

Review

A Systematic Review of the Balloon Analogue Risk Task (BART) in Alcohol Research

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Abstract

Risk-taking propensity has been crucial to the investigation of alcohol use and consequences. One measure, the balloon analogue risk task (BART), has been used consistently over the past two decades. However, it is unclear how this measure is related to alcohol outcomes. This paper systematically reviews the literature on the BART and alcohol outcomes. First, direct associations between the BART and alcohol use are reviewed including correlations, group comparisons, the BART's prediction of alcohol outcomes and BART performance after consuming alcohol. Then, potential moderators that explain when and for whom the BART is related to alcohol outcomes are reviewed. Finally, potential mechanisms that explain how the BART and alcohol outcomes are related are reviewed. This review reveals patterns in the BART suggesting risk-taking propensity may be related to changes in alcohol use over time; however, there is little evidence to suggest BART scores increase after consuming alcohol. Yet, additional research suggests adjusted average pump scores may be too simplistic for the amount of information the BART captures and understanding individual's patterns of responses on the BART is important for investigating its relation to alcohol outcomes. Finally, this review opens up several future directions for research to understand how risk-taking propensity is related to alcohol outcomes.

INTRODUCTION

Alcohol use is a hallmark risk-taking behavior where individuals seek pleasurable effects despite potential negative consequences. Behavioral economic models of alcohol use typically describe balancing costs with potential benefits of drinking (Hursh and Roma, 2016; Moore and Gullone, 1996). Adolescents who perceive greater benefits independent of perceived costs from using substances are more likely to engage in substance use (Fromme *et al.*, 1997). Willingness to pay more for alcohol, as measured by alcohol demand tasks, is associated with more weekly alcohol use and alcohol-related problems (Murphy *et al.*, 2009), heavy drinking (Murphy and MacKillop, 2006) and greater risk for alcohol use disorder (AUD) (Gray and MacKillop, 2014). Further, alcohol myopia theory (Steele and Josephs, 1990) describes how individuals can lose focus on distal negative consequences while under the influence of alcohol by attending to more salient activating cues rather than inhibitory cues. This in part accounts for increases in risky behavior after drinking such

as risky sex, driving under the influence, aggressive behavior and suicide (see Giancola *et al.*, 2010 for review). Thus, one's propensity for risk-taking both sober and intoxicated has been a key factor in understanding alcohol use and consequences.

One measure of risk-taking propensity, the balloon analogue risk task (BART) (Lejuez *et al.*, 2002), has grown in popularity within alcohol research over the past two decades. The task is designed to capture positively reinforced risk-taking where the likelihood of experiencing costs increases as risky behavior increases (Lejuez *et al.*, 2002). Similar to alcohol use, immediate decisions such as pumping a single balloon more to earn more money have both potential immediate and long-term consequences including the balloon exploding and lower earnings, respectively. Thus, the BART captures positive reinforcement of immediate rather than long-term decisions. The original BART allows participants to earn a small monetary reward for each pump to blow up a balloon, but at some point, the balloon explodes. Participants manually pump the balloon and if they choose

to ‘cash out’ before the balloon explodes, they keep the money, but if the balloon explodes they lose the money from that trial. The BART program allows researchers to manipulate the task by changing the number of trials and monetary reward per pump (Lejuez *et al.*, 2002). Additional adaptations of the BART have surfaced throughout the years making the BART extremely flexible for various research methods.

The BART has become a popular risk-taking propensity task, as one facet of impulsivity. Impulsivity is defined in different ways but can be thought of as the tendency to have difficulty controlling quick behavioral responses to both internal and external stimuli without considerations for consequences (Hollander *et al.*, 2016). Several multidimensional models of impulsivity use the BART as the sole risk-taking propensity measure (Andrews *et al.*, 2011; King *et al.*, 2014). Additionally, several preregistered protocols employ the BART to investigate alcohol-related outcomes (Bourque *et al.*, 2016; Emmerik-van Oortmerssen *et al.*, 2013; Manning *et al.*, 2018). The BART’s popularity is likely due to its broad external validity for risk-taking behaviors. It has been validated through associations with several real-world risk-taking behaviors such as alcohol use (Weafer *et al.*, 2011), substance use (Biernacki *et al.*, 2016; Lejuez *et al.*, 2002), gambling (Mishra *et al.*, 2017) and risky sexual behavior (Bornoalova *et al.*, 2008). However, research investigating the direction and magnitude of associations between the BART and alcohol outcomes has been mixed. King *et al.* (2014) reviewed neurological mechanisms of impulsivity and alcohol use, including three studies using the BART. They noted discrepancies in findings across studies, with one study suggesting the BART improves prediction over other impulsivity measures (Ferne *et al.*, 2010) and one suggesting no relation (Skeel *et al.*, 2008). As the number of alcohol studies using the BART has increased, many have failed to replicate original BART validation research. The current paper seeks to understand and contextualize these mixed findings through a systematic review of associations between the BART and alcohol outcomes. First, we discuss direct associations between alcohol use and BART performance including correlational data, group differences and prospective predictions. We review both potential moderators and mechanisms of these associations. Finally, we review the limitations of present BART literature and discuss future directions for using this task in alcohol research.

METHODS

Study identification

This systematic review looks across population, intervention, comparison and outcome (PICO) parameters to identify patterns in associations between the BART and alcohol outcomes, with the goal of offering direction for important parameters in future research. Due to the breadth of the questions posed, a systematic review was chosen over meta-analyses, which would require more precisely and narrowly defined PICO parameters (Ahn and Kang, 2018). While review procedures were not preregistered, authors reviewed the protocol and systematic review against the PRISMA checklist to ensure consistency with guidelines.

Articles were identified using University of Washington online library resources, which searches collections including Elsevier, PubMed, Web of Science, Gale, ProQuest, CrossRef, the Directory of Open Access Journals, SpringerLink, Taylor & Francis and PsychARTICLES. A Boolean search in October 2019 identified peer-reviewed journal articles published from 2002–2019 with the exact

term ‘Balloon Analogue Risk Task’ AND ‘alcohol’ OR ‘drink*.’ This search identified 585 results. Additionally, Google Scholar was used to search for the term “alcohol” within articles citing the original BART paper (Lejuez *et al.*, 2002). This produced 1100 results.

Study inclusion

The first author reviewed abstracts and method sections of each of the 1685 articles to determine if the study met inclusion criteria: (a) used the BART, (b) included at least one alcohol outcome measure (use, cravings, consequences) and (c) was not a duplicate listing. A total of 82 unique articles were initially included. After reading the full articles, seven articles were excluded for not reporting any results comparing the BART and alcohol outcomes, three were study protocols with no results, two were reviews and the source articles were already included and two reported the same BART results in the same sample so only one article was retained. Thus, a total of 69 articles are discussed in this systematic review. A PRISMA flow chart can be found in Fig. 1.

Balloon analogue risk task

As discussed earlier, the BART is an extremely flexible program, which has led to differences in how the BART is used. First, while the BART is designed to capture the latent variable of risk-taking propensity, several different indicators can be computed. Typical BART outcomes include an average number of pumps on unexploded balloons (i.e. adjusted average pumps; Lejuez *et al.*, 2002) and the number of exploded balloons (Reed *et al.*, 2012), with higher scores on each reflecting greater risk-taking. Studies have also looked at the number of pumps on individual trials (i.e. balloons; Ashenhurst *et al.*, 2014) or average pumps on trials after exploded balloons (i.e. post-failure mean pumps; Ashenhurst *et al.*, 2011). Additionally, new research suggests using inter-trial variability in pumps, defined as the standard deviation (SD) of total pumps divided by the mean of total pumps (Jentsch *et al.*, 2010). Some studies used structural equation modeling to create a latent risk-taking propensity factor from multiple BART outcomes (Courtney *et al.*, 2012; Worhunsky *et al.*, 2016; Yarosh *et al.*, 2014), while most use one or more observed indicators (e.g. average pumps, explosions, etc.).

The BART has also been adapted by adjusting the distribution of explosion point (Claus and Hutchison, 2012) for neuroimaging studies collecting activation data on each pump. The youth version (Y-BART; Lejuez *et al.*, 2007) allows children and adolescents to earn points rather than money. Finally, the automatic BART asks participants to enter the total number of pumps desired and they cannot change their response after inflation begins, allowing researchers to measure the total unadjusted number of pumps regardless of whether or not the balloon exploded (Pleskac *et al.*, 2008). Throughout this review, we refer to all outcomes across all BART settings as ‘risk-taking propensity’ for simplicity and to reflect assumptions made in the current literature of a unidimensional latent variable. However, research comparing different BART settings and outcomes is limited and is discussed later in this review. A list of the BART settings for each study is shown in Table 1.

Data extraction

Methods, results and relevant discussion from each article were extracted by at least one author and then organized into themes. Study quality was assessed using a modified Newcastle–Ottawa Scale

