychological factors associated with adolescent alcohol initiation, enabling clinicians to tailor primary and secondary prevention programs in schools and other relevant settings.

Keywords. Alcohol use, early adolescence, risk factors, disinhibition, impulsivity

Introduction

Alcohol use and heavy episodic drinking (HED) often emerge during adolescence, a critical period of brain development with profound implications for cognitive, emotional, and social development (Boer et al., 2024; Johnston et al., 2020; Marshall, 2014; Patrick et al., 2024; Ryan et al., 2019). Previous research has consistently demonstrated that initiating alcohol use during early adolescence is associated with more severe consequences (Hingson et al., 2006; Lee et al., 2024), including increased risk of neurocognitive and neurobehavioral impairments, poor academic performance, and earlier engagement in hazardous drinking, as defined as a pattern of consumption that places individuals at risk for negative health consequences (Rial et al., 2020; Ryan et al., 2019). From an individual-centered perspective, numerous studies have sought to identify individual vulnerability factors that contribute to the onset and escalation of alcohol use during early adolescence (Cooke et al., 2024; Lees et al., 2021; Swendsen & Le Moal, 2011) from different perspectives.

Neuroscientific theories, such as the maturational imbalance theory (Casey, 2015; Casey et al., 2016), propose that a particular individual vulnerability during adolescence is the imbalance between increased reward sensitivity and reduced inhibitory control, guided by the unequal development of underlying brain areas (Fuhrmann et al., 2015; Shulman et al., 2016). This imbalance can lead to heightened responsiveness to rewards and impulsive behaviors (Harden & Tucker-Drob, 2011). In parallel, maturational and neuroscientific models underscore the importance of immature inhibitory or self-regulatory systems (Casey, 2015; Casey et al., 2016; Shulman et al., 2016). Poorer performance on executive function tasks (including inhibition and decision-making) has been found to be associated with substance use in adolescents (Duell et al., 2016; Lopez-Caneda et al., 2014). Specially, baseline lower inhibitory control at 12-to-14-year-olds predicted the increase in alcohol use and HED by 17-to-18-year-olds (Squeglia et al., 2014). Consequently, the imbalance between lower inhibition

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4 and high reward sensitivity may contribute to adolescent risk-taking behaviors (Noël, 2014;
5 Peeters et al., 2014). To further elucidate the behavioral consequences of this imbalance, Gray's
6 biopsychological theory (Gray, 1981) proposes two brain-based systems that guide behavior:
7 the Behavioral Activation System (BAS) and the Behavioral Inhibition System (BIS). The BAS
8 motivates approach behaviors towards rewards and active avoidance of potential punishment
9 (Barranco-Jiménez et al., 2009). The second system, the BIS, is associated with behavioral
10 inhibition in response to punishment or the absence of expected reward. It drives passive
11 avoidance (inactivity or implicit acceptance to reduce punishment) and extinction (cessation
12 of punished behavior) (Barranco-Jiménez et al., 2009). While a high BAS has been consistently
13 linked to an earlier age of first alcohol use and higher alcohol use (Willem et al., 2010), a low
14 BIS has been associated with a later onset of alcohol use and less hazardous alcohol use and
15 HED in adolescence (Lees et al., 2020a; Lopez-Caneda et al., 2014; Willem et al., 2010). This
16 relationship may be influenced by the strong connection of high BAS and low BIS with
17 impulsivity, a well-established trait linked to alcohol use (Smillie et al., 2006). Specifically,
18 the particular strong connection of high BAS and dysfunctional impulsivity led to the initial
19 thinking that impulsivity was just a manifestation of high BAS (Smillie et al., 2006). Some
20 studies since that moment have shown that the combination of impulsivity and reward
21 sensitivity related to greater alcohol use (Hamilton et al., 2012; Martín-Pérez et al., 2022).

