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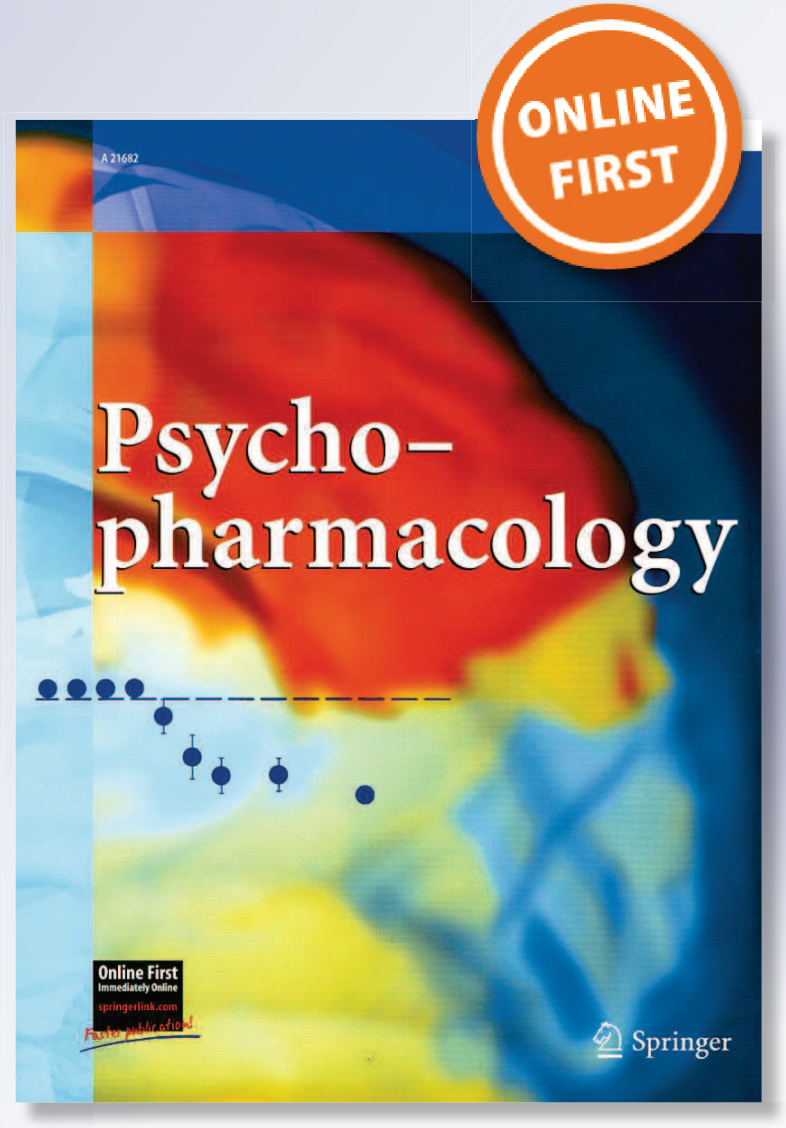
Psychopharmacology

ISSN 0033-3158

Psychopharmacology
DOI 10.1007/s00213-017-4538-4



Springer

Psychopharmacology
DOI 10.1007/s00213-017-4538-4

ORIGINAL INVESTIGATION

Differential impairments across attentional networks in binge drinking

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Received: 26 August 2016 / Accepted: 16 January 2017

Abstract
Rationale The cognitive deficits observed in young binge drinkers have been largely documented during the last decade. Yet, these earlier studies have mainly focused on high-level cognitive abilities (particularly memory and executive functions), and uncertainty thus still abounds regarding the integrity of less complex cognitive processes in binge drinking. This is particularly true for attentional abilities, which play a crucial role in behavior regulation and are impaired in other alcohol-related disorders.
Objectives and methods To specify the attentional deficits associated with binge drinking, two groups of university students (40 binge drinkers and 40 matched controls) performed the Attention Network Task, a theoretically grounded test assessing three independent attentional networks: alerting, orienting, and executive control.
Results Binge drinkers displayed preserved orienting performance but impaired alerting and executive control. Binge drinking is thus not related to a general attentional impairment but rather to specific impairments of the alerting and executive control networks.
Conclusions These results underline that, beyond the already explored high-level deficits, binge drinking is also related to impairments for attentional abilities. In view of the role played by attentional impairments in alcohol dependence, the present data also suggest that rehabilitation programs should be developed to improve attentional abilities at the early stages of alcohol-related disorders.

