INTRODUCTION

With the increasing availability of online streaming platforms, their large repertoire of shows, and policy to release entire seasons at once, binge-watching (BW)—subsequently watching multiple episodes (Trouleau et al., 2016; Walton-Pattison et al., 2018)—attracts growing theoretical and empirical interest (Schlütz, 2015). BW is a popular and rewarding leisure activity that should not be unnecessarily pathologized (Billieux et al., 2015). However, BW does entail problematic potential as it is linked to negative outcomes such as sleeping...
problems (Exelmans & Van den Bulck, 2017), malnutrition and a sedentary lifestyle (Vaterlaus et al., 2019), social withdrawal (Rubenking et al., 2018), and reduced wellbeing (de Feijter et al., 2016; Sung et al., 2015) as well as psychopathological features associated with negative affect (e.g., depression) and addiction (Ahmed, 2017; Conlin et al., 2016; Riddle et al., 2018; Tefertiller & Maxwell, 2018; Tukachinsky & Eyal, 2018; for a review see Flayelle, Maurage, et al., 2020). As such, research illuminating the mechanisms facilitating BW is warranted.

Models converge on the notion that BW should not per se be regarded as a unitary construct with negative implications (Flayelle, Maurage, et al., 2020). Indeed, BW often reflects a deliberate and controlled activity to meaningfully serve hedonistic purposes (e.g., narrative immersion, relaxation). However, more problematic BW, characterized by a lack of control over watching and social impairments, tends to be driven by compensatory tendencies (e.g., overcome boredom, loneliness, or sadness) or depression (Ort et al., 2021; Tukachinsky & Eyal, 2018), and associated with low self-control, characterized by impulsivity, automaticity, and need for immediate gratification (Riddle et al., 2018; Rubenking et al., 2018; Shim et al., 2018; Walton-Pattison et al., 2018). In fact, impulsivity and depression may be independently contribute to loss of control over watching (Steins-Loebet al., 2020). Poor self-regulation may contribute to reduced wellbeing when BW conflicts with long-term goals (Granow et al., 2018). This is particularly the case where such goal conflicts induce post-show regret or guilt. These, in turn, may trigger continued watching as a maladaptive coping mechanism (Panda & Pandey, 2017; Shim et al., 2018), which implies impaired integration of outcomes, such as the consequences of watching and neglected goals. Some authors attribute addictive properties to binge watching (Panda & Pandey, 2017; Riddle et al., 2018; Walton-Pattison et al., 2018), but this claim is yet awaiting substantiation (Flayelle, Verbruggen, et al., 2020). In sum, emerging features of BW potentially include high sensitivity to its rewarding properties; impaired regulation, particularly inhibition, of positive (deepen engagement, suspense, etc.) or negative (avoid sadness, guilt, etc.) urges; deficient valuation and insensitivity regarding consequences of behavior that should inform the decision-making process to stop or continue watching. This suggests that inhibitory and feedback-related functions are worthy subjects of study to improve our understanding of BW.

The electroencephalography (EEG) method is well suited to examine these constructs, but EEG investigations of BW are scarce (Kilian et al., 2020). Particularly uncontrolled BW is linked to problematic internet and alcohol use (Flayelle et al., 2017, 2019) and impulsivity is a well-established correlate of excessive internet use, gambling, and substance use (Chowdhury et al., 2017; Lee et al., 2019; Verdejo-Garcia et al., 2008), suggesting a potential overlap in underlying risk factors. As such, it can be useful to consider the broader EEG literature on inhibition and feedback processing in depression and excessive and addictive behaviors to inform hypotheses regarding BW.

In the feedback-locked EEG, the frontocentral feedback-related negativity (FRN; also termed reward positivity) appears ~250 ms after feedback with more negative amplitudes for outcomes worse than expected (Sambrook & Goslin, 2015). The P3 (300–500 ms) encompasses the frontocentral feedback-P3a, indexing attentional orientation (Polich, 2007), and the parietal feedback-P3b, indexing expectancy violations and feedback salience (Bellebaum & Daum, 2008; Mars et al., 2008; Yeung & Sanfey, 2004). In addicted and high-risk individuals, these event-related potentials (ERPs) are sensitive to addiction-related rewards (Baker et al., 2016, 2017; Littel et al., 2012). In contrast, and relevant to the current investigation, converging ERP evidence suggests diminished responding to performance feedback, that is, points won or lost converted to monetary reinforcement, in individuals with excessive behaviors and addiction risk, such as problematic gambling, internet use, or a family history of substance use disorders (SUD; Euser et al., 2013; Lole et al., 2015; Yau et al., 2015). This is consistent with reduced sensitivity for alternative reinforcers. Such blunting may also prospectively predict problematic substance use, particularly when interacting with inhibitory deficits (Büchel et al., 2017; Joyner et al., 2019; Soder et al., 2019). Similarly, depressive symptoms are linked to diminished ERP responses to feedback (Klawohn et al., 2020; Nelson et al., 2016; Oumeziane & Foti, 2016).