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42 Despite the established impact of impulsivity and sensitivity to reward on alcohol
43 initiation and increased consumption, research consistently highlights the significant role of
44 specific Big Five personality traits in explaining early adolescent alcohol use (Cooke et al.,
45 2017; Heinrich et al., 2016; Parchetka et al., 2016, among others). Furthermore, the Big Five
46 dimensions are closely and differently linked to Gray's BIS and BAS dimensions and
47 impulsivity traits. Specifically, sensitivity to reward correlated positively with extraversion
48 (Smillie et al., 2006; Smits & Boeck, 2006), and dysfunctional impulsivity, showing a factor
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4 including individuals with high BAS. In contrast, sensitivity to punishment showed a
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6 correlation with Neuroticism, forming another distinct factor that captures the conceptual
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8 dimension of BIS. Besides that, Lange et al. (2017) showed various associations between motor
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10 and attentional impulsivity (as measured by the Barrat Impulsiveness Scale) and extraversion
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12 and neuroticism, respectively. Therefore, the convergence of high impulsivity, heightened
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14 reward sensitivity, and high extraversion and neuroticism seem to constitute a profile that
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16 predisposes individuals to risky behaviors, like HED.
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19 Nevertheless, it remains unclear whether these individual factors contribute to
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21 increased alcohol use or are simply consequences of it (Dick et al., 2010). Fortunately,
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23 longitudinal studies have provided valuable insights into this issue. For instance, Fernie et al.
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25 (2013) found that three computer-based impulsivity measures—disinhibition, risk-taking, and
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27 delay discounting, the tendency to undervalue future rewards compared to immediate ones—
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29 were associated with increased alcohol use six months later in early adolescents who drank
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31 minimally or not at all. Intriguingly, they did not observe a reciprocal relationship between
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33 alcohol use and impulsivity. In contrast, Fernández-Artamendi et al. (2018) found no evidence
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35 that impulsivity assessed through behavioral tasks could predict alcohol-related problems, but
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37 impulsivity traits (as a unitary self-reported construct) did predict future alcohol-related
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39 problems and intoxication episodes. Similarly, research has indicated that various impulsivity
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41 domains influence alcohol use (Stautz & Cooper, 2013) and that heightened reward sensitivity
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43 may be a key driver of alcohol initiation (Heinrich et al., 2016), aligning with maturational and
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45 personality models. This suggests that other individual differences, such as neuropsychological
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47 and cognitive measures, could potentially predict adolescent alcohol use beyond personality
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49 factors (Whelan et al., 2014).
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53 Currently, there is a need for research evaluating alcohol use in early adolescence
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55 (Martínez-Loredo et al., 2015), particularly longitudinal studies examining vulnerability factors
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4 associated with early adolescence alcohol use (Fernández-Artamendi et al., 2018; Lees et al.,
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6 2020b; Squeglia et al., 2014). Therefore, the current study aimed to assess cognitive, personality
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8 and, neuropsychological factors associated with alcohol use in 12- to 14-year-old adolescents
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10 at an initial assessment (T1) and reassessed two years later (T2). Furthermore, the specific
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12 objectives were: (i) to identify factors associated with alcohol initiation at T2 among
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14 adolescents who had not consumed alcohol at T1, and (ii) to explore factors related to increased
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16 alcohol use at T2 among those who had already consumed alcohol lightly at T1. We
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18 hypothesized that mainly lower inhibition, higher reward sensitivity, low conscientiousness and
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20 high extraversion would predict initiation to alcohol use (Cooke et al., 2017; Heinrich et al.,
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22 2016; Kim-Spoon et al., 2017; López-Caneda et al., 2014; Parchetka et al., 2016; Squeglia et
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24 al., 2015; Willem et al., 2010), while low inhibition, low conscientiousness and self-reported
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26 impulsivity would predict higher involvement in alcohol use (Clark et al., 2012; Fernández-
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28 Artamendi et al., 2018; Wardell et al., 2016).
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31 32 33 **Methods**

34 *Participants*

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38 Initially, 1,267 students were selected after meeting the inclusion and exclusion criteria.
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40 All participants completed the first assessment and 792 of these agreed to participate in
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42 the second assessment, resulting in a 37.49% attrition rate. Only participants completing
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44 the two assessment sessions were included in this study. The final sample of 792
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46 adolescents (46.6% female) had a mean age of 13.80 years (SD = .569; age range 12.08-
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48 14.99). Inclusion criteria were: (i) aged 12-14 at the first evaluation, and (ii) attending
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50 secondary school in the Community of Madrid (Spain). Exclusion criteria included: a
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52 personal history of neurological or systemic diseases, personal or family alcohol use
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54 disorder (DSM-5 criteria), major mental disorder, a history of substance use disorder in
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4 first-degree relatives, or a HED alcohol use pattern in the first assessment (T1).

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6 Throughout the study, participants were classified according to their alcohol use pattern

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8 based on WHO guidelines (WHO, 2000) into three groups. First, a no-drinking group

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10 (ND) was composed of adolescents who did not consume alcohol. Second, a light

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12 drinking group (LD) included adolescents who consumed alcohol occasionally.

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14 Adolescents in this group consumed part or all of an alcoholic beverage occasionally, less

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16 than once a month, and never engaged in heavy drinking episodes. Heavy drinking

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18 episodes are characterized by consuming more than six (for boys) or four (for girls)

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20 standard drinks (10 grams of alcohol each) within a three- to four-hour period. Finally, a

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22 heavy episodic drinking group (HED) consisted of adolescents who had engaged in at

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24 least one heavy drinking episode in the past three months. These groups were categorized

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26 based on participants' self-reported alcohol use quantity and frequency, as well as their

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28 scores on the third question of the Alcohol Use Disorders Identification Test (AUDIT).

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30 In the first assessment session (T1), participants were classified into two groups based on

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32 alcohol use pattern (WHO guidelines; WHO, 2000): (1) ND-T1: n=441 (45.3% female;

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34 mean age: 13.71, SD=.544); (2) LD-T1: n=351 (47.6% female; mean age: 13.91,

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36 SD=.580). Two years later, in the second assessment session (T2), participants were again

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38 classified into three groups: (1) ND-T2, (2) LD-T2, and (3) HED-T2.