Keywords Binge drinking · Alcohol dependence · Attentional abilities · Alerting · Orienting · Executive control

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Introduction

Binge drinking—an alcohol consumption pattern characterized by the repeated alternation between excessive alcohol intakes and abstinence periods (Crego et al. 2009)—has become widespread in adolescents and young adults (Archie et al. 2012; Kanny et al. 2013). Over the last decade, the cerebral and cognitive consequences of this drinking pattern have been largely explored. Neuroscience studies have identified anatomical and functional modifications, mostly in limbic and prefrontal regions (for a review, see Hermens et al. 2013), as well as impaired electrophysiological activity (for a review, see Maurage et al. 2013a). In the same vein, behavioral studies have demonstrated various impairments in high-level cognitive abilities, particularly for memory and executive functions. First, binge drinkers show reduced performance in different subcomponents of memory abilities, such as spatial, declarative, episodic, and prospective memory (Hartley et al. 2004; Heffernan et al. 2010; Heffernan and O'Neill 2012; Mota et al. 2013; Parada et al. 2012). Second, executive function impairments have been notably indexed by slower latency for planning (Hartley et al. 2004), reduced updating performance (Parada et al. 2012), disadvantageous choices in decision-making (Goudriaan et al. 2011, 2007), and impaired inhibition (Sanhueza et al. 2011), particularly when confronted with alcohol-related stimuli (Czapla et al. 2015). Research has recently refined these explorations, suggesting that binge drinking is mostly characterized by impaired

Published online: 31 January 2017

adjustment following failures (Bø et al. 2016a) and by disadvantageous choices during decision-making under ambiguity (Bø et al. 2016b), rather than by global executive deficits. As a whole, however, these data have highlighted that binge drinking is a hazardous behavior, and the large-scale cerebral and cognitive consequences related to this alcohol consumption pattern have even led to the "continuum hypothesis" proposal, assuming that binge drinking and alcohol dependence could constitute two successive steps of a same phenomenon, leading to analogous impairments (Enoch 2006; Maurage et al. 2013b; Sanhueza et al. 2011). Yet, as previous works have focused on high-level cognitive functions, uncertainty still abounds vis-à-vis both the less complex processes and the generalizability of the continuum hypothesis towards these processes. This is particularly true for attentional abilities, which have been very little explored in binge drinking. This is unfortunate as this former may act as a core cognitive process and plays a critical role in alcohol-related disorders (Heeren et al. 2015; Maurage et al. 2014).

Attentional functions have been mainly investigated in two patterns of alcohol-related disorders. First, acute alcohol consumption, which has a negative impact on divided (Schulte et al. 2001; Wester et al. 2010), sustained, and selective attention (McKinney et al. 2012). Second, recently detoxified alcohol-dependent patients show specific deficits in selective (Cordovil De Sousa Uva et al. 2010; Evert and Oscar-Berman 2001), divided (Tedstone and Coyle 2004), and sustained (Nixon et al. 2007) attentional processes, as well as in attentional control and shifting capacity (Kopera et al. 2012). Neuroscience studies have also reported delayed and reduced electrophysiological attentional components (P300) in alcohol dependence (Cohen et al. 1995; Ramachandran et al. 1996), as well as white matter lesions (i.e., lower fiber thickness of uncinate fasciculus) and reduced prefrontal functioning during various attentional tasks (e.g., Oscar-Berman and Marinkovic 2003; Pfefferbaum et al. 2001; Schulte et al. 2012; Sullivan et al. 2000). Attentional impairments thus appear to constitute a core deficit in alcohol-related disorders, but data is currently lacking in binge drinking. Binge drinking studies exploring attentional processes have exclusively focused on attentional reactivity to alcohol cues. These studies clearly established that attentional bias, namely the automatic capture of attentional resources by alcohol-related stimuli, constitutes an important factor in the development and maintenance of excessive alcohol consumption (Petit et al. 2012; Hallgren and McCrady 2013; Roberts et al. 2014; Weafer and Fillmore 2015), but attentional processes per se have been nearly totally neglected. To the best of our knowledge, only one behavioral study (Hartley et al. 2004) has reported impaired sustained attention in binge drinking by means of a classical neuropsychological test (i.e., Paced Auditory Serial Addition Test), and these results have been extended by an fMRI study demonstrating that this reduced sustained attention was related to lower brain activations in the spatial working memory network (Squeglia et al. 2011). These studies interestingly suggested that binge drinking might be associated with impaired attentional processing. Nevertheless, as they focused on a unique task exploring a specific attentional subcomponent and simultaneously involving memory abilities, they offer only very partial insights regarding the attentional abilities of binge drinkers.