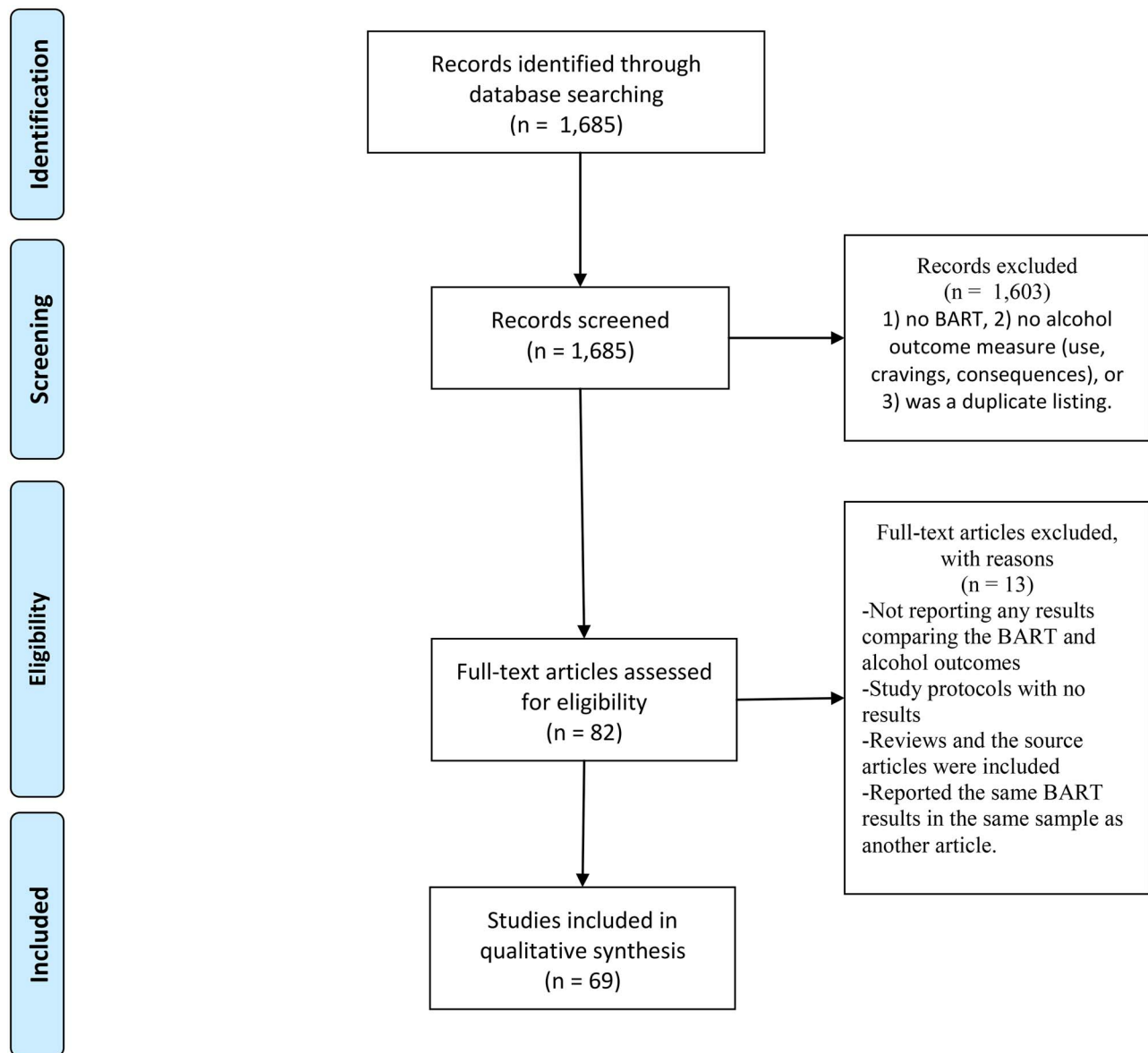


Fig. 1. PRISMA flow diagram.

(Wells *et al.*, 2000), which tailored items to be relevant for cross-sectional studies. The Cochrane Collaboration Tool for Assessing the Risk of Bias was used for randomized trials (Higgins *et al.*, 2020). Initial assessments were completed by one author and then verified by a second author. Discrepancies were discussed by at least two authors until consensus was reached. Each of the 69 articles is grouped and reviewed by the type of comparisons made. A total count of articles in each subsection is provided at the beginning of the section; however, many studies used multiple BART indicators and alcohol outcomes for their analyses or multiple samples. Some studies may be mentioned more than once due to differences in results dependent on measures or samples. Thus, the number of results reported in each subsection may not add up to the number of studies reviewed. Table 2 contains each study's methods and direct associations reported. Whether important patterns emerged based on measures or samples is discussed throughout the review.

RESULTS

Correlations between the BART and alcohol outcomes

Correlations between risk-taking measured by the BART and alcohol outcomes are mixed and often depend on outcomes used. Out of 69, 24 articles reported correlational results. Fifteen studies report no correlation between the BART and alcohol outcomes. Among college students, BART performance was unrelated to several measures of alcohol use and consequences (Ellingson *et al.*, 2018; Skeel *et al.*, 2008; Bogg *et al.*, 2012; MacPherson *et al.*, 2012; Dager *et al.*, 2014). One could argue lack of significant associations might be due to sample age, as risk-taking peaks in adolescence (Arnett, 1999; Lejuez *et al.*, 2002). However, four studies also reported no association between BART performance and alcohol use within adolescent samples (Janssen *et al.*, 2015; Crowley *et al.*, 2006; Bacio and Ray, 2016; Hamilton, *et al.*, 2014). Six studies also found no association with alcohol outcomes among the heavy-drinking community or

Table 1. Summary of BART settings and quality assessment of each study reviewed

| Article (N = 69) | BART type | BART settings | | | | Study quality assessment | | | |
|------------------|------------------------------------|----------------|-----------------------------------|--|--|---------------------------|---|---------------------|---|
| | | Max pumps | Average explosion point | Trials | Reward per pump | Selection (out of 2) | Comparability (out of 2) | Exposure (out of 3) | |
| 1 | Ahmadi <i>et al.</i> (2013) | BART | NR | NR | NR | NR | 1 | 0 | 3 |
| 2 | Aklin <i>et al.</i> (2005) | BART | 128 | NR | 30 | \$0.05 | 1 | 0 | 2 |
| 3 | Andrews <i>et al.</i> (2011) | BART | NR | NR | NR | NR | 1 | 0 | 3 |
| 4 | Ashenhurst <i>et al.</i> (2011) | BART | 64 | 32 pumps | 72 | \$0.01 | 1 | 2 | 2 |
| 5 | Ashenhurst <i>et al.</i> (2014) | BART | 64 | 32 pumps | 72 | \$0.003 | 1 | 2 | 2 |
| 6 | Bacio and Ray <i>et al.</i> (2016) | BART | NR | 32 pumps | 72 | \$0.005 | 1 | 0 | 2 |
| 7 | Banducci <i>et al.</i> (2015) | BART | 128 | 64 pumps | 60 | \$0.005, \$0.01, \$0.05 | 1 | 1 | 2 |
| 8 | Barker <i>et al.</i> (2017) | BART | NR | NR | NR | NR | 1 | 1 | 3 |
| 9 | Bogg <i>et al.</i> (2012) | BART | NR | NR | 2 8-min blocks (M = 36.63 trials total; SD = 4.79) | \$0.00–\$5.15 | 1 | 0 | 3 |
| 10 | Brailovskaia <i>et al.</i> (2018) | BART | NR | NR | 30 | €0.05 | 2 | 1 | 2 |
| 11 | Campbell <i>et al.</i> (2013) | BART | 128 | 64 pumps | 45 | \$1.00 | 1 | 1 | 2 |
| 12 | Caneto <i>et al.</i> (2018) | Y-BART | NR | NR | 30 | 5 points | Some risk of bias: assignment to groups was not reported; however, groups did not differ on key variables at baseline | | |
| 13 | Claus <i>et al.</i> (2018) | Y-BART | 8, 11 | 5, 8 | NR | Points (unspecified) | 1 | 1 | 3 |
| 14 | Claus and Hutchinson (2012) | Y-BART | 8, 11 | 5, 8 | NR | Points (unspecified) | 1 | 1 | 3 |
| 15 | Clay <i>et al.</i> (2018) | BART | NR | NR | NR | Money (unspecified) | 1 | 0 | 2 |
| 16 | Clay and Parker (2018) | BART | NR | NR | 20 | \$0.05 | 1 | 2 | 2 |
| 17 | Corbin <i>et al.</i> (2015) | BART | 128 | NR | 30 | NR | Low risk of bias | | |
| 18 | Courtney <i>et al.</i> (2012) | BART | 64 | 32 pumps | 72 | \$0.003 | 1 | 0 | 2 |
| 19 | Crowley <i>et al.</i> (2006) | BART | 128 | NR | 30 | \$0.01 | 1 | 1 | 2 |
| 20 | Dager <i>et al.</i> (2014) | BART | NR | NR | NR | NR | 1 | 2 | 3 |
| 21 | DeMartini <i>et al.</i> (2014) | Automatic BART | 128 | NR | 30 | \$0.01 | 1 | 1 | 2 |
| 22 | Dougherty <i>et al.</i> (2015) | Y-BART | NR | NR | 30 | Points (unspecified) | 1 | 1 | 2 |
| 23 | Ellingson <i>et al.</i> (2018) | BART | NR | NR | NR | NR | 1 | 1 | 2 |
| 24 | Erskine-Shaw <i>et al.</i> (2017) | Y-BART | NR | NR | 30 | Points (unspecified) | Some risk of bias: systematic differences in alcohol and risk-taking between randomized groups at baseline; however, subsequent analyses included these as covariates | | |
| 25 | Euser <i>et al.</i> (2011) | Automatic BART | 128 | 64 pumps | 60 | NR | Some Risk of Bias: Placebo manipulation check not reported | | |
| 26 | Fein and Chang (2008) | BART | 20 | NR | 60 | \$0.50 (+\$0.02 per pump) | 1 | 0 | 3 |
| 27 | Fernie <i>et al.</i> (2010) | BART | 128 | NR | 30 | 5p | 1 | 1 | 2 |
| 28 | Fernie <i>et al.</i> (2013) | Y-BART | 128 | NR | 30 | Points (unspecified) | 1 | 1 | 2 |
| 29 | Forster <i>et al.</i> (2016) | BART | NR | NR | 2 8-min blocks (M = 17 trials per block; SD = 2) | Incremental | 2 | 1 | 3 |
| 30 | Gorka <i>et al.</i> (2015) | Automatic BART | 128 | NR | 30 | \$0.02 | 1 | 2 | 2 |
| 31 | Hamilton <i>et al.</i> (2014) | Y-BART | NR | NR | NR | NR | 1 | 1 | 2 |
| 32 | Hawn <i>et al.</i> (2019) | BART | NR | NR | 30 | \$0.05 | 1 | 1 | 2 |
| 33 | Heinz <i>et al.</i> (2013) | BART | 128 | NR | 30 | \$0.01 | Low risk of bias | | |
| 34 | Heinz <i>et al.</i> (2016) | BART | NR | NR | NR | \$0.05 | 2 | 2 | 2 |
| 35 | Holmes <i>et al.</i> (2009) | Y-BART | NR | NR | 30 | 1 Point | 1 | 1 | 2 |
| 36 | Janssen <i>et al.</i> (2015) | Automatic BART | 128 | 64 pumps | 20 (2 × 10 trials) | NR | 1 | 2 | 2 |
| 37 | Kalapatapu <i>et al.</i> (2013) | BART | NR | NR | NR | NR | 2 | 0 | 2 |
| 38 | Kelley <i>et al.</i> (2012) | BART | NR | NR | NR | NR | 1 | 0 | 2 |
| 39 | Kim <i>et al.</i> (2018) | BART | NR | NR | 30 | NR | 2 | 0 | 2 |
| 40 | Lannoy <i>et al.</i> (2017) | Y-BART | 20 | NR | 90 | 1 point | 1 | 0 | 3 |
| 41 | Ledgerwood <i>et al.</i> (2009) | BART | NR | NR | 30 | \$0.05 | 1 | 2 | 2 |
| 42 | Lee and Yun (2014) | BART | NR | NR | 100 | NR | 1 | 0 | 3 |
| 43 | Lejuez <i>et al.</i> (2002) | BART | Blue 128 Orange 8 Yellow 32 | Blue 64 pumps Orange 4 pumps Yellow 16 pumps | 90 | \$0.05 | 1 | 2 | 2 |

(Continued)