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40 Regarding missing data, most cases were attributable to the withdrawal of entire schools

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42 from the study. This was primarily due to stricter COVID-19 restrictions and the

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44 challenges associated with accommodating research staff for evaluations. The remaining

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46 missing cases are likely to be missing at random. Given that the study was conducted

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48 during regular school hours in classrooms, and considering the mandatory school

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50 attendance laws in Spain, it is plausible that these missing cases resulted from factors

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4 such as illness, school transfers, or family relocations, as suggested by Fernández-
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6 Artamendi et al. (2018).
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8 *Procedure*

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10 Schools and students participated in the study voluntarily after receiving complete
11 information about the project. As participants were minors, parents signed a consent form
12 prior to the students completing the questionnaires. Individuals were assessed in their
13 own classrooms using a printed battery of questionnaires. The participants completed the
14 whole battery in a maximum of 1 hour. The first questionnaire included questions about
15 their social and personal characteristics. Alcohol use was determined through questions
16 about alcohol use, including age of initiation, regular use, quantity, and frequency, as well
17 as with the third question of the AUDIT test. Subsequently, participants were categorized
18 into groups based on their responses to the aforementioned questions, adhering to the
19 guidelines for alcohol use categorization provided by the World Health Organization
20 (WHO, 2020). The participants also completed the Spanish version of the NEO-FFI, the
21 Dysexecutive Questionnaire (DEX-sp), the Barratt Impulsiveness Scale (BIS-11) and the
22 Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ-20) to assess
23 personality domains, dysexecutive behaviors, impulsivity traits and sensitivity to
24 punishment and sensitivity to reward. All the students sat in individual desks and
25 completed the evaluation during class schedule, under the supervision of, at least, two
26 trained psychologists and researchers. The follow-up sessions were performed under the
27 same conditions with a difference between 24 and 27 months from the first assessment.
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29 The delay of the second evaluation in some schools was due to the strict schedule of
30 external visits to schools suffered by the COVID-19 situation. Those who completed
31 correctly all the questionnaires were compensated with gift cards valued at 10 euros per
32 evaluation. This research is part of a larger prospective longitudinal study aimed at
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4 identifying early neural and psychological predictors of hazardous drinking (specifically,
5 HED) in early adolescents. Thus, further sessions of assessment were carried out. The
6 project commenced after approval by the Ethical Committee of (blind to review).
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10 11 12 13 14 15 *Measures*

16 **Personality domains.** One of the most widely accepted taxonomic proposals for
17 personality structure is the Five-Factor Model (McCrae & Costa, 1995; 2003). Several scales
18 exist to assess these five factors, but this study employed the NEO-FFI, a shorter version of the
19 NEO-PI-R. The NEO-FFI measures five personality dimensions—Neuroticism, Extraversion,
20 Openness, Agreeableness, and Conscientiousness—through 60 items (12 per dimension). The
21 Spanish version of the NEO-FFI (Avia, 2000) was used in this research. The internal
22 consistency, McDonald's ω , for the scale was .78 (IC95% 76-88) and for alpha Cronbach's was
23 .84 (IC 95% .82-.87).
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33 **Dysexecutive behaviors.** The Dysexecutive Functioning Questionnaire for
34 Children (DEX-C) is part of the Behavioral Assessment of the Dysexecutive Syndrome
35 in Children (BADS-C) test battery (Wilson et al., 1996). Additionally, the Spanish version
36 of the Dysexecutive Questionnaire (DEX-sp) (Pedrero-Pérez et al., 2009) was used to
37 assess changes in the observable everyday manifestations of neurocognitive dysfunction.
38 This self-reported questionnaire comprises a 20-item checklist rated on a 5-point Likert
39 scale ranging from "rarely" (0) to "very often" (5). The questionnaire measures four
40 factors: inhibition, intentionality, executive memory, and positive and negative affect. For
41 our analysis, we focused solely on the inhibition measure to obtain a variable related to
42 inhibitory control functioning in adolescents (Casey, 2015; Casey et al., 2016; Shulman
43 et al., 2016). As this is an inverse variable, higher scores indicate lower inhibition. The
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4 internal consistency, McDonald's ω , for the scale was .89 (IC 95% .81-.90) and for alpha
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6 Cronbach's was .84 (IC 95% .82-.85).

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8 **Impulsivity traits.** To assess the various domains of impulsivity, we employed a
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10 Spanish version of the Barratt Impulsiveness Scale (BIS-11) (Oquendo et al., 2001; Patton
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12 et al., 1995). The BIS-11 is a 30-item self-report questionnaire rated on a 4-point Likert
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14 scale ranging from "rarely/never" (1) to "almost always/always" (4). This instrument
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16 measures three primary impulsivity factors and their corresponding six subfactors:
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18 cognitive impulsivity (attention and cognitive instability), motor impulsivity (motor and
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20 perseverance), and non-planning impulsivity (self-control and cognitive complexity). The
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22 internal consistency, McDonald's ω , for the scale was .70 (IC 95% .66-.73) and for alpha
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24 Cronbach's was .70 (IC 95% .63-.73).

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27 **Sensitivity to Punishment and Sensitivity to Reward.** The Sensitivity to
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29 Punishment and Sensitivity to Reward Questionnaire (SPSRQ-20) (Torrubia et al., 2001)
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31 is a self-report questionnaire grounded in Gray's (1981) biopsychological theory of
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33 personality. It consists of 20 dichotomous (yes/no) items and measures two constructs:
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35 Sensitivity to Punishment, linked to the Behavioral Inhibition System (BIS), and
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37 Sensitivity to Reward, associated with the Behavioral Activation System (BAS). The
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39 internal consistency, McDonald's ω , for the scale was .71 (IC 95% .66-.75) and for alpha
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41 Cronbach's was .72 (IC 95% .67-.76).