In view of the scarcity of the literature about attentional abilities in binge drinking, next steps would be to more precisely examine the presence and extent of attentional alterations in this population. The purpose of the present study is thus to explore attentional abilities in young binge drinkers via the Attention Network Task (ANT; Fan et al. 2002). A main advantage of the ANT is that it allows the integrated and simultaneous evaluation of the three attentional networks identified in Posner's model (Petersen and Posner 2012; Posner and Petersen 1990): (1) *alerting*, the ability to achieve and maintain a global high sensitivity or readiness, (2) *orienting*, the selection of incoming information by engaging, disengaging, and shifting the attentional resources from one stimulation to another, and (3) *executive control*, the ability to solve a cognitive conflict using top-down control of attention. This task is now widely validated and has allowed to identify attentional deficits in several psychopathological disorders including anxiety, schizophrenia, or autism (Heeren and McNally 2016; Keehn et al. 2013; Opgen-Rhein et al. 2008). Importantly, the ANT has also allowed to detect distinct patterns of impairments among attentional networks in substance use disorders, such as impaired executive control among cocaine consumers (Woicik et al. 2009) and cannabis abusers (Abdullaev et al. 2010). Moreover, this task has been used in alcohol dependence, showing significant impairment for the executive control of attention with preserved abilities in other networks (i.e., alerting and orienting) in recently detoxified alcohol-dependent inpatients (Maurage et al. 2014). In view of these earlier results in addictive disorders, showing differential results between preserved alerting-orienting networks and impaired executive control network, it can be hypothesized, in link with the continuum hypothesis, that binge drinking might already lead to attentional impairments, especially for the executive control network.

Method

Participants

A first screening phase was conducted among 3744 university students (Université catholique de Louvain, Belgium) to assess socio-demographic variables (i.e., age, gender, education level, and native language), psychological variables, and alcohol consumption [i.e., mean number of alcohol doses (a dose being defined as an alcoholic drink containing 10 g of pure ethanol) per drinking occasion, mean number of drinking occasions per week,

consumption speed in doses per hour, and drunkenness frequency]. Consequently, 1540 students were first selected, fulfilling the following conditions: being fluent French speakers, aged at least 18 years old, with no alcohol dependence and no family history of alcohol dependence, no past or present psychological disorder, no medication, no major medical problem, normal or corrected-to-normal vision, and total absence of past or current drug consumption except alcohol and tobacco. Then, a binge drinking score based on consumption speed, frequency of drunkenness episodes, and percentage of drunkenness episodes compared to the total number of drinking episodes (see Townshend and Duka 2005) was computed, allowing to create two groups: control participants (CP; binge drinking score ≤ 12; $n = 858$) and binge drinkers (BD; binge drinking score ≥ 16; $n = 377$). Participants presenting other consumption patterns ($n = 305$) were removed from the sample, including teetotalers (all participants included in the study demonstrated a binge drinking score higher than 0). Finally, participants from both groups who had agreed to take part in the experiment (i.e., who gave their e-mail address during the screening phase) were contacted. Two groups were created and 80 university students (i.e., 40 CP and 40 BD) performed the experiment. All participants (62.5% women) were between 18 and 29 years old ($M = 20.76$; SD = 2.17). Psychopathological measures were also evaluated before starting the experiment to assess the following variables: (a) depressive symptoms, using the Beck Depression Inventory (BDI-II; Beck et al. 1996, French validation: Beck et al. 1998), (b) anxiety, using the State-Trait Anxiety Inventory (STAI; Spielberger et al. 1983, French validation: Bruchon-Schweitzer and Paulhan 1993), and (c) alcohol-related disorders, using the Alcohol Use Disorder Identification Test (AUDIT; Babor et al. 2001, French validation: Gache et al. 2005). The alcohol consumption characteristics initially ecorded during the screening phase were also re-evaluated before the experiment to explore a potential evolution of alcohol consumption between screening and testing phases. The patterns initially observed were totally confirmed, and the group comparisons performed on all alcohol variables clearly supported the distinction between groups regarding alcohol consumption and binge drinking scores (see Table 1, presenting alcohol consumption characteristics for the two groups at testing time). Importantly, in each group, no significant differences were observed on alcohol consumption characteristics (all ts < 1.58, all ps > 0.11) between the participants initially included in each group (858 CP, 377 BD) and those who finally took part in the experiment (40 CP, 40 BD), thus suggesting an absence of selection bias in our final sample compared to the general population. All participants had self-reported no alcohol consumption for at least 3 days before the experiment.

Stimuli and task description

The ANT was administered to determine the efficiency of three independent attentional networks: alerting, orienting, and executive control (Fan et al. 2002). Participants had to determine as quickly and accurately as possible the direction (left or right) of a central arrow (the target) located in the middle of a horizontal line presented either at the top or bottom of the screen (Fig. 1). They responded by pressing the corresponding button (left or right) on the keyboard. Each target was preceded by either no cue, a central cue (an asterisk replacing the fixation cross), a double cue (two asterisks, one appearing above and one below the fixation cross), or a spatial cue (an asterisk appearing above or below the fixation cross and indicating the location of the upcoming target) (Fig. 1a). Moreover, flankers appeared horizontally on each side of the target. There were three possible flanker types: two arrows pointing in the same direction as the target (congruent condition), two arrows pointing in the opposite direction than the target (incongruent condition), or two dashes (neutral condition) (Fig. 1b). Each trial had the following structure: (1) a central fixation cross (random duration between 400 and 1600 ms), (2) a cue (100 ms), (3) a central fixation cross (400 ms), (4) a target and its flankers, appearing above or below the fixation cross (the target remained on the screen until the participant answered or for 1700 ms if no response occurred); and (5) a central fixation cross (lasting for 3500 ms minus the sum of the first fixation period's duration and the reaction time) (Fig. 1c). Reaction time (RT; in ms) and accuracy score (AS; percentage of correct answers) were recorded for each trial.