Table 1. Continued

| Article (N = 69) | BART type | BART settings | | | | Study quality assessment | | | |
|------------------|-----------------------------------|---------------------------|-------------------------|----------|-----------------|---------------------------|--|---------------------|---|
| | | Max pumps | Average explosion point | Trials | Reward per pump | Selection (out of 2) | Comparability (out of 2) | Exposure (out of 3) | |
| 44 | Lovallo <i>et al.</i> (2014) | BART | NR | Random | NR | NR | 1 | 1 | 3 |
| 45 | MacPhearson <i>et al.</i> (2010) | Y-BART | 128 | 64 pumps | 30 | 1 point | 1 | 1 | 2 |
| 46 | MacPhearson <i>et al.</i> (2012) | Automatic BART | 128 | 64 pumps | 30 | \$0.01 | 1 | 0 | 2 |
| 47 | Moallem and Ray (2012) | BART | NR | NR | NR | NR | 1 | 2 | 2 |
| 48 | Murray <i>et al.</i> (2015) | BART | NR | NR | NR | NR | 1 | 2 | 3 |
| 49 | Neal and Gable (2019) | BART | NR | NR | 30 | NR | 1 | 0 | 2 |
| 50 | Padovano <i>et al.</i> (2019) | Automatic BART (Baseline) | 128 | NR | 30 | NR | 1 | 2 | 2 |
| | | Automatic BART (EMA) | 128 | NR | 1 | NR | | | |
| 51 | Park <i>et al.</i> (2020) | BART | NR | NR | 30 | Money (not specified) | 2 | 1 | 3 |
| 52 | Peacock <i>et al.</i> (2013) | BART | NR | 64 pumps | 30 | \$0.05 | 1 | 1 | 2 |
| 53 | Pennington <i>et al.</i> (2014) | BART | NR | NR | NR | NR | 1 | 1 | 3 |
| 54 | Prisciandaro <i>et al.</i> (2011) | BART | NR | NR | 30 | \$0.02 | Low risk of bias | | |
| 55 | Qu <i>et al.</i> (2015) | BART | 12 | NR | NR | \$0.25 | 1 | 1 | 3 |
| 56 | Ravenzwaaij <i>et al.</i> (2011) | BART | NR | NR | 30 | NR | 1 | 0 | 2 |
| 57 | Reed <i>et al.</i> (2012) | BART | NR | 64 pumps | 15 | \$0.05 | 1 | 0 | 2 |
| 58 | Reynolds <i>et al.</i> (2006) | BART | NR | NR | 30 | \$0.10, \$0.25, \$0.50 | 1 | 1 | 2 |
| 59 | Rose <i>et al.</i> (2014) | BART | NR | 64 pumps | 20 | \$0.05 | Some risk of bias: placebo manipulation check was not reported | | |
| 60 | Schmidt <i>et al.</i> (2017) | BART | NR | NR | NR | NR | 1 | 1 | 2 |
| 61 | Sehrgig <i>et al.</i> (2019) | BART | 12 | NR | 100 | €0.05 | 2 | 1 | 3 |
| 62 | Skeel <i>et al.</i> (2008) | Y-BART | NR | 64 pumps | NR | Points (unspecified) | 1 | 0 | 2 |
| 63 | Soder <i>et al.</i> (2019) | BART | 20 | NR | 60 | \$0.50 (+\$0.02 per pump) | 1 | 1 | 3 |
| 64 | Thompson <i>et al.</i> (2012) | BART | NR | NR | NR | NR | 2 | 1 | 2 |
| 65 | Wang <i>et al.</i> (2018) | BART | NR | NR | 30 | ¥0.05 | 2 | 1 | 3 |
| 66 | Wang <i>et al.</i> (2016) | BART | NR | NR | 30 | ¥0.05 | 2 | 1 | 3 |
| 67 | Weafer <i>et al.</i> (2011) | BART | NR | NR | 20 | \$0.01 | 1 | 0 | 2 |
| 68 | Worhunsy <i>et al.</i> (2016) | BART | NR | NR | NR | NR | 1 | 2 | 3 |
| 69 | Yarosh <i>et al.</i> (2014) | BART | NR | NR | NR | NR | 1 | 1 | 3 |

Notes: Y-BART = Youth BART, NR = not reported, M = mean.

alcohol-dependent samples (Kalapatapu *et al.*, 2013; Hawn *et al.*, 2019; Forster *et al.*, 2016; Claus and Hutchison, 2012; Brailovskaia *et al.*, 2018; Heinz *et al.*, 2016). However, some studies used samples with potential comorbid disorders including inpatient psychiatric patients (Brailovskaia *et al.*, 2018) and veterans with and without post-traumatic stress disorder (PTSD) (Hawn *et al.*, 2019). Comorbid mental health disorders may impact associations between the BART and alcohol outcomes, which is discussed further in the Moderators section. Overall, cross-sectional research largely suggests BART performance is not related to alcohol use or consequences. Further, results are not dependent on sample size with samples ranging from N = 27–1038.

Seven articles reported a positive association where greater risk-taking on the BART is correlated with greater alcohol use and consequences. In the original BART paper, Lejuez *et al.* (2002) reported BART performance was associated with a greater risk for AUD. Three studies demonstrated greater risk-taking on the BART was linked to more alcohol use among US veterans with and without PTSD (Hawn *et al.*, 2019), adults with and without attention-deficit hyperactivity disorder (ADHD) (Weafer *et al.*, 2011) and adolescents (Aklin *et al.*, 2005). Over a period of 1.5 years, changes in adjusted average pumps on the BART were positively associated with changes in self-reported risk-taking, including alcohol use, among adolescents (Qu *et al.*, 2015). Two studies found BART performance was related to greater alcohol use but only for some outcome measures (DeMartini *et al.*, 2014; Gorka *et al.*, 2015). BART performance may only relate to

certain measures of alcohol outcomes; however, across studies, it is difficult to identify patterns of which alcohol outcomes are related to risk-taking propensity measured by the BART.

Only two studies reported a negative association between alcohol outcomes and BART performance, where greater BART risk-taking was associated with less alcohol use and consequences. Surprisingly, DeMartini *et al.* (2014) found average pumps were positively related to the quantity of use but negatively related to the frequency of use. This contradictory pattern of correlations with alcohol outcomes highlights how inconsistencies observed with the BART can be found even within one study sample. Among a sample of non-treatment-seeking heavy drinkers, those with higher BART scores had fewer AUD symptoms but BART performance was unrelated to alcohol use quantity, frequency, or binge drinking frequency (Ashenhurst *et al.*, 2011). Overall, correlation analyses demonstrate inconsistent associations between the BART and alcohol outcomes. Evaluating specific indicators used by each study are listed in Table 2, there is considerable variability in both alcohol outcome measures and BART indicators. However, a consistent pattern of which measures are or are not related is not easily identified between studies.

Group differences on the BART by alcohol use status

Fifteen studies grouped participants by drinking or diagnostic status to compare BART performance. Twelve studies reported no difference between groups. Four found no difference in BART risk-taking

Table 2. Summary of literature reviewed including samples, measures and direct associations

| | Article (N = 69) | Sample (size) | BART indicator | Alcohol indicator | Diagnostic criteria (if applicable) | Time-scale | Association |
|--|------------------------------------|---|--|--|--|-----------------|--------------------------------------|
| 1 | Ahmadi <i>et al.</i> (2013) | College (N = 92) | Adjusted Average pumps | Heavy vs. light drinkers | 1) AUD DSM-IV criteria OR 2) drinking more than half of the weeks over 6 months and typically binge-drink | Cross-sectional | n.s. |
| 2 | Aklin <i>et al.</i> (2005) | Adolescents (N = 51) | Adjusted average pumps | Drinking status | | Cross-sectional | Positive |
| 3 | Andrews <i>et al.</i> (2011) | Adults (N = 49) | Adjusted average pumps | Family history status | | Cross-sectional | n.s. |
| 4 | Ashenhurst <i>et al.</i> (2011) | Adult heavy drinkers (N = 198) | Adjusted average pumps | AUD symptoms | DSM-IV | Cross-sectional | Negative |
| | | | | Frequency | | | n.s. |
| | | | | Binge frequency | | | n.s. |
| | | | | Typical quantity | | | n.s. |
| | | | Variability of pumps | AUD symptoms | | | n.s. |
| | | | | Frequency | | | n.s. |
| | | | | Binge frequency | | | n.s. |
| | | | | Typical quantity | | | n.s. |
| | | | Post-failure mean pumps | AUD symptoms | | | Negative |
| | | | | Frequency | | | n.s. |
| | | | | Binge frequency | | | n.s. |
| | | | | Typical quantity | | | Negative |
| 5 | Ashenhurst <i>et al.</i> (2014) | Adult heavy drinkers (N = 295) | Number of pumps (each trial) | Alcohol problem severity (index of multiple measures) | | Cross-sectional | NR |
| 6 | Bacio and Ray <i>et al.</i> (2016) | Latino adolescents (N = 129) | Adjusted average pumps | Drinking onset | | Cross-sectional | n.s. |
| 7 | Banducci <i>et al.</i> (2015) | Mother-child dyads (N = 277) | Post-failure mean pumps Adjusted average pumps | Drinking onset Quantity | | Cross-sectional | – |
| 8 | Barker <i>et al.</i> (2017) | HIV-infected Adults (N = 201) | Adjusted average pumps | AUDIT | | Cross-sectional | NR |
| 9 | Bogg <i>et al.</i> (2012) | College students (N = 27) | Total pumps Average pumps Number of explosions Number of cash-outs Amount earned | Typical quantity Typical quantity Typical quantity Typical quantity Typical quantity | | Cross-sectional | n.s. n.s. n.s. n.s. n.s. |
| 10 | Brailovskaia <i>et al.</i> (2018) | Psychiatric inpatient adults (N = 63) Healthy adults (N = 102) | Adjusted average pumps | AUDIT-C | | Cross-sectional | n.s. |
| 11 | Campbell <i>et al.</i> (2013) | Adults (N = 34) | Adjusted average Pumps | Long-term users vs. nonusers | 15+ years of drinking 10+/8+ drinks for males/females | Cross-sectional | Negative |
| Non-drinkers reported no more than three drinks per day on average during lifetime | | | | | | | |
| 12 | Caneto <i>et al.</i> (2018) | Young adults (N = 51) | Adjusted average pumps | Alcohol (BAC 0.08%) | | 30 min, 60 min | Positive |
| 13 | Claus <i>et al.</i> (2018) | Adolescents (N = 189) | Adjusted average pumps | Alcohol (4.66) vs. MJ (1.08) vs. Alcohol + MJ (6.99) vs. control (0.62) | Used more than 1 day in the past month | Cross-sectional | n.s. |
| | | | Proportion of explosions | Alcohol vs. MJ vs. alcohol + MJ vs. control | | | n.s. |
| 14 | Claus and Hutchinson (2012) | Adult heavy drinkers (N = 79) | Adjusted average pumps | AUDIT | | Cross-sectional | Positive (trend) |
| | | | Proportions of explosions | AUDIT | | | Positive (trend) |
| 15 | Clay <i>et al.</i> (2018) | College students (N = 31) | Adjusted average Pumps | ADQ AUDIT Quantity ΔCraving | | Cross-sectional | n.s. n.s. n.s. Positive |
| 16 | Clay and Parker (2018) | Young adults (N = 39) | Adjusted average pumps | Number of drinks | | Cross-sectional | n.s. |
| 17 | Corbin <i>et al.</i> (2015) | Young adults (N = 157) | Average pumps | Alcohol (BAC .081%) | | 15 min, 30 min | NR |
| 18 | Courtney <i>et al.</i> (2012) | Adult drinkers (N = 155) | Risk factor (latent) | Alcohol use Problems | | Cross-sectional | n.s. Negative |
| 19 | Crowley <i>et al.</i> (2006) | Adolescents (N = 40) | Total pumps | Quantity Frequency | | Cross-sectional | n.s. n.s. |
| 20 | Dager <i>et al.</i> (2014) | College student drinkers (N = 27) | Adjusted average pumps | BYAAQC | | 1 year | n.s. |
| 21 | DeMartini <i>et al.</i> (2014) | Young adults (N = 58) | Total pumps | ΔAlcohol Use Peak drinking—lifetime Peak drinking—3 months Peak drinking—frequency | | Cross-sectional | n.s. Positive n.s. Negative |
| | | | Variability in pumps | Peak drinking—lifetime Peak drinking—3 months Peak drinking—frequency | | | Negative Negative Positive |