42 43 44 45 46 ***Statistical analysis***

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48 Independent samples t-tests and chi-square tests were used to analyze between-group
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50 differences in T1 (ND-T1 and LD-T1) for sociodemographic and psychological
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52 measures. A chi-square test was also used to assess the frequency changes between the
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alcohol use patterns in the first (T1; 12-14 years) and the second measurement (T2; aged 14-16).

Finally, four binary logistic models, called "logit," were developed to determine whether the dimensions of the evaluated scales in the adolescents of the sample could predict the initiation or increase in alcohol use in the following two years. These analyses were performed within the statistical software "R project" (R Development Core Team, 2008), using the function "glm". The dependent variable is specified as a parameter binomial (0-1). A binary logistic regression is applicable when the dependent variables are dichotomous. Their values may be either 1 or 0, representing the two decision possibilities. Thus, the probability of a successful result is denoted as $P(Y = 1)$, while that of an unsuccessful result is $1 - P(Y = 1)$. Assuming that X_1, X_2, \dots, X_m are m influential dimensions (latent variables for this study) of result Y and the logistic regression formula is used to calculate the ratio of successful and unsuccessful probabilities, its natural logarithm is $\text{Logit } P = \alpha + \beta_1 X_1 + \beta_2 X_2 + \dots + \beta_m X_m$ (where m is the number of independent dimension/latent variables in this case).

We performed four logistic regression models to assess the following: the predictive variables in (1) the change from ND-T1 to drinker (LD-T2 and HED-T2 together); (2) the change from ND-T1 to only LD-T2; (3) the change from ND-T1 to only HED-T2; and (4) the change from LD-T1 to HED-T2. In the four models, sex and age were entered in the regression models as control variables. Besides, only those participants who performed both assessments (T1 and T2) were included in the analyses.

Results

Between-groups differences in sociodemographic and psychological variables

Table 1 shows differences in T1 between ND and LD in age, all NEO-FFI measures except agreeableness, sensitivity to reward, motor impulsiveness, and

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4 disinhibition. Notably, those who consumed alcohol lightly at T1 (LD-T1) exhibited
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6 higher sensitivity to reward, motor impulsiveness, disinhibition, neuroticism and
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8 extraversion sub-traits on the NEO-FFI, (ND-T1) compared to those non-drinkers in T1
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10 (ND-T1). Conversely, the ND-T1 group demonstrated higher conscientiousness and
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12 openness (see Table 1) in comparison with LD-T1.
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17 ***Differences in alcohol use patterns between T1 and T2***

18 Table 2 shows the results of the Chi-Square test for the changes between the
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20 frequencies of different alcohol use patterns in T1 (12-14 years) and T2 (14-16 years).
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22 The results revealed a significant association between the first and the second assessment
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24 ($X^2= 26.06, C=.20 p < 0.05$). At T1, there were 351 (44.3%) students in the ND-T1 group
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26 and 441 (55.7 %) in the LD-T1 group. After T2, 407 (51.4 %) changed or remained in the
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28 LD-T2 group, and 196 (24.7%) changed to the HED-T2 group. Specifically, 233 (29.4%)
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30 did not drink in T1 (ND-T1) and drank lightly in T2 (LD-T2), while 80 (10.1 %)
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32 adolescents who did not drink in T1 (ND-T1) showed a HED pattern in T2 (HED-T2).
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34 Moreover, 116 (14.6%) adolescents showed a LD pattern in T1 (LD-T1) and changed to
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36 a HED pattern in T2 (HED-T2) (Table 2).
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41 ***The change from no drinking (T1) to drinker in T2 (both light and heavy episodic drinking)***

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43 The first stepwise logistic regression analyzed participants who did not drink in
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45 the first time point (ND-T1; N=441) but began drinking in any pattern (light [LD-T2] or
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47 HED [HED-T2]) in T2. For the first step, DEX Inhibition ($W = 66.99, p < .001$) was
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49 positive and significant; while the second step showed a positive and significant
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51 relationship with DEX Inhibition ($W = 63.53, p < .001$) and BIS Motor Impulsivity ($W =$
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53 $7.54, p < .006$); for the third step, there was a positive and significant relationship with
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4 DEX Inhibition ($W = 63.20, p < .001$), BIS Motor Impulsivity ($W = 5.45, p = .020$) and
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6 SPSRQ Sensitivity to Reward ($W = 4.86, p = .027$). Finally, for the fourth step, DEX
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8 Inhibition ($W = 57.84, p < .001$), BIS Motor Impulsivity ($W = 4.02, p = .045$), SPSRQ
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10 Sensitivity to Reward ($W = 4.81, p = .027$) and NEO Openness ($W = 4.87, p = .028$)
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12 emerged as predictors for the initiation of alcohol use. These variables positively impact
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14 the probability of changing from a non-drinking to HED or LD status. The Nagelkerke
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16 R^2 index, a measure of goodness of fit in logistic regression analyses, was between .838-
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18 .862, depending on the step (further details and odds ratios for each variable in each step
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20 can be found in Table S1 in Supplementary Material). Moreover, the sensitivity index
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22 was .97 and specificity index was .94.