The ANT task comprised 288 trials, divided in three blocks of 96 trials (with a short break between blocks). There were 48 possible trials, based on the combination of four cues (no cue, central cue, double cue, spatial cue), three flankers (congruent, incongruent, neutral), two directions of the target (left, right), and two localizations (upper or lower part of the screen). Trials were presented in a random order and each possible trial was showed twice within a block. The task was programmed and presented via E-Prime 2 Professional® (Psychology Software Tools, Pittsburgh, PA, USA).

Procedure

This study comprised three sections. First, participants were provided with full details regarding the aims of the study and the procedure to be followed. Then, participants were administered the ANT after completing a preliminary practice session (24 randomly selected trials). The distance between the participant's eyes and the screen was 50 cm, and the target stimuli subtended a visual angle of about 4° in the horizontal field. Finally, participants were debriefed at the end of the experiment and received

Table 1 Demographic and psychological measures for binge drinkers (BD) and control participants (CP)

Measure	BD (n = 40)	CP (n = 40)
Demographic measures		
Age [mean (SD)]	20.60 (1.65)	20.93 (2.62)
Gender ratio (female/male)	19/21	11/29
Psychological measures [mean (SD)]		
Beck depression inventory	3.75 (2.50)	3.33 (2.58)
STAI state anxiety inventory	30.58 (7.56)	31.75 (7.40)
STAI trait anxiety inventory	36.55 (6.78)	35.95 (6.66)
Number of participants with nicotine dependence	1	3
Alcohol consumption measures [mean (SD)]		
Alcohol use disorder identification test*	15.97 (5.26)	6.45 (5.50)
Total alcohol units per week**	20.04 (11.79)	6.96 (8.40)
Number of occasions per week**	2.99 (1.11)	1.24 (1.13)
Number of alcohol units per occasion**	6.99 (2.84)	3.34 (3.15)
Consumption speed (units per hour)**	3.89 (1.11)	1.29 (0.91)
Number of drunkenness episodes (last 6 months)	11.23 (14.10)	0.24 (0.55)
Percentage of drunkenness episodes	14.34 (19.03)	0.41 (1.05)

*p < .01; **p < .001

compensation (10€). Each session was administrated individually in a dimly lit and quiet room. All participants provided their written informed consent. All the procedures contributing to this work were approved by the local ethics committee and complied with the Helsinki Declaration of 1975, as revised in 2008.

Preparation of the data

We excluded data from trials with incorrect responses (2.72% of trials), RTs lower than 200 ms or greater than 3000 ms (0.09% of trials), and RTs exceeding 2 SD below or above each participant's mean for each experimental condition (5%