(Continued)

Table 2. Continued

| | Article (N = 69) | Sample (size) | BART indicator | Alcohol indicator | Diagnostic criteria (if applicable) | Time-scale | Association |
|----|----------------------------------|--|-------------------------|--|---|-----------------------------|--------------------------------------|
| 22 | Dougherty <i>et al.</i> (2015) | Early adolescents (N = 386) | Adjusted average pumps | Family history status | | 6-month intervals/36 months | NR |
| 23 | Ellingson <i>et al.</i> (2018) | College students (N = 1038) | Total pumps | Quantity | | Cross-sectional | n.s. |
| 24 | Erskin-Shaw <i>et al.</i> (2017) | College students (N = 99) | Adjusted average pumps | Alcohol (BAC 0.069%) | | 20 min | n.s. |
| 25 | Euser <i>et al.</i> (2011) | Young adult males (N = 64) | Average pumps | Alcohol (BAC ~ 0.072%) | | 50 min | n.s. |
| 26 | Fein and Chang (2008) | Alcohol dependent adults (N = 22) | Adjusted average pumps | Quantity Family History of AUD (DSM-IV) | | Cross-sectional | NR NR |
| 27 | Fernie <i>et al.</i> (2010) | University staff & students (N = 75) | Adjusted average pumps | Alcohol involvement (latent variable) | | Cross-sectional | Positive |
| 28 | Fernie <i>et al.</i> (2013) | Adolescents (N = 287) | Adjusted average pumps | Alcohol involvement (latent variable) | | 6-month intervals/24 months | Positive |
| 29 | Forster <i>et al.</i> (2016) | Alcohol or substance dependent adults (N = 21) | ΔAdjusted average pumps | Frequency | | 1-month intervals/3 months | n.s. |
| 30 | Gorka <i>et al.</i> (2015) | Adult sibling pairs (N = 87 pairs) | Average pumps | Quantity Frequency of binge Drinking AUDIT | | Cross-sectional | n.s. Positive n.s. |
| 31 | Hamilton <i>et al.</i> (2014) | Adolescents & mothers (N = 111 pairs) Adolescents (N = 180) | Adjusted average pumps | Drinking status Drinking onset | | 1-year intervals/3 years | n.s. n.s. |
| 32 | Hawn <i>et al.</i> (2019) | US Veterans (N = 302) | Adjusted average pumps | Quantity Frequency of binge Drinking | | Cross-sectional | Positive n.s. |
| 33 | Heinz <i>et al.</i> (2013) | Young adults (N = 146) | Adjusted average pumps | Alcohol (BAC .088%) | | 15 min | Positive |
| 34 | Heinz <i>et al.</i> (2016) | US Veterans (N = 68) | Adjusted average pumps | Quantity Frequency Craving | | 2–3 Assessments/week | n.s. n.s. Positive |
| 35 | Holmes <i>et al.</i> (2009) | Adults with and without Bipolar Disorder (N = 80) | Adjusted average pumps | BD + AUD history vs. control BD + AUD history vs. BD Number of explosions BD + AUD history vs. control BD + AUD History vs. BD | History of alcohol abuse or dependence (DSM-IV-TR) | Cross-sectional | n.s. n.s. Positive Positive |
| 36 | Janssen <i>et al.</i> (2015) | Adolescents (N = 284) | Average pumps | Quantity Heavy episodic Drinking | | 6 months intervals/2 years | n.s. n.s. |
| 37 | Kalapatapu <i>et al.</i> (2013) | US Veterans with PTSD + AUD (N = 30) | Adjusted Average pumps | Quantity | | Cross-sectional | n.s. |
| 38 | Kelley <i>et al.</i> (2012) | US Veterans (N = 262) | Adjusted average pumps | Quantity Frequency Desire to cut down Impaired control | | Pre- and post-deployment | NR |
| 39 | Kim <i>et al.</i> (2018) | Adults with AUD (N = 330) | Adjusted average pumps | Onset of problems Dependence severity (latent) | | Cross-sectional | n.s. Positive |
| 40 | Lannoy <i>et al.</i> (2017) | College students (N = 40) | Adjusted average pumps | Binge vs. non-binge drinkers Number of explosions Pumps before explosions Total Pumps | Cut off of 16 on binge drinking score (see source article for formula) | Cross-sectional | n.s. n.s. n.s. n.s. |
| 41 | Ledgerwood <i>et al.</i> (2009) | Adults (N = 102) | Adjusted average pumps | SUD history + gambling vs. control | Lifetime abuse or dependence of cocaine, opiates, marijuana or alcohol (DSM-IV) | Cross-sectional | n.s. |
| 42 | Lee and Yun (2014) | Adults (N = 21) | Adjusted average pumps* | Alcohol (BAC 0.068%) | | 60–90 min | n.s. |
| 43 | Lejuez <i>et al.</i> (2002) | Young adults (N = 86) | Adjusted average Pumps | AUDIT | | Cross-sectional | Positive |

(Continued)

Table 2. Continued

| | Article (N = 69) | Sample (size) | BART indicator | Alcohol indicator | Diagnostic criteria (if applicable) | Time-scale | Association |
|----|--|--------------------------------------|---|---|--|--------------------------|-------------|
| 44 | Lovallo <i>et al.</i> (2014) | Young adults (N = 314) | Adjusted average pumps | AUDIT | | Cross-sectional | NR |
| | | | | Family history of AUD | | | n.s. |
| 45 | MacPhearson <i>et al.</i> (2010) | Early adolescents (N = 277) | Adjusted average pumps | Drinking status | | 1-year intervals/3 years | n.s. |
| | | | ΔAdjusted average pumps | Drinking status | | | Positive |
| 46 | MacPhearson <i>et al.</i> (2012) | College students (N = 116) | Average pumps | Frequency of binge | | Cross-sectional | n.s. |
| | | | | Drinking onset | | | n.s. |
| | | | | Consequences | | | n.s. |
| 47 | Moallem and Ray (2012) | Adult drinkers and smokers (N = 387) | Adjusted average pumps | Heavy drinkers vs. Smokers vs. Heavy drinker + smoker | NIAAA heavy drinking (14+/7+ drinks in a week or 5+/4+ drinks on one occasion in the past month) | | |
| | Smoking only groups may be drinkers but they did not meet the above criteria | Cross-sectional | n.s. | | | | |
| | | | Variability in pumps | Heavy drinkers vs. Smokers vs. Heavy drinker + Smoker | | | n.s. |
| 48 | Murray <i>et al.</i> (2015) | Adults (N = 77) | Adjusted average pumps | Alcohol vs. poly use alcohol/poly vs. control | Alcohol dependence | Cross-sectional | n.s. |
| | | | | | DSM-IV (abstinent for 1 month) | | Positive |
| 49 | Neal and Gable (2019) | College students (N = 44) | Number of pumps | Alcohol cues vs. neutral cues | Within-subjects design | Cross-sectional | n.s. |
| | | | | Alcohol cues vs. neutral cues | | | n.s. |
| 50 | Padovano <i>et al.</i> (2019) | Adolescents (N = 29) | Adjusted average pumps number of explosions | Craving | | Daily/1 week | n.s. |
| | | | Number of pumps (1 trial) | Craving | | | n.s. |
| 51 | Park <i>et al.</i> (2020) | Adults (N = 638) | Adjusted average pumps | AUD severity (DSM-IV) | | Cross-sectional | NR |
| 52 | Peacock <i>et al.</i> (2013) | Young adults (N = 28) | Adjusted Average pumps | Alcohol (BAC .06%) | | 40 min | n.s. |
| | | | Total earnings | Alcohol (BAC 0.06%) | | | n.s. |
| | | | Number of explosions | Alcohol (BAC 0.06%) | | | n.s. |
| 53 | Pennington <i>et al.</i> (2014) | US Male Veterans with AUD (N = 10) | Adjusted average pumps* | Quantity | | Cross-sectional | NR |
| | | | | AUDIT | | | |
| 54 | Prisciandaro <i>et al.</i> (2011) | Adults with AUD + BD (N = 30) | Adjusted average pumps | Treatment dropout | | Cross-sectional | Positive |
| 55 | Qu <i>et al.</i> (2015) | Adolescents (N = 24) | ΔAdjusted average pumps | ΔRule breaking (including alcohol use) | | 1.5 years | Positive |
| 56 | Ravenzwaaj <i>et al.</i> (2011) | Male college students (N = 18) | Number of pumps | Alcohol (BAC 0.05%) | | 40 min | n.s. |
| | | | | Alcohol (BAC 0.10%) | | | n.s. |
| | | | % of cash-outs | Alcohol (BAC .05%) | | | n.s. |
| | | | | Alcohol (BAC .10%) | | | n.s. |
| 57 | Reed <i>et al.</i> (2012) | Adult females (N = 46) | Adjusted average pumps | Heavy drinkers vs. light drinkers | NIAAA 7+ drinks per week | 15 min, 1 h, 2 h, 4 h | n.s. |
| | | | | Alcohol (BAC 0.056%) | | | n.s. |
| | | | | Alcohol (BAC 0.092%) | | | n.s. |
| 58 | Reynold <i>et al.</i> (2006) | Young adults (N = 24) | Adjusted average pumps | Alcohol (BAC 0.02–0.04%) | | 15 min, 105 min | n.s. |

(Continued)