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24 Then, we subdivided the results of those who did not drink in T1 (ND-T1) according to
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26 the pattern of alcohol use followed to perform the second and third stepwise logistic
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28 regressions.
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31 32 33 ***The change from no drinking (T1) to only light drinking (T2)***

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35 Firstly, in the second regression, we display the results of those who did not drink
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37 at T1 (ND-T1) but showed a LD pattern in T2 (LD-T2). For the first step, DEX Inhibition
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39 ($W = 44.79, p < .001$) was positive and significantly different from zero, while the second
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41 step showed a positive and significant relationship with DEX Inhibition ($W = 46.90, p <$
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43 $.001$) and NEO Openness ($W = 5.21, p = .022$). The Nagelkerke R^2 index was between
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45 .901-.909, depending on the step (further details and odds ratios for each variable in each
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47 step can be found in Table S2 in Supplementary Material). Moreover, the sensitivity index
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49 was .98 and specificity index was .98.
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52 53 54 ***The change from no drinking (T1) to only heavy episodic drinking (T2)***

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4 Secondly, in the third regression, the results from those who did not drink in T1
5 (ND-T1) but showed a HED pattern in T2 are presented (HED-T2). For the first step,
6 DEX Inhibition ($W = 51.79, p < .001$) was positive and significantly different from zero,
7 while the second step revealed a positive a significant relationship with DEX Inhibition
8 ($W = 21.64, p < .001$) and SPSRQ Sensitivity to Reward ($W = 11.15, p < .001$). For the
9 third step, there were positive and significant predictive models regarding DEX Inhibition
10 ($W=18.69; p < 0.001$), SPSRQ Sensitivity to reward ($W=10.29; p < 0.001$), and NEO-FII
11 Neuroticism ($W=3.94; p=0.48$). The Nagelkerke R^2 index was between .873-.938,
12 depending on the step (further details and odds ratios for each variable in each step can
13 be found in Table S3 in Supplementary Material). Moreover, the sensitivity index was
14 .95 and specificity index was .98.
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30 ***The change from light drinking (T1) to heavy episodic drinking (T2)***

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32 Finally, the results of the stepwise logistic regression for those who had a LD pattern of
33 alcohol use in T1 (LD-T1) and increased their alcohol use to a HED pattern in T2 (HED-
34 T2) are presented. For the first step, DEX Inhibition ($W = 73.36, p < .001$) was positive
35 and significantly different from zero; the second step revealed a positive and significant
36 relationship for DEX Inhibition ($W = 67.19, p < .001$) and SPSRQ Sensitivity to Reward
37 ($W = 16.27, p < .001$); in the third step, there were positive and significant relationships
38 with DEX Inhibition ($W = 67.02, p < .001$), SPSRQ Sensitivity to Reward ($W = 14.298,$
39 $p < .001$) and a positive relationship with sex ($W = 4.434, p < .035$); and finally, the last
40 step revealed significant relationships with: DEX Inhibition ($W = 60.52, p < .001$),
41 SPSRQ Sensitivity to Reward ($W = 13.76, p < .001$), BIS11 Cognitive Impulsivity ($W =$
42 $4.56, p = .033$) and sex ($W = 4.760, p = .029$). The Nagelkerke R^2 index was between
43 .674-.736, depending on the step (further details and odds ratios for each variable in each
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4 step can be found in Table S4 in Supplementary Material). Moreover, the sensitivity index
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6 was .84 and specificity index was .90.
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10 **Discussion**