The go/nogo task (GNGT) measures inhibition—suppressing a motor response before it is initiated—and the stop-signal task (SST) assesses stopping—canceling an already initiated response. While similar conceptually and regarding their electrophysiological signature, inhibition and stopping appear to be distinct processes. They involve different neurotransmitter systems, patterns of muscular activity, and temporal dynamics in the allocation of cognitive control, and are differently affected by distraction (Eagle et al., 2008; Littman & Takacs, 2017; Raud et al., 2020), indicating that investigating both inhibition and stopping is warranted. A frontocentral N2 at ~250 ms after nogo and stop signals indicates conflict detection and a subsequent central P3 (300–500 ms) is associated with implementing inhibition or stopping (Enriquez-Geppert et al., 2010; Folstein & Van Petten, 2008; Kok et al., 2004; Ramautar et al., 2004; Waller et al., 2019; Wessel & Aron, 2015). ERP studies suggest deficient inhibition in substance-related and behavioral addiction (Colrain et al., 2011; Littel, van den Berg, et al., 2012) and such impairments predict relapse (Luijten et al., 2016). However, the overall pattern regarding such alterations is inconclusive in substance use disorders (see Luijten et al., 2014 for a review), online gaming disorder (Kim et al., 2017; Li...
et al., 2020), as well as excessive internet (Dong et al., 2010; see D’Hondt et al., 2015 for a review) and smartphone (Chen et al., 2016; Gao et al., 2019) use. A similarly ambivalent picture emerges regarding inhibition- and stopping-related ERPs in depression (Cavanagh et al., 2019; Kaiser et al., 2003; Palmwood et al., 2017; Ruchsw et al., 2008).

In addition to these mixed inhibition results in the EEG domain, prior work failed to show behavioral deficits in inhibitory performance in high binge-watchers (Flayelle, Verbruggen, et al., 2020). This should discourage examining group differences (e.g., in ERP amplitudes) in isolation. Instead, a promising avenue to better understand BW could involve investigating the relationship between inhibitory function and sensitivity to action outcomes. For instance, at the juncture between episodes, the urge to continue collides with its potential negative consequences. For this decision, both inhibitory control as well as integrating the potential negative consequences of continued watching and benefits of alternatives are crucial, such that the interaction of both domains may be particularly relevant. Interestingly, models of problematic internet use (Brand et al., 2019) and addiction (Zilverstand et al., 2018) make similar claims. Their core assumption is an imbalance, such that low capacity or motivation to inhibit approach tendencies interacts with high motivation for the object of desire, while discounting negative consequences and alternative reinforcers. Indeed, the approach to investigate the relationship between inhibition and reward or outcome sensitivity in addiction has proven particularly useful in the past, for example, in first-degree relatives of patients with substance-use disorders (Volkow et al., 2006) or to predict SUD symptoms (Joyner et al., 2019) and drug sensitivity (Weafer et al., 2017, 2020). Moreover, a recent neuroimaging study found an inverse relationship between altered feedback-related and inhibitory brain activity in high but not in low binge drinkers (Weafer et al., 2019). Whereas binge-drinkers may be at high risk of developing alcohol use disorder (Gowin et al., 2017), this relationship within high binge-drinkers was observed in the absence of group differences in feedback-related or inhibitory brain function (Weafer et al., 2019). We thus adopted this apparently risk-sensitive, multi-outcome approach.

The current study aimed to investigate the relationship between outcome processing and inhibition in high binge-watching individuals. We recorded EEG in high (HBW) and non-binge-watchers (NBW) during performance feedback in a modified flanker task to capture outcome processing, during a GNGT to assess inhibitory activity, and during a SST to measure brain activity associated with stopping. Participants also reported a subjective loss of control over watching. We hypothesized that within HBW but not NBW, lower inhibitory and stopping-related brain activity was associated with reduced discrimination between positive and negative performance feedback (Hypothesis 1). We also formulated two more exploratory hypotheses. If outcome (in)sensitivity was specifically related to altered implementations of the inhibition or stopping process, the P3 as a marker of successful inhibition/stopping should show this association, rather than the N2 given its role in conflict detection prior to inhibition (Hypothesis 2). Further, if these relationships were relevant to potentially problematic BW, associations between neural activity should be most prominent in HBW reporting a high loss of control over watching (Hypothesis 3).

We used single-trial regression-based EEG analyses (Fischer et al., 2016; Fischer & Ullsperger, 2013), which examine activity at each electrode and time point and therefore entail several benefits. Employing general linear models (GLMs) to predict neural activity from task parameters closely resembles the analytic approach of relevant functional magnetic resonance imaging work motivating the present investigation (Weafer et al., 2019). Their data-driven nature is agnostic as to which components should be selected to examine task effects at the individual (first) level. Task effects can also be isolated by controlling for single-trial sources of noise. Importantly, the high temporal resolution of EEG is retained at the group (second) level. This enabled us to elucidate in a fine-grained manner which electrodes and time points—that is, which subprocesses of the outcome processing and inhibition/stopping domains—specifically showed a relationship (serving Hypothesis 2).