Table 2. Continued

| | Article (N = 69) | Sample (size) | BART indicator | Alcohol indicator | Diagnostic criteria (if applicable) | Time-scale | Association |
|----|--------------------------------|--------------------------------------|------------------------|---|---|--------------------------|-------------------------|
| 59 | Rose <i>et al.</i> (2014) | College students (N = 142) | Adjusted average pumps | Alcohol (BAC 0.06–0.08%) Craving | | 20 min | n.s. Positive |
| 60 | Schmidt <i>et al.</i> (2017) | Adult treatment seekers (N = 105) | Adjusted average pumps | Alcohol (BAC 0.072%) quantity AUD vs. poly SUD | Any alcohol use disorder (DSM-IV-TR; at least 29 days abstinent) | 4 months | n.s. Positive |
| 61 | Sehrig <i>et al.</i> (2019) | Adults in-patient treatment (N = 74) | Average pumps | AUD vs. control | AUD (DSM-5) post-detox | Cross-sectional | n.s. |
| | | | Number of explosions | AUD vs. control | | | n.s. |
| 62 | Skeel <i>et al.</i> (2008) | College students (N = 114) | Adjusted average pumps | Quantity | | Daily/2 weeks | n.s. |
| 63 | Soder <i>et al.</i> (2019) | College Students (N = 85) | Number of pumps | Quantity | | Cross-sectional | NR |
| 64 | Thompson <i>et al.</i> (2012) | Adults in-patient treatment (N = 58) | Adjusted average pumps | SUD vs. control | Any SUD (DSM-IV; 57% AUD) | Cross-sectional | n.s. |
| 65 | Wang <i>et al.</i> (2018) | Male adults (N = 81) | Adjusted average pumps | Relapsed vs. no relapse | AUD (DSM-IV) | 2 sessions/1 week | Positive |
| | | | | No relapse vs. control | Relapse was defined as having at least 1 drink containing alcohol after treatment | | n.s. |
| 66 | Wang <i>et al.</i> (2016) | Male adults (N = 40) | Adjusted average pumps | AUD vs. control | AUD (DSM-IV) | 2 sessions/1 week | Positive |
| 67 | Weafer <i>et al.</i> (2011) | Young adults (N = 54) | Number of pumps | Quantity | | | Positive |
| | | | | Frequency | | | Positive |
| 68 | Worhunsky <i>et al.</i> (2016) | Young adults (N = 36) | Risky choice (latent) | ≤ 3 drinks vs. 4+ drinks | Maximum number of drinks consumed in 24 h period in past 6 months | 1-month intervals/1 year | n.s. |
| | | | | ΔAlcohol Use | | | n.s. |
| 69 | Yarosh <i>et al.</i> (2014) | Adults (N = 69) | Risk-taking (latent) | Family history of AUD (DSM-IV) | | Cross-sectional | n.s. |

Note: Significant results are in bold. n.s. = non-significant, NR = not reported.

depending on the amount of alcohol typically consumed (Worhunsky *et al.*, 2016; Ahmadi *et al.*, 2013; Reed *et al.*, 2012; Lannoy *et al.*, 2017). An additional three studies found no difference in BART performance between those diagnosed with AUD and healthy control groups (Thompson *et al.*, 2012; Sehrig *et al.*, 2019; Wang *et al.*, 2018). Additionally, one study found no BART difference between those with comorbid substance use disorders (SUDs) (including alcohol) and gambling problems compared to healthy controls (Ledgerwood *et al.*, 2009). Finally, four studies compared alcohol with other types of substance use and found no difference in BART performance, suggesting risk-taking propensity does not differ between users of alcohol, marijuana (MJ) or tobacco (Claus *et al.*, 2018; Moallem and Ray, 2012) nor between polysubstance compared to single substance users (Murray *et al.*, 2015; Schmidt *et al.*, 2017).

Four studies found differences between groups suggesting more alcohol use is associated with greater risk-taking propensity measured by the BART. One study found those with comorbid AUD and bipolar disorder (BD) had more explosions on the BART compared to those with only BD and healthy controls; however, other BART outcomes were non-significant (Holmes *et al.*, 2009). Further, alcohol and polysubstance dependent groups had significantly higher BART scores compared to a control group (Murray *et al.*, 2015). Male patients diagnosed with alcohol dependence according to DSM-IV criteria had higher BART scores than healthy controls (Wang *et al.*, 2016). Participants had significantly higher BART scores if they relapsed within 3 months of treatment compared to healthy controls. However, there was not a significant difference between alcohol-dependent individuals who did not relapse and other groups (Wang *et al.*, 2018). The latter two studies were conducted by the same research group

in very similar samples using the same recruitment methods. Based on these findings, those with AUD may have increased risk-taking propensity measured by the BART compared to those who have never met the criteria for AUD; however, this might only be the case for individuals still currently using alcohol. Several studies use participants currently receiving or have recently completed abstinence-oriented treatment, meaning their current alcohol use would have been no to low use (Murray *et al.*, 2015; Schmidt *et al.*, 2017; Sehrig *et al.*, 2019; Thompson *et al.*, 2012; Wang *et al.*, 2016, 2018). Since many of these studies did not report on current alcohol use for any study groups, it is possible the groups had similar current alcohol use or the control group had heavier use than those who received treatment.

Interestingly, one study by Campbell *et al.* (2013) found long-term heavy drinkers had significantly lower BART scores than light or non-drinkers. Overall, group comparisons on BART performance suggest few differences in risk-taking propensity between alcohol groups and comparison groups. Differences have only been seen between AUD and control groups. While there is currently no evidence of differences in BART performance between groups based on alcohol quantity or use of different types of substances, it may be those experiencing clinically significant consequences from alcohol also have a higher risk-taking propensity. However, lack of consistency in how studies grouped participants on alcohol quantity suggests more research is needed to make conclusions about how BART performance differs between those who drink heavily and those who do not.

Prediction of alcohol outcomes with the BART

The predictive utility of the BART is frequently conceptualized as risk-taking propensity ostensibly predicting later risk-taking behavior

including alcohol use. Thus, 15 studies investigated whether the BART may be a prospective risk factor for alcohol use and its consequences. Unsurprisingly, literature is mixed on whether BART performance predicts later alcohol outcomes. Seven studies report no significant predictive relationship. Three found that the BART was not related to alcohol use onset or escalation among adolescents (Hamilton *et al.*, 2014; MacPherson *et al.*, 2010; Worhunsky *et al.*, 2016), and another found age of onset of consequences was unrelated to the risk-taking propensity in a sample of alcohol-dependent patients (Kim *et al.*, 2018). These studies fail to provide evidence BART performance is associated with the age of onset for alcohol use or problems. This may be because the BART was designed as a state measure sensitive to changes over time; among these studies, the most proximal BART measurement was within 1 year of onset. Unfortunately, this does not tell us much about an individual's risk-taking propensity at the time of their first drink.

Three remaining studies failed to find a predictive association between the BART and alcohol use and craving (Courtney *et al.*, 2012; Clay and Parker, 2018; Padovano *et al.*, 2019). Two of these studies had small sample sizes (both $N < 40$), which could hinder their ability to detect significant effects. This is in contrast to several studies that found significant results predicting alcohol outcomes with a risk-taking propensity on the BART.

Nine studies demonstrated higher BART scores are predictive of higher alcohol outcomes. Two found BART scores predicted substance use, including alcohol use, smoking and drug use, over and above self-reported impulsivity and sensation seeking (Aklin *et al.*, 2005; Fernie *et al.*, 2010). Kim *et al.* (2018) found BART performance had a direct positive association with a latent alcohol dependence severity score. Additionally, the BART may not only predict alcohol consequences but treatment outcomes for AUD as well. While unable to directly assess craving or alcohol use, one study found risk-taking on the BART predicted treatment dropout among a sample of patients with comorbid AUD and BD (Prisciandaro *et al.*, 2011). A common symptom of BD is increased engagement in risky behaviors such as drinking or shopping sprees and among those with BD, some may have more propensity for risk-taking captured by the BART.

Two studies also suggested that BART performance is associated with changes in alcohol use over time. In one sample of early adolescents (12–13 years old), higher BART scores predicted more alcohol involvement in the next 6 months and alcohol involvement did not predict BART scores 6 months later (Fernie *et al.*, 2013). Further, increases in risk-taking over a 3-year period were associated with increased odds of using alcohol in years two and three among adolescents (MacPherson *et al.*, 2010). Based on these findings, both subsequent alcohol use and consequences may be partially explained by risk-taking propensity on the BART. This has also been demonstrated by investigating changes in craving for alcohol more acutely. Greater risk-taking on the BART was associated with more craving for alcohol over and above PTSD symptoms in a sample of veterans (Heinz *et al.*, 2016), as well as following a stress induction (Clay *et al.*, 2018) and following a priming dose of alcohol (Rose *et al.*, 2014). These findings suggest risk-taking propensity may be related to an increased craving for alcohol use; however, it is unclear whether this translates to increased alcohol use.

Only one study reported a negative association. Higher risk-taking was associated with fewer negative alcohol consequences and unrelated to alcohol use when controlling for impulsivity (Courtney *et al.*, 2012). The authors concluded the unexpected direction of the effect was because of controlling for impulsive decisions using the delayed discounting task, and suggested risk-taking itself is not

always problematic (Courtney *et al.*, 2012). Many studies include several other impulsivity-related tasks including response inhibition tasks (Lannoy *et al.*, 2017; Heinz *et al.*, 2013), the Iowa gambling task (Kelley *et al.*, 2012; Lovallo *et al.*, 2014; Schmidt *et al.*, 2017) and discounting tasks (Ashenhurst *et al.*, 2014; Courtney *et al.*, 2012; Ellingson *et al.*, 2018). Research has demonstrated that these tasks measure unique constructs and complex dynamics of impulsivity (Buelow and Blaine, 2015; Xu *et al.*, 2013). It is possible the BART captures several types of decision-making processes during the task, which are in part related to impulsive decisions, but also distinct constructs. Weafer and Fillmore (2016) provide a detailed review of low doses of alcohol on several of these tasks.

Effects of acute alcohol consumption on the BART

As risky decisions increase under the influence of alcohol (Giancola *et al.*, 2010) then risk-taking propensity may also increase after consuming alcohol. Ten studies investigated changes in BART performance after controlled administration of a set dose of alcohol. Throughout this section, all reports of breath alcohol levels, breath alcohol concentrations and blood alcohol concentrations (BAC) have been converted into an approximate BAC in grams of alcohol per deciliter of blood for consistency in reports. Despite behavioral changes in risky behavior after drinking, seven studies suggest that alcohol use does not increase risk-taking measured by the BART. A moderate dose of alcohol (BAC ~ 0.06 – 0.072%) did not impact BART performance using either the traditional BART (Peacock *et al.*, 2013) or the automatic BART (Euser *et al.*, 2011). Further, within-person differences in BART scores were non-significant at a moderate dose (Lee and Yun, 2014; Erskine-Shaw *et al.*, 2017). It is possible that these studies did not use a high enough dose to increase risk-taking, as many consequences of alcohol occur at much higher BACs.