11 This study aimed to identify individual psychological vulnerability factors linked to
12 early adolescent alcohol initiation and increased consumption. We hypothesized that mainly
13 lower inhibition, higher reward sensitivity, low conscientiousness and high extraversion would
14 predict alcohol use initiation at T2 (Cooke et al., 2017; Heinrich et al., 2016; Kim-Spoon et al.,
15 2017; López-Caneda et al., 2013; Parchetka et al., 2016; Squeglia et al., 2015; Willem et al.,
16 2010), while low conscientiousness and impulsivity would predict riskier involvement in
17 alcohol use at T2 (Clark et al., 2012; Fernández-Artamendi et al., 2017; Wardell et al., 2016).
18 Descriptive and comparative data revealed that LD at T1 exhibited higher sensitivity to reward,
19 motor impulsivity, disinhibition, and extraversion and neuroticism (NEO-FFI), while lower
20 conscientiousness and openness, compared to ND. These findings are consistent with previous
21 research suggesting increased impulsivity and impaired inhibition in early-onset drinkers
22 (Lejuez et al., 2010). Furthermore, the observed pattern of higher extraversion and lower
23 conscientiousness partially aligns with previous studies on early drinkers (Anderson et al.,
24 2007; Jones et al., 2022; Malouff et al., 2007; Parchetka et al., 2016).
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41 Our first objective was to identify individual factors at baseline (T1) associated with
42 the onset of alcohol use at follow-up (T2) among non-drinkers. Disinhibition emerged as the
43 strongest predictor of alcohol initiation. It consistently showed the highest odd ratio across all
44 model stages, indicating a strong effect. This finding aligns with previous research
45 demonstrating a strong link between low inhibition and alcohol use (Latzman et al., 2011;
46 Lopez-Caneda et al., 2014; Squeglia et al., 2014). Squeglia et al. (2014) further emphasized
47 that poorer cognitive inhibition-interference in early adolescents with no or light alcohol use
48 predicted higher HED at 17-18 years old. While disinhibition remained the most important
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4 factor throughout the model, our findings also revealed that motor impulsivity, sensitivity to
5 reward, and openness had positive, albeit smaller, statistically significant effects. This aligns
6 with previous research suggesting that individuals with heightened sensitivity to reward are
7 more prone to initiate alcohol use at early ages (Willem et al., 2010). Moreover, motor
8 impulsivity has been related in many studies to a large spectrum of alcohol use patterns, ranging
9 from social drinkers to individuals with AUD or HED (see the review of Herman & Duka,
10 (2020). Additionally, while the relationship is not entirely consistent across studies, openness
11 has been related to increased alcohol use across various age groups, (Jones et al., 2022).
12 Although the specific role of motor impulsivity and openness in alcohol initiation has not been
13 explicitly explored in this previous research, our findings suggest that these traits may
14 contribute to the onset of alcohol use.
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27 Among ND at T1, those who initiated occasional/light drinking (LD) at T2 were
28 characterized by higher levels of disinhibition and openness, with disinhibition showing again
29 the highest odds ratio. Specifically, disinhibition increase the probability of transitioning from
30 ND to LD by 428%. While disinhibition is a well-established predictor of early adolescent
31 alcohol use (Lopez-Caneda et al., 2014; Squeglia et al., 2014, among others), the role of
32 openness in alcohol initiation is less clear. Although some studies have found no association
33 between openness and alcohol use (Anderson et al., 2007; Parchetka et al., 2016), others have
34 found positive associations in children (Malouff et al., 2007). Given that openness
35 encompasses curiosity and a propensity for atypical experiences (McCrae, 1995), and is
36 strongly associated with sensation seeking (Aluja et al., 2003), it is unsurprising that this trait
37 is implicated in alcohol initiation. A clarifying study is that of Malouff et al.'s (2007). This
38 meta-analysis revealed significant differences in the association between openness and alcohol
39 involvement between adult and child studies, with children exhibiting a significant
40 relationship. The authors propose a potential explanation: openness may be linked to initial
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4 alcohol experimentation in childhood but does not necessarily lead to more hazardous alcohol
5 use (Malouff et al., 2007). This interpretation aligns well with our findings in nondrinkers who
6 evolve to a light drinking pattern. Interestingly, participants in the ND group at T1 who
7 transitioned to HED at T2 did not exhibit openness as a predictive factor. Instead, they
8 displayed heightened reward sensitivity and reduced neuroticism. However, similar to the
9 subgroup transitioning to LD, this subgroup also demonstrated lower inhibition, underscoring
10 the role of disinhibition in alcohol initiation.
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19 Both disinhibition and reward sensitivity are well-studied correlates of hazardous
20 drinking (Kim-Spoon et al., 2016; Rossiter et al., 2012). In our model, these two variables
21 showed the highest odds ratios (2.83 and 1.72, respectively), indicating that they significantly
22 increase the probability of transitioning from no drinking to HED by 183% and 72%,
23 respectively. Furthermore, we found a significant, albeit smaller, effect of low neuroticism over
24 the transition from ND to HED. This finding appears to contradict previous research linking
25 hazardous alcohol use with higher neuroticism (Fischer et al., 2007; Malouff et al., 2007;
26 Papachristou et al., 2016). However, higher neuroticism has also been positively correlated
27 with emotional disorders (Chinneck et al., 2018) and coping-anxiety and coping-depression
28 drinking motives (Mezquita et al., 2010). It is noteworthy that among the assessed adolescents
29 who were abstinent in T1, we found no evidence of high levels of neuroticism or severe mental
30 health issues. Therefore, a potential explanation for the discrepancy with previous studies is
31 that a potential drinking pattern driven by coping motives may not be applicable to our sample
32 of adolescents without addictive drinking problems. However, future research on individual
33 differences in alcohol use should consider drinking motives.
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50 Alternatively, our findings regarding factors predictive of the transition of ND to BD
51 may be consistent with existing research linking these factors to risk-taking behavior. Smillie
52 et al. (2006) found that impulsivity is positively correlated with high sensitivity to reward and
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4 extraversion, and negatively related to high sensitivity to punishment and high neuroticism
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6 (Smillie et al., 2006). Our results partially align with this. We found lower neuroticism and
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8 higher sensitivity to reward, coupled with higher disinhibition, showing a similar profile to this
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10 in Smillie et al. (2006). These shared characteristics may be particularly relevant for those who
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12 progress to riskier patterns of alcohol use, such as HED. Finally, adolescents who escalated
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14 from light (T1) to HED (T2) exhibited low inhibition, high reward sensitivity, high cognitive
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16 impulsivity, and sex as significant predictors, with males being more likely to transition to
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18 HED. Our findings are consistent with previous research linking reward sensitivity,
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20 disinhibition, and impulsivity to hazardous drinking (Rossiter et al., 2012) and escalated
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22 alcohol use (Hamilton et al., 2019) among adolescents. In addition to the psychological
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24 measures, only in this model sex appeared as a predictor of the transition. Indeed, sex showed
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26 the highest odds ratio, highly increasing the probability of transitioning from LD to HED. This
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28 aligns with previous studies demonstrating that male sex, in the presence of high impulsivity,
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30 contribute to alcohol use problems (Gowin et al., 2017; Nagoshi et al., 1991; Stoltenberg et al.,
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32 2008). Interestingly, sex was not a predictor in models examining the transition from ND to
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34 any drinking pattern. This suggests that sex may be more relevant to the progression from
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36 initiated drinkers to more deleterious patterns of alcohol use (Clark et al., 2012) than to the
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38 initiation of alcohol use. Further longitudinal studies should delve deeper into these
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40 relationships.
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44 This study has several limitations, and thus, our findings should be interpreted and
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46 generalized with caution. For example, the Nagelkerke R^2 values in some of the resulting
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48 regression models did not increase significantly between the different steps. However, we
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50 believe it is important to present the main variables related to the initiation or increase in
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52 alcohol use to better understand the differences between subgroups. Another potential
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54 limitation of our study lies in the categorization of alcohol consumption. This approach may
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4 have reduced statistical power compared to a continuous measure. Future research could
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6 consider employing quantitative measures, such as the composite total score of the AUDIT, to
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8 assess alcohol use more precisely. Instead, we followed the recommendations of the WHO
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10 (2000) and other similar studies (Correas et al., 2019; Crego et al., 2012) for the division of
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12 alcohol use groups. The percentage of dropouts during the second evaluation must also be
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14 considered when interpreting the results. While this loss was expected to be random, future
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16 studies should investigate differences between participants who completed the longitudinal
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18 evaluation and those who were lost to follow-up. Identifying potential characteristics of
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20 dropouts can help to contextualize the results more accurately. A further limitation is that this
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22 study only used cognitive, personality, and demographic measures. Therefore, future research
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24 should include both functional and structural neuroimaging indices to explore the brain areas
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26 involved in the increase and initiation of alcohol use, expanding our knowledge of the neural
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28 basis of these processes. Moreover, studies encompassing not only individual factors, but also
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30 social and contextual ones are necessary to provide a comprehensive understanding of alcohol
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32 use issues. Furthermore, a two-year gap between our assessments may limit our ability to
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34 control confounding variables that could influence changes in alcohol use among the
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36 adolescent sample. Despite these limitations, this research is significant and innovative as it is
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38 one of the few existing studies examining individual vulnerability factors related to the
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40 transition from nondrinkers to alcohol use in early adolescence. It's notable that one group
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42 reported no initial alcohol use, allowing us to observe the onset of consumption and associated
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44 factors in early adolescents before exposure. In addition to the above, the sample size is
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46 considerable given the longitudinal nature of the data. Additionally, we focused on adolescents
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48 who did not drink in the first assessment. This research is one of the few examining longitudinal
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50 data on alcohol measures at such an early age.
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4 In conclusion, our findings indicate that lower inhibition is the primary predictor of
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6 both the initiation and increase of alcohol use in the second evaluation, regardless of
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8 adolescents newly acquired drinking pattern (light or HED). While high openness
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10 characterized the transition to LD, high reward sensitivity and low neuroticism were associated
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12 with the shift to HED. Interestingly, reward sensitivity also predicted the progression from
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14 light to HED, emerging as a factor for developing hazardous drinking among those who did
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16 not or drank lightly at T1. Our results align with dual system models emphasizing the complex
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18 neurobehavioral changes during early adolescence (Lees et al., 2021). These changes may be
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20 linked to an imbalance between socioemotional and cognitive control neural circuits (Casey,
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22 2015; Casey, 2016). Such alterations could be crucial in understanding early individual
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24 vulnerability to risky behaviors like initiating or escalating alcohol use (Shulman et al., 2016).
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26 Our findings support the maturational imbalance theory (Casey et al., 2011) by identifying
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28 lower inhibition and higher impulsivity as predictive factors in early adolescents. The data from
29
30 this study could serve as a foundation for future research on adolescent alcohol use, ultimately
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32 leading to the development of more effective, tailored primary and secondary prevention
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34 programs that address the specific needs of each group before they may develop hazardous
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36 drinking patterns.
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Table 1.