2 | METHOD

2.1 | Sample

Thirty-five HBW and 33 NBW were recruited via local advertisement and screened via telephone interview. Note that we previously published data from this sample with a focus on error processing (Kilian et al., 2020). Exclusion criteria were neurological disorders, head trauma, intellectual disability, lifetime borderline personality disorder, schizophrenia, psychotic disorder and/or manic episodes; current eating disorder, severe depressive episode, and previous or current daily alcohol use, cannabis use more than twice per month, and use of other illegal drugs more than twice per year. A BW event was defined as watching more than three episodes in one session. HBW versus NBW reported at least 10 versus no BW events in the past 4 weeks. We excluded $n = 2$ HBW and $n = 1$ NBW from analyses because they had less than two valid blocks in the SST (see 2.2 Tasks and procedure, stop-signal task), $n = 1$ NBW who did not finish the monetary flanker incentive delay task, and $n = 1$ HBW due to insufficient performance in the monetary incentive delay task (>38% errors). This left a final sample with $n = 32$ HBW and $n = 31$ NBW (Table 1). When we conducted this study, no validated measure of problematic BW was available in
German. To nevertheless record loss of control over watching, we adopted items from the Eating Loss of Control Scale (ELOCS; Blomquist et al., 2014). Participants rated ten items (listed in the Supplementary Materials) on a five-point scale (never, rarely, sometimes, frequently, always; scored as 0 to 4) pertaining to watching behavior (ability or willingness to stop, negative feelings involved, etc.). We used the sum score to indicate loss of control over watching (LOCW). The instrument had an internal consistency of $\alpha = .89$. Impulsivity was assessed with the Barratt-Impulsiveness-Scale (BIS-11; Patton et al., 1995) and anxiety and depressive symptoms with the depression anxiety stress scales (DASS; Henry & Crawford, 2005). The study was conducted in accordance with the Declaration of Helsinki and approved by the TU Dresden ethics committee (EK 465112018). Participants gave written informed consent and received monetary compensation.

### 2.2 | Tasks and procedure

Participants completed questionnaires and then performed three tasks during EEG in a balanced order.

#### 2.2.1 | Go/nogo task

A white circle on a gray background remained empty for 200–500 ms (see Figure 1). Then, a green square in its center served as a go stimulus (75% of trials), asking to respond as...
quickly as possible with the right index finger; a red square served as a nogo stimulus (25% of trials), asking to withhold a response. Stimuli were presented for 500 ms and followed by inter-trial intervals (400–1,000 ms). Up to five go trials separated a maximum of two subsequent nogo trials. Participants performed two blocks with 128 trials.

2.2.2 | Stop-signal task

A white circle on a gray background remained empty for 300–500 ms. Then a green right- or left-pointing arrow appeared in its center for 1,000 ms, asking to respond according to its direction as quickly and accurately as possible with the index fingers. On stop trials (25%), the arrow turned red after a certain interval (stop signal delay, SSD), asking to cancel the already initiated response. SSDs increased or decreased by 50 ms (lower and upper limits: 50 and 350 ms) after successful or failed inhibitions, respectively. The task started with four SSDs (100, 150, 200, and 250 ms), each active on 25% of stop trials and tracked by its own adaptation algorithm (Aron & Poldrack, 2006). Go and stop trials were presented in a pseudorandom order with the following constraints: The first three trials were going trials, left and right arrows occurred equally often within go and stop trials, stop trials and arrow direction did not repeat more than twice. The inter-trial interval was 400–800 ms. The task stopped after two valid blocks (stopping accuracies between 40% and 60%) or a maximum of four blocks (128 trials each). Invalid blocks were not analyzed and all participants included in the analyses completed at least two valid blocks (n = 3 were excluded with < two valid blocks, see 2.1 Sample).

2.2.3 | Monetary incentive flanker task

This modified flanker task (Endrass et al., 2010) involved two motivational contexts: A green or red frame (500 ms),
respectively, indicating a gain context or loss avoidance context (50% of trials each), surrounded a fixation cross. Four vertical arrows replaced the cross and after 100 ms the target arrow was presented in their center for 30 ms. Arrow direction was compatible on half of the trials and incompatible (target and flanker arrows pointed in opposite directions) in the remaining trials. Context, compatibility, and target direction were presented in pseudo-random order and balanced between conditions. Participants indicated the direction of the target arrow with their left and right index fingers as quickly and accurately as possible. Performance feedback (800 ms) occurred 900 ms following target onset or 600 ms after the response. An adaptive response deadline was calculated based on performance and reaction time in order to obtain a rate of 20% negative feedbacks after correct responses for each context. In the gain condition, fast (below the deadline) and correct responses were rewarded (40 points; happy green emoji) and errors and slow responses resulted in reward omission (0 points; sad red emoji; regret trials). In the loss avoidance condition, errors and slow (exceeding the deadline) responses were punished (minus 40 points; sad red emoji) and fast and correct responses resulted in punishment omission (0 points; happy green emoji; relief trials). Participants could earn a bonus of up to 5 EUR. The task included 640 trials. This modified flanker task was chosen over traditional monetary incentive paradigms to also examine performance monitoring as a function of motivational context, as reported elsewhere (Kilian et al., 2020).

2.3 | Electroencephalogram recording and data reduction

The EEG was recorded at 500 Hz with 64 Ag/AgCl electrodes using two 32-channel BrainAmp amplifiers (Brain Products GmbH, Munich, Germany) and an EasyCap electrode cap with equidistant electrode locations (EasyCap GmbH, Herrsching-Breitbrunn, Germany). Impedances were below 10 kOhm. We placed reference and ground electrodes next to Fz and two external electrodes below the left and right eye.

Data were analyzed using EEGLAB 14.1 (Delorme & Makeig, 2004). We applied a common average reference and filter between 0.5 and 30 Hz before creating epochs from 200 ms before and 800 ms after go stimulus (GNGT and SST) and feedback (Monetary incentive flanker task [MIFLAT]) onset. We excluded trials with early (<80 ms in SST; <100 ms in GNGT; <150 ms in MIFLAT) or late responses (>600 ms) from behavioral and EEG analyses and go trials with omitted responses (SST and GNGT), go trials with erroneous responses (SST), and error, relief, and regret trials (MIFLAT) from EEG analyses. We removed epochs containing improbable data relative to the activity of all electrodes and epochs. Epochs containing deviations >5 SDs of the mean probability distribution were automatically excluded with the constraint to reject no more than 5% of trials or to otherwise increase the rejection threshold (Fischer & Ullsperger, 2013). We removed ocular and cardiac artifacts with independent component analysis. The 200 ms pre-stimulus windows served as a baseline.

