Neurodevelopment and Risk-Taking: Exploring the Role of Response Inhibition and Feedback-Related Processes in Mid-to-late Adolescents and Young Adults

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BSc (Honours)

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Thesis Declaration

I, An Thanh Nguyen, certify that:

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Regarding regulation 33 (points 1 and 2) from the regulations governing higher research degrees of the Postgraduate Research School of the University of Western Australia, the following outlines the contribution by the candidate to each manuscript:

This thesis contains work that has been published and/or prepared for publication. Each of the studies (chapters 2-5) contained within this thesis was designed by the candidate in collaboration with his supervisors, Dr. Allison M. Fox and Dr. Jonson J. Moyle. The candidate recruited and tested all participants in conjunction with other Psychology PhD students in the Neurocognitive Development Unit. The candidate modified and wrote stimulus presentation and analysis programs, conducted all data and statistical analyses. The candidate wrote all the manuscripts and made revisions in accordance with feedback provided by supervisors and journal reviewers.

Two conference poster presentations, one conference oral presentation, and one publication resulted from this thesis:


Regarding regulation 1.3.1.33(2)(a) and (b), the signatures of the co-authors are contained below, certifying that the above descriptions accurately detail the work of the candidate to the publications and that they agree to the inclusion of these manuscripts in this thesis.

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Signed approval of the co-authors for the discussed chapters to be included in the thesis:

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Signed: 

Dr. Allison M. Fox 

Dr. Jonson J. Moyle
Thesis Abstract

Adolescence is a critical transitional period from child to adulthood characterised by increases in risk-taking. From an evolutionary perspective, these changes are argued to be adaptive, promoting independence, and improving chances of survival and reproductive success. From a neurodevelopmental perspective, it is theorised that these changes are partly driven by differences in the development of cognitive control and reward processing brain systems resulting in a greater but under-controlled tendency to pursue rewards. Whilst these changes are both normal and adaptive, the bias towards rewarding stimuli could make adolescence a period of increased vulnerability for the development of maladaptive behaviours.

Although the neurodevelopmental model provides an intuitive account of adolescent risk-taking, there are gaps in our knowledge regarding the development of specific cognitive control and reward-related processes (response inhibition and feedback monitoring). With respect to response inhibition, the existing evidence has been largely derived from child-adult comparisons with fewer studies focusing on adolescence and the adolescent-adult transition. For feedback monitoring during risk-taking, there has been a greater emphasis on adolescents. However, there is a large focus on static risky decision-making tasks where risks are static and feedback is all together random and uninformative. These previous studies provide an important contribution; however, they do not capture the affective nature and novelty of risk-taking that are evident in a real-world environment.

Thesis aims and investigations

This thesis presents four investigations examining the development of risk-taking, response inhibition and feedback monitoring processes during the adolescent-adult transition using behavioural and event-related potential (ERP) techniques. The first two studies focus on response inhibition. In a sample of young adults, the first study explores the use of a modified go/nogo task which measures partial inhibitions, to improve task sensitivity to performance differences. Results showed that partial inhibitions accounted for a substantial proportion of errors on nogo trials, and significant modulations in the nogo N2 and P3 on
partial compared to successful inhibitions were also observed. The results highlight the importance of partial inhibitions and provided insight into the functional significance of the inhibitory process.

Capitalising on the sensitivity of this go/nogo task to inhibitory failures, the second study examines response inhibition during the adolescent-adult transition, comparing mid-to-late adolescents and young adults ($N(adolescents) = 28$ adults and $N(adults) = 22$). Behavioural results showed that adolescents made more nogo errors than adults. Similarities in the nogo N2 and P3 suggest that conflict monitoring and inhibitory processes are relatively mature by mid-to-late adolescence, and observed delays in P2 latency were thought to reflect early attentional processing delays. Poorer performance in adolescents was driven by a failure to compensate for slower stimulus processing when responding, rather than an immature inhibitory system.