Descriptive information and between-groups differences in the variables measured in T1.

	No drinking (ND) N=441	Light consumption (LC) N=351	Statistic*	<i>p</i>
Age	Mean (SD) 13.71 (.54)	Mean (SD) 13.91 (.58)	4.966	<.001
Sex (females)	210 (47.6%)	159 (45.3%)	3	.136
NEO-FII				
Agreeableness	25.55 (6.44)	25.93 (6.55)	0.808	.419
Neuroticism	17.67 (7.45)	19.87 (7.63)	4.090	<.001
Extraversion	32.00 (7.05)	33.06 (6.35)	2.201	.028
Conscientiousness	30.98 (5.39)	29.47 (5.71)	-3.807	<.001
Openness	31.13 (7.94)	28.41 (7.08)	-5.012	<.001
SPSRQ				
Sensitivity to punishment	19.43 (5.57)	19.66 (5.56)	0.569	.569
Sensitivity to reward	19.11 (5.12)	21.19 (5.58)	5.467	<.001
BIS-11				
Cognitive	15.06 (4.44)	15.63 (4.44)	1.769	.077
Motor	14.11 (6.87)	15.48 (6.90)	2.786	.005
Non-planning	18.93 (6.85)	19.19 (6.77)	0.530	.596
DEX				
Inhibition*	6.76 (4.94)	8.90 (5.59)	5.708	<.001

Note: All these variables represent self-reported measures. *Statistic: T values were estimated for all variables except for sex, where X^2 are shown. Abbreviations: SD, standard deviation. *Inhibition is an inverse variable, thus, the higher is the score, the lower is the inhibition.