2.4 | Electroencephalogram analyses

EEG analyses were conducted in two steps. First-level analyses did not directly serve hypothesis testing, but helped establish when and where EEG activity was sensitive to task effects, that is, the neural signatures of feedback valence, stopping, and inhibition. First-level results were then used for second-level analyses. Here, we tested Hypothesis 1 that brain activity associated with feedback and inhibition/stopping were associated in HBW but not NBW; Hypothesis 2 that these relationships were specific to implementing inhibition/stopping, that is, activity corresponding to the inhibition/stopping-P3, and not conflict detection, that is, N2-related activity; Hypothesis 3 that loss of control over watching was driving these relationships.

2.4.1 | First-level analyses

We used multiple single-trial robust regression (Fischer et al., 2016; Fischer & Ullsperger, 2013) to regress individual EEG data at each electrode and time point on task-wise task parameters. We employed robust regression because outliers or heteroskedastic distributions bias its performance less than standard regression. All analyses were conducted with MATLAB 2018a (MathWorks) and EEGLAB 14.1. For the GNGT, trial type (go, stop; coded as −1 and 1) was the predictor of interest, whereas the duration of the preceding inter-trial interval served as a regressor of no interest. We used the following GLM for the stimulus-locked EEG:

$$\text{EEG} = \beta_0 + \beta_1 \times \text{trial type} + \beta_2 \times \text{inter-trial interval} + \text{Error}$$

(1)

For the SST, trial type (go, successful stop; coded as −1 and 1) was the predictor of interest, whereas arrow direction and the duration of the preceding inter-trial interval served as regressors of no interest. Erroneous go trials, failed inhibition stop trials, and trials from invalid blocks were not considered in SST analyses. We used the following GLM for the stimulus-locked EEG:
EEG = β₀ + β₁ × trial type + β₂ × arrow direction + β₃ × inter-trial interval + Error

For the MIFLAT, the GLM included feedback valence (positive in the gain context, negative in the loss avoidance context; coded as −1 and 1). We created this contrast to ensure that the direction of the resulting regression weights would correspond to what is normally expected for ERPs (e.g., negative for activity associated with the FRN, which is usually more negative for losses than gains) and thus make them more accessible. Relief and regret trials were not considered in MIFLAT analyses. We used the following GLM for the feedback-locked EEG:

EEG = β₀ + β₁ × feedback valence + Error

EEG data were smoothed using activity ±4 ms around each data point. These first-level analyses yielded individual t values for each regressor at each electrode and time point, which were subjected to one-sample t tests, employing false discovery rate (Benjamini & Yekutieli, 2001) to correct for multiple comparisons. We inspected the resulting timevariant topographies of significant relationships between predictors and EEG data for reflections of the inhibition-N2 and inhibition-P3 in the GNGT, the stopping-N2 and stopping-P3 in the SST, and FRN, feedback-P3a, and feedback-P3b in the MIFLAT. We observed central inhibition-N2 (~240 ms) and inhibition-P3 (~390 ms) equivalents (see Figure 2a); central stopping-N2 (~300 ms) and stopping-P3 (~500 ms) equivalents (see Figure 2b); a frontocentral FRN (~270 ms) and feedback-P3a (~400 ms), and a parietal feedback-P3b (~515 ms) equivalent (see Figure 2c).

2.4.2 Second-level analyses to test Hypotheses 1 and 2

We then examined whether (first-level) FRN, feedback-P3a, and feedback-P3b were specifically related to (first-level) (a) stopping-N2 or stopping-P3 and (b) inhibition-N2 or inhibition-P3. For Hypothesis 2 we examined whether outcome processing was specifically related to implementing inhibition/stoping rather than conflict detection. To test that associations were specific to HBW (Hypothesis 1), we investigated interactions of respective feedback components with group to target these relationships as a function of BW. Models to predict inhibition (GNGT) and stopping (SST) effects are captured in the following general formulae:

Model 1: First-level t = β₀ + β₁ × feedback effect + Error

Model 2: First-level t = β₀ + β₁ × feedback effect + β₂ × feedback effect × BW group + Error

Here, first-level t refers to the effect of the trial type regressor in the GNGT or SST at each electrode and time point. As feedback effect, we used the mean of first-level t-values of the feedback valence regressor in the MIFLAT, corresponding to the FRN (at FCz between 262–272 ms), feedback-P3a (FCz, 388–408 ms), or feedback-P3b (Pz, 500–530 ms). For the interaction term, HBW and NBW were coded as −1 and 1, respectively, and multiplied with the feedback effect regressor.