In Study 3 the reward processing component of the model was examined by investigating feedback monitoring processes elicited during the Balloon Analogue Risk Task, (BART) in mid-to-late adolescents and young adults, ($N(adolescents) = 28$ and $N(adults) = 22$). Similar valence effects on the feedback-related negativity (FRN) and feedback P3 were observed suggesting that feedback discrimination and evaluative processes are relatively mature by mid-to-late adolescence. Overall P2 and FRN amplitudes were larger in adolescents than adults, reflecting a greater allocation of attention resources to process external feedback.

The final study explored the role of previously identified inhibitory and feedback processes in low and high drinking behaviours in young adults ($N(low
drinking) = 37$ and $N(high
drinking) = 32$). Results showed that high drinkers exhibited greater preference for immediate reward than low risk drinkers. However, no ERP differences were observed between groups. These results indicate that differences in drinking behaviours in typically functioning young adults may not necessarily be reflected in alterations of the underlying psychophysiological processes.
Conclusions

The thesis makes an important contribution to our understanding of risk-taking in adolescents by highlighting the relative maturity of inhibition and feedback monitoring processes, as well as characterising ongoing changes in the efficiency of attentional processes throughout this important developmental period. The thesis provides insight regarding the functional significance of the inhibitory process, reward processing and risk-taking.
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Chapter 1: General Introduction

Risk-taking

Risk-taking is an umbrella term that refers to engagement of behaviours that are considered risky, which can be defined in many ways. Traditionally, it has been used to describe behaviours with potentially severe negative outcomes. It is typically used to label behaviours that are anti-social or self-destructive, often referring to addictive and delinquent behaviours (e.g. excessive alcohol consumption, smoking, illicit substance use, problem gambling, violence, theft, reckless driving, and unprotected sex) (Arnett, 1992; Reyna & Farley, 2006). These behaviours are considered risky because when an individual engages in these behaviours, they expose themselves to potential harm.

To study risk-taking behaviour in the laboratory, researchers have developed a range of experimental tasks where individuals make decisions under varying conditions of uncertainty (e.g. IOWA gambling task, Cambridge Card Task, Balloon analogue Risk Task). In these tasks, the magnitude of reward and punishment, likelihood of these outcomes are varied, and the context of whether decisions are individually or sequentially are varied (referred to as static or dynamic risk-taking), and risk-taking is evaluated based on the frequency of selecting options that are pre-defined as risky. For example, risk-taking has been described under the decision-making framework where it refers to preference for options with high outcome variability (Paulsen, Platt, Huettel, & Brannon, 2011). Options with greater outcome variability are considered risky because they are associated with a greater range of outcomes and greater uncertainty, which makes it more difficult to anticipate the consequences. An example might be when the participant selects an option with the potential to gain or lose four points compared to the potential to gain or lose two.

Risk-taking has also been described as ability to quantitatively trade-off between different options. In this definition, risk-taking refers to poor decision-making and is represented by selection of sub-optimal or disadvantageous options. It is argued that risk-taking is driven by poor decision-making which is likely to occur if one does not understand the probability of outcomes, and neglects or misrepresents relevant information such as the
value of potential gains and losses (Figner, Mackinlay, Wilkening, & Weber, 2009). An example of this definition is when the participant selects an option with the highest single pay-out but neglects the fact that this option is associated with long-term negative outcomes. This definition is focused on the overall value of each option. Collectively, there are many definitions, with each definition focusing on different aspects of risky behaviour.

Overall, risk-taking research is largely motivated by the potential negative outcomes of risky behaviour. Researchers endeavour to understand factors that contribute to risk-taking so that one can develop strategies to manage risk-taking and mitigate the negative consequences.

Based on previous research highlighting the utility of the Balloon Analogue Risk task (BART), a dynamic risk-taking task, the current thesis focused on the use of the BART as a measure of risk-taking. Highlighting the reliability and validity of the BART, various comparisons between risky versus non-risky groups have shown associations between engagement in real-world risky behaviours and increased risky decision-making in the BART (Aklin, Lejuez, Zvolensky, Kahler, & Gwadz, 2005; Bornovalova, Daughters, Hernandez, Richards, & Lejuez, 2005; Coffey, Schumacher, Baschnagel, Hawk, & Holloman, 2011; Hopko et al., 2006; Ledgerwood, Alessi, Phoenix, & Petry, 2009; Lejuez et al., 2002, 2003; Lejuez, Simmons, Aklin, Daughters, & Dvir, 2004), and has also been shown to have good test-retest reliability across days ($r = +.77, p < .001$) (White, Lejuez and de Wit, 2008). There is also emerging evidence revealing its utility in revealing developmental differences in compared to static risk-taking tasks (Figner et al., 2009).