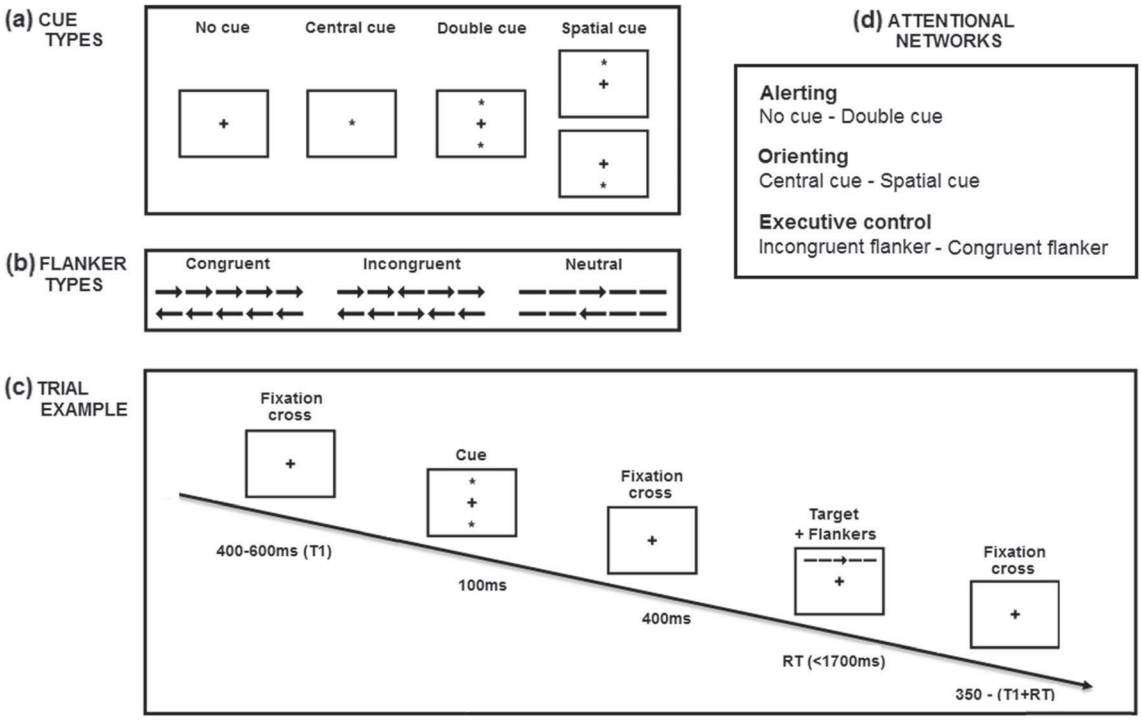

Fig. 1 Description of the Attention Network Task, presenting **a** the four possible cues; **b** the six possible targets; **c** a trial example (i.e., neutral trial preceded by a double cue, the correct response being "right"); and **d** a summary of the scores computation. Adapted from Fan et al. (2002)

of trials with correct responses). Following Fan et al. (2002), we computed indexes (i.e., subtraction between two experimental conditions which had the same requirements in terms of working memory and motor planning but differed regarding the attentional resources involved) for the three attentional networks, both for RT and AS, and for each participant individually. This approach thus yields three distinct indices (e.g., Fan et al. 2002; Maurage et al. 2014). The *alerting effect* is computed by subtracting the mean score for the double cue trials from the mean score for the no cue trials (i.e., no cue − double cue); the *orienting effect* is calculated by subtracting the mean score for the spatial cue trials from the mean score for the central cue trials (i.e., central cue − spatial cue); the *executive control effect* is computed by subtracting the mean score for congruent flanker trials from the mean score for incongruent flanker trials (i.e., incongruent flanker − congruent flanker) (Fig. 1d). For both alerting and orienting effects, greater subtraction scores for RT (and lower for AS) indicate greater efficiency. In contrast, greater subtraction scores for RT (and lower for AS) on executive conflict indicated increased difficulty with executive control of attention (Fan et al. 2005).

Statistical analyses

All statistical analyses were performed using SPSS 21.0 ® (IBM, Inc.). The significance level was set at an alpha level of .05 (bilateral). First, descriptive statistics were performed for the two groups (BD and CP) and independent sample *t* tests were computed to explore group differences. Second, two types of analysis of variance (ANOVA) were performed, separately for AS and RT: (1) 2 × 4 × 3 ANOVA with group (CP, BD) as between-subjects factor and cue (no cue, central cue, double cue, spatial cue) and flanker (congruent, incongruent, neutral) as within-subjects factors; (2) 2 × 3 ANOVA with group (CP, BD) as between-subjects factor and attentional network (alerting, orienting, executive control) as within-subjects factor. For each ANOVA, significant main effects and interactions were followed up by post hoc independent samples *t* tests. As our main focus concerned the exploration of a potential deficit in binge drinking, the "Results" section will focus on group comparisons and the overall effects for each ANOVA (i.e., significant results not related to group differences) will be reported in the Supplementary materials.

Results

Demographic and psychopathological measures

As described in Table 1, groups did not significantly differ for age [$t(78) = 0.67$, $p = 0.51$], gender [$\chi^2(1, N = 80) = 3.41$, $p = 0.06$], depression [$t(78) = 0.75$, $p = 0.46$], state anxiety [$t(78) = 0.70$, $p = 0.49$], trait anxiety [$t(78) = 0.40$, $p = 0.69$], and tobacco consumption [$\chi^2(1, N = 80) = 1.05$, $p = 0.31$], confirming the correct group matching.

Experimental measures

General analysis

- AS: No main group effect was found [$F(1,78) = 1.24$, $p = 0.27$], nor any group x cue [$F(3234) = 1.88$, $p = 0.14$], or group x flanker [$F(2156) = 0.75$, $p = 0.48$] interactions.
- RT: No main group effect was found [$F(1,78) = 0.45$, $p = 0.51$] but significant group x cue [$F(3234) = 2.91$, $p < 0.05$, $\eta^2_p = .04$] and group x flanker [$F(2156) = 6.01$, $p < 0.01$, $\eta^2_p = .07$] interactions were found. Post hoc *t* tests between groups did not show significant difference for cue and flanker effects. In both groups, double cues led to shorter RT than central and no cues, but this difference was significantly stronger in CP than in BD [i.e., for central cues, $t(39) = 3.10$, $p < 0.01$, $\eta^2 = .11$, and for no cues, $t(39) = 2.54$, $p < 0.05$, $\eta^2 = .08$]. Concerning flanker, incongruent conditions led to slower RT than neutral and congruent ones, and this difference was larger in CP than BD [i.e., for neutral flankers, $t(39) = 2.86$, $p < 0.01$, $\eta^2 = .09$, and for Congruent flankers, $t(39) = 3.25$, $p < 0.01$, $\eta^2 = .12$] (Table 2).