The models in Equations 4 and 5 were applied in a hierarchical fashion. Thus, to predict the stopping effect, we regressed t values of the SST trial type regressor at each electrode and time point (a) on the FRN and subsequently on the interaction of the FRN with group, (b) on the feedback-P3a and subsequently on the interaction of the feedback-P3a with group, and (c) on the feedback-P3b and subsequently on the interaction of the feedback-P3b with group (masked at p = .010). We applied the same procedure to the GNGT trial type regressor. Our stepwise approach enabled us to identify significantly increased proportions of explained variance. Significant effects of the interactions with group were followed-up with group-wise regressions including only the feedback effect as a predictor, cf. Equation 4. Here, observing a relationship between the effect of feedback valence and trial type (inhibitory/stopping activity) in HBW, but not NBW, would support Hypothesis 1. Further, observing these relationships only for EEG activity associated with the inhibition/stopping-P3, but not N2, would support Hypothesis 2. We also investigated, but did not observe, functionally relevant group differences (see Supplementary Materials).

2.4.3 Second-level analyses to test Hypothesis 3

We examined the moderating role of LOCW by testing whether associations between neural activity in HBW are driven by the loss of control over BW. To this end, we followed-up significant interactions of feedback-related activity with group and tested whether associations of neural activity within HBW were stronger, or only occurred, in HBW with high LOCW. We assigned HBW individuals to groups with low and high LOCW (coded as −1 and 1, respectively) based on a median split of the LOCW sum score. We then submitted the feedback-related regressor in question (again termed feedback effect) and its interaction with LOCW group status to the following model:

First-level t = β₀ + β₁ × feedback effect + β₂ × feedback effect × LOCW group + Error

First-level t again represents the trial type effect in the GNGT or SST at each electrode and time point. A significant
FIGURE 2 Depiction of first-level regression effects for inhibition, stopping, and feedback processing. (a) Effects of the trial type regressor (go, nogo) at Cz in the go/nogo task. (b) Effect of the trial type regressor (go, successful stop) at Cz in the stop-signal task. (c) Effect of the feedback valence regressor (gain, loss) at FCz and Pz in the monetary incentive flanker task. In each figure section, top rows present topographical maps of significant associations between EEG activity and the regressor in question (t values, red: positive, blue: negative, masked at p = 1 × 10^{-6}). The lower left, center, and right show original event-related potential waveforms (shades reflect standard error of the mean), trajectories of t values for the regressor at selected electrodes (gray shades reflect significance at p = 1 × 10^{-6}), and time courses of corresponding p values, respectively.
interaction indicating that the association between the feedback and trial type effects is stronger or only present in the high LOCW group would support Hypothesis 3.

3 | RESULTS

3.1 | First-level regression

3.1.1 | Go/nogo task

We observed (a) negative-going EEG activity at Cz, peaking 288 ms after go onset and reflecting the inhibition-N2 with more negative amplitudes on nogo relative to go trials and (b) positive-going EEG activity (Cz, 388 ms) capturing the inhibition-P3 with more positive amplitudes on nogo relative to go trials (see Figure 2a). Regression weights deviating more strongly from zero around these peaks (negatively for inhibition-N2, positively for inhibition-P3) indicate increased brain activity when inhibiting versus executing a response.

3.1.2 | Stop-signal task

We observed (a) negative-going EEG activity at Cz, peaking 282 ms after go onset and reflecting the stopping-N2 with more negative amplitudes on stop relative to go trials (see Table 1 for mean SSD) and (b) positive-going EEG activity (Cz, 510 ms) capturing the stopping-P3 with more positive amplitudes on stop relative to go trials (see Figure 2b). Regression weights deviating more strongly from zero around these peaks (negatively for stopping-N2, positively for stopping-P3) indicate increased brain activity when successfully stopping an already initiated response versus executing a response.

3.1.3 | Monetary incentive flanker task

We observed (a) negatively inflected EEG activity at FCz, peaking 264 ms following feedback onset and resembling the FRN with more negative amplitudes for negative relative to positive feedback, (b) positive-going EEG activity (FCz, 394 ms) reflecting the feedback-P3a with more positive amplitudes for negative relative to positive feedback, and c) positively inflected EEG activity (Pz, 514 ms) compatible with the feedback-P3b, that is, more positive amplitudes for negative relative to positive feedback (see Figure 2c). As such, regression weights deviating more strongly from zero around these peaks (negatively for FRN, positively for feedback-P3a, and feedback-P3b) reflect brain activity more sensitive to loss than gain.

3.2 | Second-level regression

3.2.1 | Inhibition and outcome processing: Testing Hypotheses 1 and 2

FRN regression weights significantly explained the inhibition effect at centroparietal sites ~250 ms after go onset, that is, the interval of the inhibition-N2 ($p < .010$). Increased FRN predicted larger inhibition-N2 (see Figure S1a in the Supplementary Materials). The interaction term was not significantly associated with the inhibition effect attributed to the inhibition-N2 or inhibition-P3 (Figure S1b). The feedback-P3a regressor explained the inhibition effect at central sites ~415 ms after go onset ($p < .010$), but the interaction with group was not significant. Larger feedback-P3a predicted smaller inhibition-P3 (Figure S2). The feedback-P3b regressor was not significantly associated with the inhibition effect attributed to the inhibition-N2 or inhibition-P3 (Figure 3a). However, adding the interaction term to the model increased $R^2$ at centroparietal sites ($\Delta R^2 = 0.132$ at Cz and 462 ms, $F(2,60) = 4.56$, $p < .050$; $\Delta R^2 = 0.168$ at P1 and 474 ms, $F(2,60) = 6.06$, $p < .050$), that is, the later portions of the inhibition-P3 (Figure 3b). In follow-up analyses, we observed that a significant relationship between feedback-P3b and inhibition-P3 was present at parietal sites (Pz: peak at 400 ms; range 358–460 ms, $p < .010$) in HBW. Consistent with Hypothesis 1, in HBW the effect of loss versus gain (feedback-P3b) was related to inhibitory activity on nogo versus go trials (Figure 4). Consistent with Hypothesis 2, this effect only occurred for EEG activity associated with the inhibition-P3. A transient effect localized to Cz (peak at 464 ms; range 448–478, $p < .010$) in NBW indicated the opposite: NBW with a larger effect of loss versus gain (feedback-P3b) recruited less inhibitory activity on nogo versus go trials (inhibition-P3).