**Adolescence and risk-taking**

Adolescence is the transitional period from child- to adulthood where substantial biological, cognitive and behavioural changes occur. Most notably, it has been characterised as a period of increased impulsivity and risk-taking, with reports of heightened engagement in risk-taking in adolescents compared to adults (Defoe, Dubas, Figner, & Aken, 2015). From an evolutionary perspective, these changes are argued to be adaptive, promoting independence, and improving chances of survival and reproductive success. From a neurodevelopmental perspective, it is theorised that these changes are partly driven by
relative differences in the development of cognitive control and reward processing brain systems, where the coupling of an early maturing reward processing system with a relatively immature cognitive control system leads to a greater but under-controlled tendency to pursue rewards (Shulman et al., 2016). Whilst these changes in risk-taking are both normal and adaptive, the bias towards rewarding stimuli could make adolescence a period of increased vulnerability for the development of maladaptive behaviours.

This model of risk-taking has been studied using various methods including structural and functional magnetic resonance imaging (sMRI, fMRI), and electroencephalography (EEG) with the event-related potential (ERP) technique. Whilst there is evidence for regional differences in the development of the structure and activation of the brain from child- to adulthood, our understanding about the changes occurring during the adolescent-adult transition, and how differences in specific processes relate to risk-taking are unclear. This general introduction will provide an overview of the neurodevelopmental model of risk-taking, briefly introducing the implicated brain systems and reviewing key developmental studies using structural and functional neuroimaging techniques from which the model was derived. However, the thesis will be focusing brain electrophysiology using ERP technique, a method with high temporal resolution. The general introduction will provide an overview of the ERP method and relevant ERP indices, followed by a review of developmental ERP studies. It will highlight the gaps in the current research and the chapter will be concluded with an outline of investigations conducted to provide insight into these areas.

**Neurodevelopmental Model of Risk-Taking**

Several versions of the neurodevelopmental model exist, implicating the role of cognitive control and reward processing systems (Casey, Jones, & Hare, 2008; Shulman et al., 2016; Steinberg, 2008). In Steinberg’s (2008) dual-systems model, heightened risk-taking is attributed to the coupling of a slowly developing cognitive control system with a reward processing system that follows an inverted-U shaped developmental trajectory, which peaks in its sensitivity during mid-adolescence. Similarly, Casey et al.’s (2008) maturational imbalance model also proposes a slowly developing cognitive control system, but posits that the sensitivity of the reward processing system increases in early adolescence but reaches a
plateau in mid-adolescence. Although there are slight differences in the proposed developmental trajectory of each system, they both consider that relative differences in maturity of these systems underlies developmental changes in risk-taking behaviour.

Cognitive control refers to a set of high-order processes which are involved in modulating information processing and behavioural output in a goal-directed manner. One sub-process that is commonly examined in risk-taking is response inhibition, which refers to the ability to suppress compelling but inappropriate responses. Response inhibition is commonly examined using the go/nogo task and the stop-signal task, and is primarily associated with the functioning of the prefrontal cortex (e.g. dorsolateral and ventrolateral regions, inferior and middle frontal gyri) but is also implicated with the functioning of a distributed network including the striatum, pre-supplementary motor area, parietal, and temporal lobes (Chikazoe, 2010; Garavan, Ross, & Stein, 1999; Liddle, Kiehl, & Smith, 2001; Swick, Ashley, & Turken, 2011).