Attentional networks analyses

- AS: no main group effect was found [$F(1,78) = 1.38$, $p = 0.24$] nor any group × attentional network interaction [$F(2156) = 1.38$, $p = 0.26$].
- RT: no main group effect was found [$F(1,78) = 0.005$, $p = 0.95$] but a group × attentional network interaction [$F(2156) = 10.33$, $p < 0.001$, $\eta^2_p = .12$] was observed. Post hoc *t* tests indicated that BD presented reduced efficiency for alerting [$t(78) = 2.31$, $p < 0.05$, $\eta^2 = .06$] and executive control [$t(78) = 3.18$, $p < 0.01$] networks, with a preserved performance for the orienting network [$t(78) = 1.03$, $p = 0.31$, $\eta^2 = .11$] (Fig. 2).

Complementary analyses

To ensure that the observed group differences did not merely mirror a more global alcohol use disorder, correlations were performed between attentional performance (i.e., RT for the three attentional networks) and AUDIT score. No significant relation was found between attentional networks and AUDIT score among BD [alerting

Table 2 Reaction times (RT; in milliseconds) and accuracy score (AS; percentage of correct answers) for binge drinkers (BD) and control participants (CP) in each experimental condition of the Attention Network Task

Cue types: mean (SD)							Flanker means
	Variable	Group	No cue	Central cue	Double cue	Spatial cue	
Flanker types: mean (SD)							
Congruent							Congruent
	RT	BD	491 (55.51)	449 (43.84)	450 (43.22)	412 (39.57)	451.16 (43.24)
		CP	509 (71.47)	466 (51.94)	450 (48.78)	426 (48.50)	463.67 (51.96)
	AS	BD	99.90 (0.66)	99.48 (1.68)	99.69 (1.11)	99.48 (1.40)	99.64 (0.77)
		CP	99.38 (1.78)	99.48 (1.93)	99.79 (0.92)	99.38 (1.78)	99.51 (0.85)
Incongruent							Incongruent
	RT	BD	588 (64.20)	574 (53.11)	571 (54.48)	508 (50.16)	560.42 (52.11)
		CP	593 (62.03)	578 (65.08)	562 (54.53)	500 (53.91)	557.96 (55.12)
	AS	BD	93.65 (7.49)	92.08 (7.54)	93.44 (6.11)	95.73 (6.29)	93.72 (5.08)
		CP	93.23 (7.59)	89.06 (11.31)	90.73 (10.49)	96.35 (4.64)	92.34 (7.42)
Neutral							Neutral
	RT	BD	484 (44.96)	448 (47.05)	444 (39.44)	409 (41.85)	446.96 (40.28)
		CP	493 (43.84)	461 (47.53)	453 (47.16)	421 (46.60)	457.23 (43.60)
	AS	BD	98.85 (2.11)	99.79 (0.92)	99.69 (1.11)	99.38 (1.51)	99.43 (0.94)
		CP	98.33 (2.80)	99.27 (1.60)	99.58 (1.27)	99.38 (1.78)	99.14 (1.15)
Cue means	RT	BD	520.22 (52.67)	488.83 (45.95)	487.24 (43.32)	442.73 (42.22)	
		CP	530.93 (55.98)	499.48 (51.56)	486.66 (48.92)	449.09 (48.53)	
	AS	BD	97.47 (2.65)	97.12 (2.66)	97.60 (2.18)	98.19 (2.46)	
		CP	96.98 (3.30)	95.94 (4.08)	96.70 (3.47)	98.37 (2.04)	

($r = -0.08$, $p = 0.64$), orienting ($r = 0.18$, $p = 0.26$), and executive control ($r = -0.01$, $p = 0.09$)]. In CP, no correlation was found for alerting ($r = 0.13$, $p = 0.43$) and executive control ($r = -0.02$, $p = 0.09$), but a positive correlation was found for orienting ($r = 0.36$, $p < 0.05$).