3.2.2 | Stopping and outcome processing: Testing Hypotheses 1 and 2

Neither the FRN regressor nor its interaction with group significantly explained the stopping effect attributed to the stopping-N2 and stopping-P3 (Figure S3). The same was true for the feedback-P3a regressor (Figure S4). As can be seen in Figure 5, the feedback-P3b regressor significantly explained the stopping effect at centroparietal sites ~500–550 ms after go onset, that is, the interval of the stopping-P3. Adding the interaction term to the model increased $R^2$ at parietal sites ($\Delta R^2 = 0.10$ at PO4 and 508 ms, $F(2,60) = 3.41$, $p < .050$). In follow-up analyses, we observed that a significant relationship between feedback-P3b and stopping-P3 was only present in HBW (see Figure 6). Consistent with Hypothesis 1, in HBW the effect of loss versus gain (feedback-P3b) was
3.2.3 Examining the role of loss of control for the relationship between feedback and inhibition/stopping effects: Testing Hypothesis 3

After observing that the feedback-P3b was related to inhibition-P3 and stopping-P3 in HBW, we examined the influence of LOCW on these relationships. The interaction of feedback-P3b with degree of LOCW significantly explained the stopping effect at centroparietal sites ~440–550 ms after go onset (peak at Pz and 446 ms, second-level \( t = 0.31, p = .033 \)), that is, the interval of the stopping-P3 (see Figure 7a). Similarly, the interaction of feedback-P3b with LOCW was significantly associated with the inhibition effect at centroparietal electrodes ~300–400 ms after go onset (peak at CPz and 334 ms, second-level \( t = 0.29, p = .034 \)), that is, the interval of the inhibition-P3 (see Figure 7b). In high but not low LOCW individuals, the feedback-P3b was associated with the inhibition-P3 as well as stopping-P3.

4 DISCUSSION

This study aimed to investigate the relationship of brain activity related to monetary loss versus gain with motor inhibition as well as stopping in binge-watchers. We observed that HBW, but not NBW, showed a relationship between outcome processing (losses versus gains) and inhibition and stopping processes. This was found for the association between the feedback-P3b (rather than FRN or
feedback-P3a), which is relevant for processing the motivational salience of outcomes as well as the implementation of adaption (Fischer & Ullsperger, 2013; Yeung & Sanfey, 2004), and the inhibition/stopping-P3 (rather than the inhibition/stopping-N2), which are relevant for implementing the actual inhibition and stopping processes. Exploratory analyses further suggested that the observed relationships were prominent in HBW with greater impairments as indicated by the degree of LOCW, but not in HBW with lower LOCW. Additionally, these findings occurred in the absence of differences between HBW and NBW in performance or EEG magnitude.

The observed relationships of brain activity are consistent with our main hypothesis (Hypothesis 1) that outcome and inhibitory processing are related in HBW. This supports the notion that proneness to binge-watch can be understood in terms of the interplay between brain functions rather than isolated alterations thereof. The observed association can be construed such that HBW with lower inhibition/stopping related activity also show reduced feedback-P3b activity. Consequently, BW risk might be conferred by the fact that deficits in one domain (e.g., feedback sensitivity) are accompanied by deficits in another (e.g., stopping). According to this account, binge-watching individuals with low feedback-related brain activity might be characterized by deficits in other goals, and alternative rewarding behaviors. Though speculative, this could be conferred by a shared risk for engaging in excessive and addictive behaviors or for depression, respectively, which are reportedly associated with reduced P300 to monetary outcomes (Euser et al., 2013; OUMEZIANE & Foti, 2016). Binge-watching

**FIGURE 4** Group-specific depiction of the second-level relationship between (first-level) inhibition effects in the go/nogo task and (first-level) feedback-P3b. (a) Main effect of feedback-P3b on inhibition in high binge-watchers. (b) Main effect of feedback-P3b on inhibition in no-binge-watchers. In each figure section, top rows present topographical maps of significant associations between first-level inhibition effects and feedback-P3b (t values, red: positive, blue: negative, masked at p = .010). Panels below present (from left to right) progressions of first-level t values associated with inhibition as a function of feedback-P3b (shades reflect standard error of the mean), trajectories of second-level t values at Pz for the relationship (gray shades reflect significance at p = .010), time courses of corresponding p values, and scatterplots of the relationship between the feedback-P3b regressor as entered into the model (Equation 4) and first-level t values for the inhibition effect at Pz and 400 ms.
individuals with low brain activity during inhibition and stopping might have a low capacity or willingness to recruit cognitive resources in response to inhibitory demands. If these dispositions co-occur, (a) low sensitivity to negative consequences and low motivation toward goals unrelated to BW could incentivize continued watching (e.g., as a compensatory behavior), and (b) a simultaneous inability to stop watching or change behavior, even when in conflict with alternative activities or responsibilities, could facilitate BW. This interaction would be consistent with findings that the effect of depression on BW is mediated by low self-control (Tukachinsky & Eyal, 2018) and that need for instant gratification and low self-control particularly characterize unintentional BW (Riddle et al., 2018; Shim et al., 2018). Of note, it is yet unclear whether associations of brain activity indicate causality or result from an underlying, unobserved source. For instance, Weafer et al., (2019) suggested low striatal D2 dopamine receptor density as a potential mechanism for a similar relationship between feedback and inhibitory processing in binge-drinkers.