Although the insufficient dose of alcohol is one possible explanation, existing research on dose–response relationships has not shown dose to be related to lack of impact on BART performance. For example, Reed *et al.* (2012) found no difference in BART performance between three alcohol doses (0.00, 0.056 and .092%) served on different days within the same sample of women. Reynolds *et al.* (2006) found that neither a small dose (BAC 0.02–0.04%) nor a moderate dose of alcohol (BAC 0.06–0.08%) changed risk-taking on the BART compared to placebo. Ravenzwaaij *et al.* (2011) dosed the same sample with placebo (0.00%), a small dose (0.05%) or a large dose (0.10%) of alcohol and found no difference in BART performance.

The three studies reporting increases in risk-taking propensity on the BART following alcohol administration used moderate-high doses close to 0.08%. One study found average adjusted pumps increased within-person after consuming a moderate alcohol dose (peak BAC $M = 0.088\%$) in a sample of young adults (Heinz *et al.*, 2013). Among college students and young adults, two studies found alcohol (BAC ~ 0.072 – 0.08%) compared to placebo was related to higher BART scores (Rose *et al.*, 2014; Caneto *et al.*, 2018). However, all of the dose-dependent studies used within-person procedures on different days rather than within the same day and reported no differences. Three of four studies that are used random assignment to alcohol conditions found significant results. Thus, it is possible alcohol use does impact BART risk-taking propensity at higher doses; however, systematic differences in study design impact the ability to detect effects on the BART. In the next section, we discuss several moderators that may also attenuate direct associations between the BART and alcohol outcomes.

Moderators

The following section reviews potential moderators of relations between BART performance and alcohol outcomes. Potential moderators were measured in 15 studies, as listed in Table 3. These moderators may explain some inconsistencies in the literature on BART risk-taking propensity, as it may only be related to alcohol outcomes under certain conditions.

Some studies suggest the BART captures risk-taking propensity as a state influenced by momentary moderators. Some of these moderators impact the relation between BART performance and alcohol outcomes, yet with only 15 studies including moderators, conclusions are limited. Various contexts such as environment, social context, affect and being intoxicated impacted BART performance in several studies (Corbin *et al.*, 2015; Erskine-Shaw *et al.*, 2017; Padovano *et al.*, 2019; Rose *et al.*, 2014). For women but not men, drinking alcohol in a lab environment predicted higher risk-taking propensity while drinking alcohol in a bar environment predicted lower risk-taking propensity (Corbin *et al.*, 2015). Women may feel unsafely intoxicated in certain contexts due to the increased likelihood of sexual harassment and assault. Thus, they may recruit more inhibitory control in these locations. However, further research is necessary to understand how context affects the relation between BART performance, alcohol use and consequences. For example, one study suggested that the BART predicted alcohol cravings at high levels of positive affect, yet, did not report predictions of actual alcohol use (Padovano *et al.*, 2019). Several other traits may also serve as moderators contributing to inconsistencies in the literature. Impulsive traits may attenuate relations between alcohol use and risk-taking propensity (Hamilton *et al.*, 2014; Skeel *et al.*, 2008). This may be because of a lack of sensitivity to risk-taking propensity among those high in impulsivity. Finally, how mental health moderates relations between BART performance and alcohol use depends on the diagnosis. Some symptom profiles, such as ADHD, may attenuate the influence of risk-taking propensities on alcohol use while others, such as gambling problems, increase the strength of this relation (Ledgerwood *et al.*, 2009; Weafer *et al.*, 2011). This may be related to increased impulsivity as in ADHD compared to increases in risky behaviors as in gambling and BD. Research demonstrates the need to evaluate potential moderators when studying the associations between alcohol outcomes and risk-taking propensity using the BART.

Mechanisms

Twenty-five studies evaluated several potential mechanisms of relations between BART scores and alcohol outcomes. Detailed findings of each study are listed in Table 4. Across all reviewed mechanisms, a particularly apparent pattern emerges regarding learning from outcomes during the BART. Evidence suggests both those with more alcohol consequences on average and intoxicated individuals have impaired learning for negative feedback on the BART (Ashenhurst *et al.*, 2011; Holmes *et al.*, 2009). Intoxicated individuals may also approach the task differently with greater risk-taking at first and decreasing pumps over time (Euser *et al.*, 2011). These learning differences were supported by neural activation in brain regions associated with decision-making and error-valuation. Among heavy drinking adults, greater alcohol use and consequences were associated with a smaller increase in activity in the insula and dorsal anterior cingulate cortex (ACC) during explosions compared to cash-out trials (Claus and Hutchison, 2012). Further, electroencephalography (EEG) studies suggest that reduced feedback-related negativity (FRN) responses to balloon explosions are associated with risk for AUD (Sehrig *et al.*, 2019; Soder *et al.*, 2019). Individuals with greater risk-taking

propensity may be more likely to engage in riskier decisions when they have little contextual information and may be less likely to learn from negative consequences. Some neurological studies suggest that this may be because of poor valuation of consequences and greater required cognitive control (Forster *et al.*, 2016; Yarosh *et al.*, 2014). In addition to learning from the BART, structural and connectivity differences between disordered and healthy samples show neuro correlates of BART performance including lower perfusion (i.e. blood flow) in the caudate and thalamus, reduced gray matter volume and weaker connectivity between the right cuneus and bilateral ACC (Murray *et al.*, 2015; Wang *et al.*, 2018, 2016). However, this research is limited, and researchers have yet to evaluate if these neurological markers are indeed mechanisms of the relation between BART performance and alcohol outcomes. Finally, gene X environment interactions may explain some relations between BART performance and alcohol outcomes. Specifically, dopaminergic genotypes appear to be related to both alcohol use and greater risk-taking propensity on the BART among samples of college students and adults with HIV (Barker *et al.*, 2017; Soder *et al.*, 2019). Individuals with greater risk-taking propensity may be hypersensitive to dopamine and seek rewards despite consequences. These findings were consistent with dopamine hypersensitivity being related to more bias toward immediate rewards (Mason *et al.*, 2012) and less reactivity to negative outcomes (Dagher and Robbins, 2009). Additionally, associations among family alcohol use and BART performance suggest genetic as well as environmental factors (Banducci *et al.*, 2015; Gorka *et al.*, 2015). However, it is unclear if the expressed genes are ultimately a predictor or consequence of risk-taking and alcohol use. Given the complexity of genetics, it will be some time before we understand how these factors explain the relation between BART performance and alcohol outcomes.

CONCLUSION

Overall, it is difficult to say how risk-taking propensity measured by the BART is directly related to alcohol use. There is likely no one right answer, as the relation appears to depend on several moderators reviewed: demographics, context, personality and mental health. There is also considerable variability depending on the measures capturing alcohol outcomes. However, even within this variability, some patterns have emerged. Research suggests greater risk-taking on the BART may put individuals at risk for greater alcohol use and consequences (Aklin *et al.*, 2005; Clay and Parker, 2018; Fernie *et al.*, 2010, 2013; Kim *et al.*, 2018; MacPherson *et al.*, 2010; Prisciandaro *et al.*, 2011; Rose *et al.*, 2014). Conversely, there is little evidence to suggest risk-taking on the BART increases after consuming alcohol (Caneto *et al.*, 2018; Heinz *et al.*, 2013; Rose *et al.*, 2014). Thus, the BART may be more useful for predicting changes in alcohol-related behaviors over time rather than risky behaviors one may engage in while intoxicated. Several potential mechanisms have been proposed to explain associations between risk-taking propensity and alcohol outcomes; however, many of these studies did not directly test these mechanisms as mediators. Still, differences in learning, neurological and biological markers may help us understand how risk-taking propensity influences alcohol use and consequences.

LIMITATIONS

Several limitations impede our ability to compare alcohol research studies using the BART as an indicator of risk-taking propensity. One

Table 3. Summary of studies testing potential moderators of the relations between BART and alcohol outcomes

| Article (N = 15) | Sample (size) | BART indicator | Alcohol indicator | Moderator | Results | |
|----------------------|-----------------------------------|-----------------------------|--|---|---|---|
| Demographics | | | | | | |
| 11 | Campbell <i>et al.</i> (2013) | Adults (N = 34) | Adjusted average pumps | Long-term users vs. non-users | Gender | BART performance did not differ depending on gender among either the long-term users or nonusers groups |
| 17 | Corbin <i>et al.</i> (2015) | Young adults (N = 157) | Average pumps | Alcohol (BAC 0.081%) | Gender | Women but not men differed on BART performance between alcohol and placebo conditions |
| 31 | Hamilton <i>et al.</i> (2014) | Adolescents (N = 180) | Adjusted average pumps | Drinking status drinking onset | Race | White adolescents had greater risk-taking than African American/Black adolescents. However, an interaction to predict drinking outcomes was not tested |
| Context | | | | | | |
| 17 | Corbin <i>et al.</i> (2015) | Young adults (N = 157) | Average pumps | Alcohol (BAC 0.081%) | Location | Three-way interaction such that among women but not men, risk-taking on the BART was higher for women who consumed alcohol relative to placebo in a laboratory context. However, risk-taking on the BART was lower for women who consumed alcohol relative to placebo in a simulated bar context |
| 24 | Erskine-Shaw <i>et al.</i> (2017) | College students (N = 99) | Adjusted average pumps | Alcohol (BAC 0.069%) | Social context | Being in a group rather than alone predicted significantly higher BART scores; however, there were no differences between BART scores for alcohol and placebo conditions within either social context |
| 33 | Heinz <i>et al.</i> (2013) | Young adults (N = 146) | Adjusted average pumps | Alcohol (BAC .088%) | Caffeine | Alcohol use predicted higher BART scores, yet, caffeine consumption with alcohol use did not interact to predict changes in BART performance |
| 50 | Padovano <i>et al.</i> (2019) | Adolescents (N = 29) | Number of pumps (1 trial) | Craving | Positive affect | At low levels of positive affect, greater risk-taking was not associated with craving for alcohol; however, at high levels of positive affect, greater risk-taking predicted both greater likelihood of craving and stronger cravings for alcohol |
| 52 | Peacock <i>et al.</i> (2013) | Young adults (N = 28) | Adjusted average pumps Total earnings Number of explosions | Alcohol (BAC .06%) | Caffeine | Consuming alcohol relative to placebo did not predict BART performance regardless of whether the drink was mixed with an energy drink or not |
| 59 | Rose <i>et al.</i> (2014) | College students (N = 142) | Adjusted average pumps | Craving alcohol (BAC .072%) quantity | Alcohol | BART performance while intoxicated but not after receiving placebo predicted weekly alcohol use over and above impulsivity and sensation-seeking |
| Personality | | | | | | |
| 31 | Hamilton <i>et al.</i> (2014) | Adolescents (N = 180) | Adjusted average Pumps | Drinking status drinking onset | Impulsivity | At lower levels of risk-taking, impulsivity was significantly predictive of drinking onset but was unrelated at higher levels of risk-taking |
| 62 | Skeel <i>et al.</i> (2008) | College students (N = 114) | Adjusted average pumps | Quantity | Type II personality (high novelty seeking and low harm avoidance) | At high to moderate levels of type II personality, BART performance was not significantly related to alcohol use, yet, at low levels of type II personality, higher BART scores were associated with more alcohol use |
| Mental Health | | | | | | |
| 12 | Caneto <i>et al.</i> (2018) | Young adults (N = 51) | Adjusted average pumps | Alcohol (BAC .08%) | Family History | Those with a family history of alcohol use disorder increased their BART average adjusted pumps compared to placebo; however, those with no family history of alcohol use disorder showed no difference between alcohol and placebo conditions |
| 22 | Dougherty <i>et al.</i> (2015) | Early adolescents (N = 386) | Adjusted average pumps | Family history status | Family history | Early adolescents increased their adjusted mean pumps on the BART from age 10 to 15; however, family history of alcoholism did not moderate this change in risk-taking |
| 32 | Hawn <i>et al.</i> (2019) | US Veterans (N = 302) | Adjusted average pumps | Quantity frequency of binge drinking | PTSD | PTSD symptoms in a sample of veterans were not found to moderate the relation between risk-taking on the BART and higher alcohol use |
| 38 | Kelley <i>et al.</i> (2012) | US Veterans (N = 262) | Adjusted average Pumps | Quantity Frequency desire to cut down Impaired control | PTSD | Those diagnosed with either PTSD or PTSD and a traumatic brain injury had increased alcohol use post-deployment compared to those only diagnosed with a traumatic brain injury and healthy controls. However, BART adjusted average pumps were not found to interact with diagnosis to predict post-deployment drinking |
| 41 | Ledgerwood <i>et al.</i> (2009) | Adults (N = 102) | Adjusted average pumps | SUD + gambling vs. control | Gambling | Those with gambling problems and a history of substance use disorders (including alcohol) had higher adjusted average pumps than gamblers without a history of SUDs. However, both gambling groups were not significantly different than the control group, who had BART scores mid-way between the two gambling groups |
| 67 | Weafer <i>et al.</i> (2011) | Young adults (N = 54) | Number of pumps | Quantity frequency | ADHD | Greater BART scores were associated with more frequent alcohol use but only for young adults without ADHD |