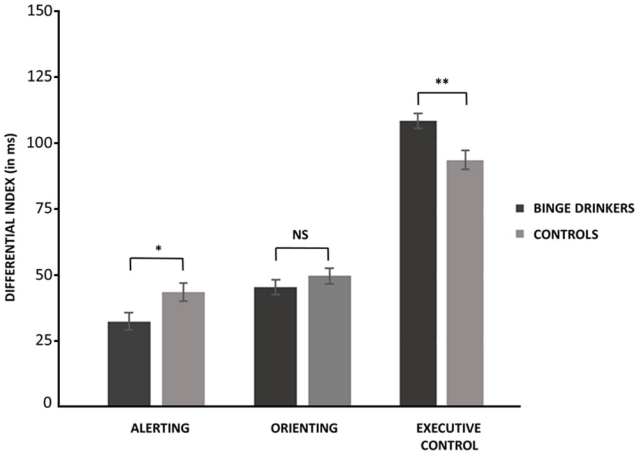

Fig. 2 Differential indexes (in milliseconds) for the three attentional networks (i.e., alerting, orienting, and executive control) among binge drinkers and control participants. *Bars* represent the mean value for each index in each group, and *whiskers* represent the standard error. *$p < .05$; **$p < .01$

Moreover, this absence of correlation between attentional performance and AUDIT score in BD was supported when the sample was split between participants who had an AUDIT score below the alcohol dependence threshold (AUDIT <20; $n = 32$) [alerting ($r = -0.12$, $p = 0.53$), orienting ($r = 0.33$, $p = 0.07$), executive control ($r = 0.10$, $p = 0.59$)], and those who had an AUDIT score above this threshold (AUDIT ≥20; $n = 8$) [alerting ($r = 0.20$, $p = 0.63$), orienting ($r = 0.50$, $p = 0.21$), executive control ($r = -0.28$, $p = 0.50$)]. Independent sample t tests also revealed no difference between these two groups [alerting ($t(37) = 0.24$, $p = 0.81$), orienting ($t(37) = 0.30$, $p = 0.76$), executive control ($t(37) = 0.39$, $p = 0.70$)].

Discussion

The main aim of the present study was to explore attentional abilities among BD using the Attention Network Task, which allows the integrated exploration of three attentional networks: alerting, orienting, and executive control. Results indicate that binge drinking is not related to a global impairment in all attentional networks but rather to a distinct pattern of alterations between impaired alerting/executive control networks, and preserved orienting abilities.

A first important insight is that, relative to control participants, BD demonstrate reduced ability in the executive control of attentional resources. Indeed, an increased interference effect of flanker incongruency on RT was observed in BD, indexing a difficulty to inhibit confusing and irrelevant contents (i.e., incongruent flankers) in order to (re)focus attentional resources on significant stimulations (i.e., the central arrow to be processed). This first central finding, showing impaired conflict resolution between pertinent and non-pertinent information in binge drinking, lends support to earlier studies exploring executive functions in binge drinking. As the executive control network is related to frontal activations (more specifically in the anterior cingulate cortex) and is correlated with more global inhibition abilities (Fan et al. 2005; Maurage et al. 2014), these results are in line with previous studies showing reduced activation in prefrontal areas among BD (e.g., Schweinsburg et al. 2010), greater motor impulsivity in a reaction time task (Scaife and Duka 2009), and poorer inhibition of prepotent response abilities (Henges and Marczinski 2012). As shown in Table 2, and while these differences did not reach significance, BD also presented globally faster RT than CP, this visuo-motor impulsivity being in line with previously reported data exploring attentional abilities (Scaife and Duka 2009; Townshend and Duka 2005). The present study thus corroborates that some executive subcomponents are impaired in binge drinking, and this deficit is also found in the executive control of attentional processing. Importantly, the current results actually showed that attentional impairments in binge drinking are not only observed for executive control but also for the alerting network; BD present a reduced ability to take advantage from the double cue in order to fasten the processing of the upcoming target. This indicates a difficulty to increase one's phasic alertness and therefore maintain a vigilance state throughout the task. Although impaired sustained attention had already been described in binge drinking using non-specific tasks also involving working memory (Hartley et al. 2004; Squeglia et al. 2011), the present result constitutes the first observation that binge drinking is also associated with an impairment of more basic attentional processing. This finding appears quite surprising regarding the expected result of a specific executive alteration, referring to the continuum hypothesis. However, recent investigations increasingly showed that binge drinking is a complex pattern of alcohol consumption with some divergences in the selection criteria across studies. In the current research, participants were selected via the BD score (≥16 for the BD group) which represents intense binge drinking pattern. It can thus be that, while high-level functions (memory, inhibition) are impaired even for moderate binge drinking habits as observed earlier, more basic abilities like attentional alert are only compromised among intense BD. These impaired alerting and executive control abilities were specifically observed for RT, which is in line with previous studies on psychopathological populations (e.g., Fernández et al. 2011; Heeren et al. 2014; Maurage et al. 2014; Pacheco-Unguetti et al. 2011) and reflects the fact that the easiness of this task led to a ceiling effect for AS.