The role of low self-control is emphasized by our exploratory finding that the relationship between brain activity from different domains in HBW was moderated by self-reported LOCW (Hypothesis 3). Indeed, the fact that high LOCW seemed to be driving the effects regarding HBW might support the notion that coupling between outcome and inhibitory processing reflects a neural risk marker for problematic BW. As such, our study provides a potential way forward to more distinctly separate problematic and unproblematic BW at the neural level. This is warranted given recent unfruitful attempts to differentiate between these concepts with behavioral indices (Flayelle, Verbruggen, et al., 2020). Moreover, some authors postulate that mitigating post-binge negative affect and need for gratification with further watching reflects a vicious
cycle with addictive properties (Panda & Pandey, 2017; Riddle et al., 2018; Walton-Pattison et al., 2018). The present results might be tied in with this account if confirmed in larger samples. Also, it would be interesting to examine whether symptoms related to the loss of control are linked to depression or facets of impulsivity. However, conclusions conceptualizing BW as an addictive behavior should only be drawn tentatively. The general caveat to consider the motivation for excessive behaviors before unnecessarily pathologizing them applies (Kardefelt-Winther et al., 2017).

Whereas the conclusion that BW carries addictive potential cannot be inferred from the present data, this aspect deserves further consideration. The present associations between inhibitory and outcome processing echo a core assumption of addiction models that impaired inhibition of positive (craving) or negative urges (e.g., withdrawal) accompanies hypersensitivity to addictive and hyposensitivity to alternative rewards (Zilverstand et al., 2018). A recent meta-analysis showed that pathological gambling is characterized by reduced striatal reward signals (Luijten et al., 2017). Further, diminished reward processing (captured with the reward positivity) combined with disinhibition was shown to prospectively predict SUD (Joyner et al., 2019). Weafer et al., (2019), investigating the contrast gain minus loss, observed an inverse relation between striatal feedback responses and prefrontal inhibitory activity in individuals at high risk of compulsive alcohol use. In the current study, we examined outcome processing with the reverse contrast, that is, loss minus gain. Therefore, the current results are consistent with those of Weafer et al., (2019). In light of this evidence, the
present approach might be applied to clinical samples with addictive disorders, or at risk of developing addictions, to examine its utility for investigating neural risk markers for addictive behaviors.

The high temporal resolution of our recordings enables us to draw conclusions regarding specific subprocesses involved in inhibitory and outcome processing. As hypothesized (Hypothesis 2), the associations were specific to EEG activity assumed to reflect the actual implementation of inhibiting or stopping a response (P3; Waller et al., 2019; Wessel & Aron, 2015), rather than detecting conflict and signaling the need to overcome an automatic response tendency (N2; Falkenstein, 2006; Nieuwenhuis et al., 2003). The need to inhibit or stop, therefore, seems adequately represented, whereas problems may arise from a subsequent capacitive or motivational deficit in addressing this demand.

This is likely tied to alterations in the motor and premotor cortices, that is, the suggested neural source of the inhibition P3 (Huster et al., 2010; Ramautar et al., 2004). With respect to outcome processing, alterations were observed after early computations of expectancy violations (FRN) or attentional processing (feedback-P3a). The observed associations rather concerned EEG activity assumed to reflect the motivational salience of outcomes and later assessments of expectancy violation (feedback-P3b; Bellebaum & Daum, 2008; Mars et al., 2008; Yeung & Sanfey, 2004). This finding is particularly interesting given prior observations that the feedback-P3b is tied to subsequent behavioral adaptations (Fischer & Ullsperger, 2013; San Martin et al., 2013; von Borries et al., 2013). This further suggests that the present alterations may, indeed, carry behavioral significance, for example, regarding impairments in choosing alternative behaviors as a

**FIGURE 7** Loss of Control over Watching (LOCW) moderates the relationship of the feedback-P3b with the first-level (a) stopping and (b) inhibition effects in high binge-watchers. Top rows present topographical maps of significant effects of the interaction between feedback-P3b and low versus high LOCW on stopping and inhibition (t values, red: positive, blue: negative, masked at p = .050). Center panels show corresponding progressions of first-level t values associated with stopping and inhibition as a function of the interaction between feedback-P3b and LOCW (shades reflect standard error of the mean; gray areas reflect significance at p = .050). Bottom panels detail the interaction by showing first-level t-values of the stopping effect (M ± SEM between 450–550 ms) and inhibition effect (M ± SEM between 320–370 ms) as a function of LOCW and feedback-P3b.
function of BW-related consequences. The neural sources of the feedback-P3b are broadly distributed (Nieuwenhuis et al., 2005; Polich, 2007), preventing well-supported speculations regarding the neural circuits involved in the present findings. However, recent work indicated that inhibitory activity in the inferior frontal gyrus and medial prefrontal cortex was negatively associated with feedback-related striatal signals in binge-drinkers (Weafer et al., 2019). This is consistent with the frontostriatal interactions assumed to be relevant for addiction-related dysregulation (Zilverstand et al., 2018). Last, FRN and N2 were positively associated across both groups. This may be due to their shared neural source in the medial prefrontal cortex (Gruendler et al., 2011; Kok et al., 2004; Nieuwenhuis et al., 2003).