Notes: Significant moderators are in bold. Δ = change.

Table 4. Summary of studies testing potential mechanisms

| Article (N = 25) | Sample (size) | BART indicator | Alcohol indicator | Mechanism | Results | |
|---------------------|--------------------------------|---|--|--|--------------------------------------|--|
| Learning | | | | | | |
| 4 | Ashenurst <i>et al.</i> (2011) | Adult heavy drinkers (N = 198) | Adjusted average pumps Variability of pumps Post-failure mean Pumps | AUD symptoms Frequency Binge frequency Typical quantity | Learning | Participants with greater severity of alcohol consequences decreased their number of pumps after explosions relative to successful trials more than those reporting less severity in consequences. The magnitude (measured by a number of pumps) of the explosion also impacted pumps on the following trial. Those 1.2 standard deviations above the mean on severity did not change their responses after large magnitude explosions, while those reporting less severity of consequences increased pumps after this type of trial. Alcohol consequences severity did not moderate responsiveness to any other type of trial |
| 25 | Euser <i>et al.</i> (2011) | Young adult males (N = 64) | Average pumps | Alcohol (BAC ~ 0.072%) | Learning | Block analysis of 20 balloons per block on the BART revealed the alcohol group significantly decreased their number of pumps between the first and second block, then had no significant change to block three. In contrast, the placebo group started with a lower number of pumps than the alcohol group on the first block and increased their number of pumps across the three blocks. Thus, intoxicated and sober individuals approached and learned from the task differently |
| 35 | Holmes <i>et al.</i> (2009) | Adults with and without bipolar disorder (N = 80) | Adjusted average pumps | BD + AUD vs. control BD + AUD vs. BD | Learning | When evaluating pumps on trials following either an exploded or successful balloon, those diagnosed with comorbid bipolar disorder and AUD did not change their behavior based on the outcome of the previous balloon. In contrast, those diagnosed with only bipolar disorder and the healthy control group decreased their number of pumps on trials immediately after exploded balloons |
| Neurological | | | | | | |
| 9 | Bogg <i>et al.</i> (2012) | College students (N = 27) | Total pumps Average pumps Number of explosions Number of cash-outs Amount earned | Typical quantity | fMRI connectivity during BART | Looking at fMRI BOLD response during a short delay after the decision to inflate the balloon, college students who showed greater signal decrease as the probability of explosion increased in the mPFC/ACC reported more typical weekly alcohol use. Additionally, those who showed greater signal increases as the probability of explosion increased in the mPFC/ACC during successful trials reported more typical weekly alcohol use. This may suggest not only down-regulation of reactivity to increased risky choices, but that increased reactivity to the reinforcement of positive outcomes may put individuals at risk for increased alcohol use |
| 13 | Claus <i>et al.</i> (2018) | Adolescents (N = 189) | Adjusted average pumps Proportion of explosions | Alcohol vs. MJ vs. Alcohol + MJ vs. control | fMRI activity during BART | Larger responses in the ventral ACC, bilateral orbitofrontal cortex/insula, bilateral inferior frontal gyrus and precentral gyrus all correlated with fewer adjusted average pumps on the BART. These associations were not different between substance use groups. However, this BART version had limited variability and only allowed a maximum of 11 pumps |
| 14 | Claus and Hutchinson (2012) | Adult heavy drinkers (N = 79) | Adjusted average pumps Proportion of explosions | AUDIT | fMRI activity during BART | Among heavy drinking adults, higher scores on the AUDIT were associated with less increase in activity in the insula and dorsal ACC during explosions compared to cash-out trials. The insula has been found to represent outcomes from decisions and dACC often represents error likelihood. Together, these areas may suggest individuals with greater alcohol use and consequences have a weaker learning response to negative feedback |
| 25 | Euser <i>et al.</i> (2011) | Young adult Males (N = 64) | Average pumps | Alcohol (BAC ~ 0.072%) | EEG—FRN & P3 amplitude | P3 and FRN event-related potentials were larger for explosions on the BART than cash-out balloons during the automatic BART. However, responses did not differ between students who were served alcohol and students who received placebo |
| 29 | Forster <i>et al.</i> (2016) | Alcohol or substance dependent adults (N = 21) | Adjusted average pumps | Frequency | fMRI BOLD—ACC & vmPFC | Those who reported less substance 3 months post-treatment use had decreased response in the dorsal ACC and vmPFC during pumps at follow-up compared to those reporting more substance use. Further, the decreased response in vmPFC was correlated with less risk-taking on the BART |
| 40 | Lannoy <i>et al.</i> (2017) | College students (N = 40) | Adjusted average pumps Number of explosions Pumps before explosions Total pumps | Binge vs. non-binge drinkers | EEG—FRN & P3 Amplitude | Neither P3 nor FRN response during the BART differed significantly between groups of those who binge drank and those who never binge drink |
| 48 | Murray <i>et al.</i> (2015) | Adults (N = 77) | Adjusted average pumps | Alcohol vs. poly use alcohol/poly vs. control | MRI—Thalamus & Caudate | Among the alcohol use disorder group but not the light drinking group, lower perfusion (i.e. regional blood flow) in cortical regions was associated with higher BART scores. The regions of interest in this study were a neural network involved in the development and maintenance of addiction including the thalamus and caudate |

(Continued)

Table 4. Continued

| Article (N = 25) | Sample (size) | BART indicator | Alcohol indicator | Mechanism | Results | |
|--------------------------|-------------------------------|-----------------------------------|---------------------------------------|---|-----------------------------|--|
| 49 | Neal and Gable (2019) | College students (N = 44) | Number of pumps | Alcohol cues vs. neutral cues | EEG—left frontal activation | Participants demonstrated more left frontal activation on balloons with alcohol cues compared to balloons with neutral cues. The authors concluded approach motivation was being enhanced by the alcohol cues. Further, a three-way interaction was found such that on alcohol trials specifically, participants had greater activation for the last half of trials where the balloon exploded compared to the first half of explosions and all successful trials. This could suggest activation of approach motivation occurs after repeated exposure to cues |
| 55 | Qu <i>et al.</i> (2015) | Adolescents (N = 24) | ΔAdjusted Average Pumps | ΔRule breaking (including alcohol use) | fMRI—mPFC/VS | Over 1.5 years, adolescents who had larger reductions in risk-taking on the BART also had larger declines in the coupling between the medial prefrontal cortex and the ventral striatum. Adolescents who also self-reported less risk-taking behaviors, including alcohol use, over time also demonstrated less reactivity in the ventral lateral prefrontal cortex and the ventral striatum. These areas are involved in cognitive control, decision-making, and reward-seeking behavior |
| 61 | Schrig <i>et al.</i> (2019) | Adults (N = 74) | Average pumps Number of explosions | AUD vs. control | EEG—FRN & P3 Amplitude | Healthy individuals had greater FRN responses after explosions than those diagnosed with alcohol use disorder. Further, larger differences between P3 reactivity between high-risk (50 balloons per participant with the most pumps) and low risk (50 balloons per participant with three pumps) trials on the BART predicted more risky choices among those diagnosed with alcohol use disorder. There were no relations between P3 reactivity and performance on the BART for healthy controls |
| 63 | Soder <i>et al.</i> (2019) | College students (N = 85) | Number of pumps | Quantity | EEG—Activation | Individuals with greater risk for alcohol use disorder based on high sensitivity to dopamine had weaker EEG responses to explosions than those with average to low sensitivity to dopamine |
| 65 | Wang <i>et al.</i> (2018) | Male adults (N = 81) | Adjusted average pumps | Relapsed vs. no relapse No relapse vs. control | MRI—cuneus/ACC | Higher BART scores were associated with weaker negative connectivity between the right cuneus and bilateral ACC among those who relapsed 3 months post-treatment. Abstainers had no significant correlations between neural connectivity and BART performance. The authors concluded these findings suggest those who relapsed need to recruit greater neurological networks to maintain impulse control |
| 66 | Wang <i>et al.</i> (2016) | Male adults (N = 40) | Adjusted average pumps | AUD vs. control | MRI—gray matter volume | Adult males diagnosed with alcohol dependence had significantly less gray matter volume in the left medial prefrontal cortex, which is involved in cognitive control, than a healthy control group. Further, less gray matter volume in the medial prefrontal cortex was associated with greater risk-taking on the BART after controlling for typical alcohol use per day |
| 69 | Yarosh <i>et al.</i> (2014) | Adults (N = 69) | Risk-taking (latent) | Family history of AUD | fMRI—ventral caudate | Higher risk-taking measured by the BART was associated with increased activity in the right ventral caudate in response to rewards compared to punishments on a decision-making task. This area is associated with cognitive control and reward circuitry. The authors posited this difference was indicative of atypical development in mesocorticolimbic circuitry. However, this activation was unrelated to the family history of alcohol use disorders and no direct alcohol use measures were used in the study |
| Bio & Genetic | | | | | | |
| 3 | Andrews <i>et al.</i> (2011) | Adults (N = 49) | Adjusted average pumps | Family history status | Family history | BART adjusted average pumps did not differ for those with and without a family history of alcohol use disorders. |
| 7 | Banducci <i>et al.</i> (2015) | Mother–Child Dyads (N = 277) | Adjusted average pumps | Quantity | Mother-Child Association | Higher average adjusted pumps on the BART completed by mothers was unrelated to their child's risk-taking cross-sectionally; however, risk-taking propensity for mothers predicted increases in their child's alcohol use from 8th to 10th grade |
| 8 | Barker <i>et al.</i> (2017) | HIV-infected Adults (N = 201) | Adjusted average pumps | AUDIT | DAT1 genotypes | Those with the DAT1*10R genotype were both higher in hazardous drinking measured by the AUDIT and higher adjusted average pumps on the BART compared to those with the DAT1*9R genotype. The DAT1 gene is involved in coding the dopamine active transporter 1 and the DAT1*10R genotype is associated with greater risk-taking compared to the DAT1*9R genotype (Guo <i>et al.</i> , 2010; Mata <i>et al.</i> , 2012) |
| 26 | Fein and Chang (2008) | Alcohol-dependent adults (N = 22) | Adjusted average pumps | Quantity Family history of AUD | Family history | Those with greater family history density of alcohol problems (number of first degree relatives with an AUD) had weaker FRN measured by EEG during the BART task. However, FRN was unrelated to drinking history after controlling for age |