Conversely, the orienting network appeared preserved in our sample of BD. As this network assesses the ability to orient attentional resources towards a specific information, this result suggests that even intense binge drinking habits are associated with preserved capability to efficiently orient one's attention towards an upcoming pertinent target. It is thus important to underline that the deficits observed here for alerting and executive control networks cannot be the mere consequence of a global attentional deficit in BD, as the orienting network is preserved, which also excludes the hypothesis that the present results might be related to a general group difference in terms of cognitive functioning or motivation. This proposal is further reinforced by the fact that attentional networks' integrity is based on the computation of a differential index (i.e., the subtraction between two experimental conditions), excluding that group differences are related to a global slowing down or misunderstanding of task requirements by BD. Moreover, the efficient group matching regarding sociodemographic and psychopathological variables, the absence of family history of alcohol dependence in all participants, as well as the correlational analyses showing that the general alcohol consumption pattern was not related to BD performance support the proposal that group differences observed on attentional abilities are specifically related to binge drinking. Yet, the cross-sectional nature of our design does not allow drawing any firm conclusion regarding the causality between binge drinking and attentional deficits, as attentional impairments may predate the development of binge drinking habits. It should also be noted that, as binge drinking scores now constitute a widely used way to explore binge drinking (e.g., Czapla et al. 2015; Kessler et al. 2013; Townshend et al. 2014), we relied on this procedure to select our groups and facilitate the comparison with other studies. However, there is currently no standard to determine cutoff scores for binge drinking and various cutoffs have been used since the initial proposal to focus on this score (Townshend and Duka 2005). While our participants' selection was not exclusively relying on this binge drinking score (as groups were clearly distinct for a large range of alcohol-related variables, as shown in Table 1), a definition of the more effective cutoff scores should be conducted in future works to standardize the exploration of binge drinking habits (also taking into account the variation across countries regarding the number of ethanol grams contained in a standard drink). Additionally, as some comorbid substance use or addictive disorder (e.g., cannabis) constituted exclusion criteria in the present study to ensure that observed

impairments were specifically related to alcohol consumption, the current results could not be generalizable to the whole population of binge drinkers and particularly to poly-user individuals. The influence of comorbidities between binge drinking and other drug consumption on attentional processes should be further explored, in line with what has already been conducted for other cognitive functions (e.g., Schweinsburg et al. 2011; Squeglia et al. 2014, 2015). Finally, even if the experimental paradigm used in this study (i.e., Attention Network Task) is widely validated, future studies might confirm the current results by using tasks specifically related to each attentional networks (e.g., interference task for executive control, vigilance task for alerting).

This first systematic exploration of attentional abilities in binge drinking bares several implications. At the fundamental level, it offers new insights regarding the neuropsychological pattern demonstrated by BD. Indeed, earlier studies had nearly exclusively focused on memory and executive functions, leading to the proposal that the deficits presented by BD were centrally related to high-level cognitive functions. The present study rather shows that BDs are also characterized by impairments for less complex cognitive abilities, such as alerting. Moreover, this study partially supports the continuum hypothesis, by showing that BD demonstrate similar deficits as alcohol-dependent individuals (Maurage et al. 2014) regarding executive attentional process. Nevertheless, alerting network is altered in BD while this result was not reported in alcohol-dependent patients during the ANT task (Maurage et al. 2014). Even if other studies have shown deteriorated alertness processing in alcohol dependence (Cordovil De Sousa Uva et al. 2010; Evert and Oscar-Berman 2001; Fein and Andrew 2011), the present study does not extend the continuum hypothesis towards all attentional processes. A direct comparison between BD and alcohol-dependent groups is thus necessary to clarify the validity and extent of the continuum hypothesis. At the clinical level, these results claim for reinforcing the current prevention campaigns to reduce binge drinking in youth, as they further prove the association between excessive episodic alcohol consumption and cognitive impairments in university students. These findings also highlight the potential usefulness of rehabilitation programs in binge drinking: as attentional abilities are crucial for efficient functioning in everyday life, the impairments observed here for alerting and executive control networks could be involved in the persistence of binge drinking behaviors and in the transition towards alcohol dependence, notably by facilitating the emergence and intensification of attentional biases. Programs focusing on attentional rehabilitation have indeed been successfully developed in alcohol dependence (Rupp et al. 2012) and specific rehabilitations of each attentional network have been proposed (Serino et al. 2007; Thimm et al. 2006). By implementing such programs as prophylactic tools, clinicians may foster beneficial cascade of downstream benefits.

Acknowledgements This research has been supported by the Belgian Fund for Scientific Research, the European Commission for Research on the problematic usage of information and communication technology [Grant ID: FP7-PEOPLE-2013-IEF-627999], and the "Fondation pour la Recherche en Alcoologie (FRA)". This research also received the support from a postdoctoral fellowship in clinical neuroscience from the "Helaers Foundation" and the "WBI World Excellence Grant: Competitive Cluster in Life Sciences—BIOWIN" (sub/2015/228106243177), both awarded to Alexandre Heeren.

Compliance with ethical standards All participants provided their written informed consent. All the procedures contributing to this work were approved by the local ethics committee and complied with the Helsinki Declaration of 1975, as revised in 2008.

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