Table 2.

Observed Frequencies and Chi-Square test results between the first and second measurements.

		Second measurement (T2)			
		No Drinking	Light Drinking	HED	Total
First measurement (T1)	No Drinking	128 16.2%	233 29.4%	80 10.1%	351 44.3%
	Light Drinking	61 7.7%	174 22.0%	116 14.6%	441 55.7%
	Total	189 23.9%	407 51.4%	196 24.7%	792 100%

Note: Frequencies and percentages are shown. Abbreviations: HED, heavy episodic drinking

Table S1.

Logit stepwise regression by for the change from non-drinking to light or binge drinking.

	Variables	B	Wald	Odds Ratio	Sig.
First step	Constant (α)	-5.13	59.90		.000
	DEX Inhibition	1.57	66.99	4.86	.000
Second step	Constant (α)	-6.32	53.67		.000
	DEX Inhibition	1.55	63.53	4.71	.000
	BIS Motor Impulsivity	.115	7.54	1.12	.006
Third step	Constant (α)	-8.69	34.60		.000
	DEX Inhibition	1.63	63.20	5.12	.000
	BIS Motor Impulsivity	.098	5.45	1.10	.020
	SPSRQ Sensitivity to reward	.087	4.86	1.09	.027
Fourth step	Constant (α)	-11.09	30.77		.000
	DEX Inhibition	1.73	57.84	5.69	.000
	BIS Motor Impulsivity	.085	4.02	1.09	.045
	SPSRQ Sensitivity to reward	.115	4.81	1.10	.027
	NEO Openness	.093	4.87	1.12	.028

$p < .05$; Nagelkerke R^2 for the first step = .838; Nagelkerke R^2 for the second step = .849; Nagelkerke R^2 for the third step = .856; Nagelkerke R^2 for the fourth step = .862.

Table S2.

Logit stepwise regression for non-drinking to light drinking.

	Variables	B	Wald	Odds Ratio	Sig.
First step	Constant (α)	-8.89	42.64		.000
	DEX Inhibition	2.57	44.79	4.81	.000
Second step	Constant (α)	-12.33	29.78		.000
	DEX Inhibition	2.63	46.90	5.28	.000
	NEO Openness	.125	5.21	1.10	.022

p < .05; Nagelkerke R^2 for the first step = .901; Nagelkerke R^2 for the second step = .909.

Table S3.

Logit stepwise regression for non-drinking to binge drinking.

	<i>Variables</i>	<i>B</i>	<i>Wald</i>	Odds Ratio	<i>Sig.</i>
First step	Constant (α)	-4.08	63.61		.000
	DEX Inhibition	.601	51.79	1.75	.000
Second step	Constant (α)	-16.32	14.47		.000
	DEX Inhibition	.821	21.64	2.27	.000
	SPSRQ Sensitivity to Reward	.534	11.15	1.75	.001
Third step	Constant (α)	-14.92	11.51		.001
	DEX Inhibition	1.02	18.79	2.83	.000
	SPSRQ Sensitivity to Reward	.532	10.29	1.72	.001
	NEO-FII Neuroticism	-.151	3.94	0.86	.048

$p < .05$; Nagelkerke R^2 for the first step = .873; Nagelkerke R^2 for the second step = .928;

Nagelkerke R^2 for the third step = .938.

Table S4.

Logit stepwise regression for light drinking to binge drinking.

	Variables	B	Wald	Odds Ratio	Sig.
First step	Constant (α)	-7.06	75.57		.000
	DEX Inhibition	.621	73.63	1.86	.000
Second step	Constant (α)	-10.63	61.52		.000
	DEX Inhibition	.629	67.19	1.87	.000
	SPSRQ Sensitivity to reward	.155	16.27	1.17	.000
Third step	Constant (α)	-11.23	61.62		.000
	DEX Inhibition	.653	67.02	1.92	.000
	SPSRQ Sensitivity to reward	.146	14.30	1.19	.000
	Sex	.879	4.43	2.41	.035
Fourth step	Constant (α)	-12.85	56.37		.000
	DEX Inhibition	.624	60.52	1.87	.000
	BIS Cognitive Impulsivity	.114	4.56	1.12	.033
	SPSRQ Sensitivity to reward	.146	13.76	1.16	.000
	Sex	.935	4.76	2.55	.029

$p < .05$; Nagelkerke R^2 for the first step = .674; Nagelkerke R^2 for the second step = .716; Nagelkerke R^2 for the third step = .726; Nagelkerke R^2 for the fourth step = .736.