The present study should be regarded in the light of its limitations. We investigated monetary rather than BW-related outcomes and did not analyze data in terms of BW motives. Further, we defined BW by the number of BW episodes even though the definition of what BW entails is a matter of ongoing debate (Flayelle, Maurage, et al., 2020). We also recruited binge-watchers generally rather than focusing on problematic BW. Including only problematic BW would have enabled us to address the important role of loss of control in BW with more statistical power. Our samples did not significantly differ in anxiety, depression, or impulsivity. However, this also aided our analyses by excluding potential confounds. Last, a less highly educated sample might have introduced more variance in the domain of cognitive control.

In conclusion, this is the first study to report that diminished neural correlates in two domains of motor inhibition are associated with reduced neural differentiation of gains and losses in high binge-watching individuals. The fact that we found associations of outcome processing with both inhibition and stopping—physiologically and psychologically distinct constructs (Littman & Takacs, 2017; Raud et al., 2020)—corroborates the reliability of our findings and suggests that this is a consistent underlying pattern. Notably, this pattern emerged in the absence of behavioral or neural group differences, underlining the superior sensitivity of our multi-measure approach over investigating single-outcome group effects. Investigating cross-modal relationships may be useful and particularly sensitive in establishing patterns and profiles conferring risk.

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CONFLICT OF INTEREST
None of the authors have known financial interests or potential conflicts of interest to disclose.

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Raoul Dieterich: Conceptualization; Formal analysis; Software; Visualization; Writing-original draft. Verena Willhorst: Software; Writing-review & editing. Julia Berghäuser: Investigation; Project administration; Writing-review & editing. Rebecca Overmeyer: Conceptualization; Investigation; Project administration; Writing-review & editing. Tanja Endrass: Conceptualization; Funding acquisition; Investigation; Methodology; Project administration; Software; Supervision; Visualization; Writing-review & editing.

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SUPPORTING INFORMATION
Additional supporting information may be found online in the Supporting Information section.

FIGURE S1 Second-level relationship between (first-level) inhibition effects in the go/nogo task and (first-level) FRN. (a) Main effect of FRN on inhibition. (b) Effect of the interaction between FRN and group on inhibition. In each figure section, top rows present topographical maps of significant associations between first-level inhibition effects and the regressor in question (t-values, red: positive, blue: negative, masked at p = .010). The lower left, center, and right show progressions of first-level t-values associated with inhibition as a function of FRN or its interaction with group (shades reflect standard error of the mean), trajectories of second-level t-values at electrode Cz for the relationship in question (gray shades reflect significance at p = .010), and time courses of corresponding p-values, respectively.

FIGURE S2 Second-level relationship between (first-level) inhibition effects in the go/nogo task and (first-level) feedback-P3a. (a) Main effect of feedback-P3a on inhibition. (b) Effect of the interaction between feedback-P3a and group on inhibition. In each figure section, top rows present topographical maps of significant associations between first-level inhibition effects and the regressor in question (t-values, red: positive, blue: negative, masked at p = .010). The lower left, center, and right show progressions of first-level t-values associated with inhibition as a function of FRN or its interaction with group (shades reflect standard error of the mean), trajectories of second-level t-values at electrode Cz for the relationship in question (gray shades reflect significance at p = .010), and time courses of corresponding p-values, respectively.

FIGURE S3 Second-level relationship between (first-level) stopping effects in the stop signal task and (first-level) FRN. (a) Main effect of FRN on stopping. (b) Effect of the interaction between FRN and group on stopping. In each figure section, top rows present topographical maps of significant associations between first-level stopping effects and the regressor in question (t-values, red: positive, blue: negative, masked at p = .010). The lower left, center, and right show progressions of first-level t-values associated with stopping as a function of FRN or its interaction with group (shades reflect standard error of the mean), trajectories of second-level t-values at electrode Cz for the relationship in question (gray shades reflect significance at p = .010), and time courses of corresponding p-values, respectively.

FIGURE S4 Second-level relationship between (first-level) stopping effects in the stop signal task and (first-level) feedback-P3a. (a) Main effect of feedback-P3a on stopping. (b) Effect of the interaction between feedback-P3a and group on stopping. In each figure section, top rows present topographical maps of significant associations between first-level stopping effects and the regressor in question (t-values, red: positive, blue: negative, masked at p = .010). The lower left, center, and right show progressions of first-level t-values associated with stopping as a function of feedback-P3a or its interaction with group (shades reflect standard error of the mean), trajectories of second-level t-values at electrode Cz for the relationship in question (gray shades reflect significance at p = .010), and time courses of corresponding p-values, respectively.

FIGURE S5 Main effects of group in the go/nogo task (a), stop signal task (b), and monetary incentive flanker task (c). In each figure section, top rows present topographical maps of group effects at a liberal threshold (t-values, red: positive, blue: negative, masked at p = .050). The lower left, center, and right show progressions of first-level t-values for each group (shades reflect standard error of the mean), trajectories of second-level t-values for the group effect (gray shades reflect significance at p = .010), and time courses of corresponding p-values, respectively.

TABLE S1 Items of the loss of control over watching scale