(Continued)

Table 4. Continued

| Article (N = 25) | Sample (size) | BART indicator | Alcohol indicator | Mechanism | Results | |
|------------------|---------------------------------|--|-------------------------|--|--|---|
| 30 | <i>Gorka et al. (2015)</i> | Adult sibling pairs (N = 87 pairs) | Average pumps | Quantity Frequency of binge drinking | Sibling Association | The sibling who was a heavier drinker (e.g. the proband) was used to predict the risk-taking propensity of the lighter drinking sibling. Both the proband's alcohol use quantity and frequency of binge drinking predicted their sibling's average pumps on the automatic BART, while the sibling's drinking outcomes were not significant predictors. The proband's binge drinking but not alcohol use quantity was related to their own average pumps on the BART |
| | | Adolescents & mothers (N = 111 pairs) | Average pumps | AUDIT | Mother-Child Association | Mother's scores on the AUDIT were unrelated to their own risk-taking propensity but predicted higher average pumps among their children. Further, the child's score on the substance problems scale was not significantly related to their average pumps |
| 37 | <i>Kalapatapu et al. (2013)</i> | US Veterans with PTSD + AUD (N = 30) | Adjusted average pumps | Quantity | Biomarkers | Of all the direct and indirect biomarkers evaluated, only mean corpuscular volume, an indirect biomarker of alcohol use, was associated with adjusted average pumps on the BART. However, after adding mental-health-related covariates (depression, PTSD, and prescription medications) the association became non-significant. |
| 44 | <i>Lovallo et al. (2014)</i> | Young adults (N = 314) | Adjusted average pumps | AUDIT Family history of AUD | Family history Serotonin genotypes | Risk-taking on the BART did not differ based on either family history status or serotonin genotype, and there was no interaction for risk. However, the authors questioned whether the serotonin transporter activity was a direct risk factor for alcohol use, as evidence suggested the combination of having a positive family history and high serotonin activity genotype only puts individuals at an indirect risk due to elevated traits correlated with alcohol use including depression, neuroticism, harm avoidance and sensitivity to loss |
| 51 | <i>Park et al. (2020)</i> | Adults (N = 638) | Adjusted average pumps | AUD severity | Opioid genotypes | The authors found limited evidence that any opioid genotypes were associated with alcohol use disorder or severity. Further, adjusted average pumps on the BART were unrelated to all opioid genes |
| 53 | <i>Pennington et al. (2014)</i> | US Male Veterans with AUD (N = 10) | Adjusted average pumps* | Quantity AUDIT | MRI— metabolites of alcohol | Veterans completed resting MRI scans to evaluate regional brain metabolites in those with PTSD, with PTSD and AUD and healthy controls. Despite differences seen in alcohol metabolites (i.e. NAA, Cho, ml, Glu and GABA), none of these metabolites were associated with risk-taking on the BART |
| 63 | <i>Soder et al. (2019)</i> | College students (N = 85) | Number of pumps | Quantity | Dopamine Genotypes | College students were classified as high dopamine reactivity or low dopamine reactivity based on associated gene variants. Those in the high dopamine group (i.e. hypersensitivity) had more typical weekly drinking measured by the daily drinking questionnaire than those in the low dopamine group. BART performance during EEG revealed those in the high dopamine group had less neurological reactivity to balloon explosions than those in the low dopamine group. |
| 69 | <i>Yarosh et al. (2014)</i> | Adults (N = 69) | Risk-taking (latent) | Family history of AUD | Family History | A latent risk-taking variable indicated by the BART did not differ between those with a history of AUD and those without any family history of AUD |

Notes: Significant results are in bold. n.s. = not significant, AUDIT = alcohol use disorders identification test, ADQ = alcohol disorders questionnaire, MRI = magnetic resonance imaging, VS = ventral striatum, vmPFC = ventral medial prefrontal cortex.

barrier is the number of versions of the BART task. Various versions of the BART have few direct comparisons in the literature. A key component of the original BART task is actively making increasing risky decisions with each pump; however, the automatic BART requires only one decision before each balloon and participants watch their decision play out. While *Pleskac et al. (2008)* reported no difference in behavior between these two tasks when using adjusted average pumps, it is still unclear whether they measure the same risk-taking decision. Additionally, many studies modified the BART to be a daily measure with only one trial or reduced the number of maximum pumps for neuroimaging purposes. While these modifications are necessary to test the research questions, it is unclear how comparable different versions of the BART are.

Additionally, the flexibility of the BART includes the ability to change the payment structure for each pump, which has ranged from a fraction of a cent (0.003 cents) to larger rewards (\$0.25). Some

studies chose to not pay participants for their performance on the BART. However, payment for participation has been found to impact risk-taking on the BART (*Ferrey and Mishra, 2014*). Thus, this may account for some inconsistencies in results across studies. Finally, regarding alcohol use, no studies directly tested age as a moderator. Adolescence is a period of heightened risk-taking behavior (*Arnett, 1999; Lejuez et al., 2002*), where risk-taking propensity may be more proximal to actual behavior. Longitudinal analyses suggest that developmental changes in risk-taking propensity during adolescence may relate to developmental changes in alcohol use (*Qu et al., 2015*). However, no studies have investigated development into adulthood or across the development of AUD. It is possible that the development of risk-taking propensity rather than one assessment will tell us more about how alcohol use changes throughout one's lifetime. This may explain some null results for cross-sectional associations between BART scores and alcohol outcomes.

To adequately understand the development of risk-taking propensity, it is imperative to understand if the BART is sensitive to within-person changes. While the BART was intended as a state-like measure, research suggests the BART is largely stable over time with test-retest reliabilities >0.75 (King *et al.*, 2014). However, recent work using ecological momentary assessment (EMA) suggests modest BART score reliabilities (ICCs = 0.57–0.76) and scores change across time and context (MacLean *et al.*, 2018). One study reviewed found an interaction between daily effect and BART performance predicted craving for alcohol (Padovano *et al.*, 2019). These two studies suggest the BART does capture within-person changes, yet, the sensitivity of the BART to contextual influences is relatively understudied. In contrast, many experimental studies failed to find differences in BART scores within-individuals at least after consuming alcohol (Erskine-Shaw *et al.*, 2017; Euser *et al.*, 2011; Lee and Yun, 2014; Peacock *et al.*, 2013; Ravenzwaaij *et al.*, 2011; Reed *et al.*, 2012; Reynolds *et al.*, 2006). However, experimental studies may have inadequately accounted for the contextual influences and individual differences on the BART, limiting their ability to detect differences after consuming alcohol. Location, social context, affect, and personality all seem to impact BART performance, which was not accounted for, and in some cases, could have changed between days of assessment.

Inconsistencies in measurement and methods may account for discrepancies in the literature. Across these 69 studies, there was significant variability in how alcohol outcomes were defined. Of the four studies on the family history of AUD, there were three different definitions of family history. Alcohol use, consequences and craving were each measured with several different methods as well. Some studies have attempted to use latent variables with indicators from multiple alcohol measures to model commonalities between these measures (Fernie *et al.*, 2010, 2013; Kim *et al.*, 2018). However, these more complex models require significantly larger sample sizes, which may not be feasible for all alcohol research.

Finally, this review is limited to only published peer-reviewed journal articles using both the BART and alcohol use, consequence or craving measures. Due to biases in publication and peer review processes, there may be unpublished studies not contributing to our understanding of the BART. Further, there may be additional journal collections not searched in this review which would reveal additional alcohol research using the BART. This review represents an initial overview of associations between the BART and alcohol outcomes to provide direction for future research, which will further contribute to our understanding of risky alcohol use.

Future directions

Overall, literature on the BART and alcohol outcomes is quite complex and difficult to reconcile based on many factors, which presents many opportunities for future research. More research is needed to investigate differences in the use of the BART measure. This systematic review presents a broad first overview of the current literature; however, some of these studies along with ongoing work may be grouped into more specific PICO parameters where meta-analyses may be conducted to determine the magnitude of any effects. Future research may benefit from a standardized measure of the BART. The BART appears to be most useful when investigating patterns of responding rather than simply using adjusted average pump scores across all balloons. More research is needed to understand how alcohol outcomes and risk-taking propensity are related through responsiveness within the BART trials. Based on current literature,

responses to negative consequences (i.e. explosions) seem to be important in delineating how alcohol is related to BART performance. Additionally, future research should continue to explore moderators, such as age, to determine when and for whom risk-taking propensity captured by the BART is related to alcohol outcomes. Many potential moderators have yet to be explored thoroughly, yet it is clear that BART scores are not consistently related to alcohol outcomes. Finally, several potential mechanisms have yet to be investigated as mediators of the relation between BART performance and alcohol outcomes. Substantial research is needed to confirm whether learning effects, neurological and genetic markers are in fact mechanisms. Alcohol research using the BART is only just beginning and this review may provide direction as this research continues to improve our understanding of risk-taking propensity and alcohol outcomes.

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DATA AVAILABILITY

No new data were generated or analyzed in support of this research.

CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

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