Numerous studies have reported significant deficits in response inhibition (reflected by reductions in accuracy) in heavy and dependent alcohol using groups relative to controls (Bjork, Hommer, Grant, & Danube, 2004; Goudriaan, Oosterlaan, De Beurs, & Van Den Brink, 2005; Noël et al., 2007; Pandey et al., 2012; Rubio et al., 2007), with a recent meta-analysis by Smith et al. (2014) reporting a statistically significant combined effect across these investigations. Poor inhibition is thought to contribute to heightened risk-taking due to an inability to regulate appetitive tendencies towards rewarding stimuli. Several studies using the go/nogo task have reported differences in inhibitory processes between alcohol dependent and at-risk groups compared to healthy control (Cohen, Porjesz, Begleiter, & Wang, 1997; Colrain et al., 2011; Fallgatter, Wiesbeck, Weijers, Boening, & Strik, 1998; Kamarajan et al., 2005; Karch et al., 2008; Luijtjen et al., 2014; Pandey et al., 2012; Pfefferbaum, Rosenbloom, & Ford, 1987).

Reward processing refers to a set of processes involved in monitoring, predicting, discriminating and evaluating outcomes and has received greater attention in risk-taking research. It is associated with dopaminergic neurons and the functioning of various cortical (e.g. ventromedial prefrontal cortex, orbitofrontal cortex) and sub-cortical structures (e.g.}
ventral tegmental area, substantia nigra, striatum, thalamus). However, there is a greater focus on sub-cortical structures where these signals are thought to originate. The sub-cortical structures involved in reward processing have been collectively referred to by many terms, including the sub-cortical limbic-striatal system, the mesolimbic dopaminergic system and the socioemotional-incentive processing system (Steinberg, 2008).

Early evidence supporting the role of the dopaminergic system in reward processing came from electrophysiological studies in rodents and non-human primates (Ljungberg, Apicella, & Schultz, 1992; Richardson & Gratton, 1996; Schultz, Apicella, & Ljungberg, 1993). These studies reported that dopamine neurons were responsive to the presentation and omission of rewards or reward-conditioned stimuli. Specifically, phasic increases in activity were elicited following the presentation of rewarding stimuli and phasic reductions were observed when rewards were omitted in the timeframe that rewards would have normally been presented.

It was theorised that the phasic changes in the dopaminergic system, referred to as reward prediction error signals, reflect processes involved in making predictions about ongoing events and the comparison of these predictions with actual outcomes (Schultz, Dayan, & Montague, 1997). Phasic increases signal that an outcome was better than predicted, whereas phasic decreases signal that an outcome was worse than predicted. These dopaminergic signals have downstream effects on various cortical and sub-cortical strictures, such as the striatum, anterior cingulate cortex, ventromedial and orbitofrontal cortices, where the activity in these regions are thought to be involved in the reinforcement and suppression of behaviours (Holroyd & Coles, 2002).

**Development of brain systems implicated in cognitive control**

With respect to the development of the prefrontal cortex, evidence for its prolonged development is largely derived from structural studies examining various gross and microstructural features (e.g. synaptic density, cortical thickness/grey matter volume, white matter volume and diffusion indices). Most measures show regional differences in development with prefrontal regions showing later maturation – although results vary across measures. In a post-mortem histological examination of synaptic density, it was reported that
the reduction in synaptic density in auditory and visual cortices reached adult levels around 12 years whereas prefrontal regions reached adult levels during mid-adolescence (Huttenlocher & Dabholkar, 1997). In another study, mean diffusivity showed a posterior to anterior development, with occipital, posterior and parietal regions peaking between 10 to 20 years whereas temporal and prefrontal regions peak after 20 years (Tamnes et al., 2010). However, there are also studies that have reported no regional differences. In one study, overall estimates of white matter volume showed no statistically significant regional difference were reported (Giedd et al., 1999).

With respect to cortical thickness or grey matter volume, Gogtay et al. (2004) examined changes in cortical thickness during childhood and adolescents using MRI, recorded every 2 years for 8-10 years between 4 and 21 years of age. They found that primary functions such as the primary sensorimotor cortex and visual cortices reached adult levels earlier, whereas areas involved with more complex and integrative tasks such as the prefrontal cortex took longer to develop. More recent findings suggest that developmental changes in cortical thickness in most regions are prolonged. Tamnes et al. (2010) also examined changes in cortical thickness, reporting greatest reductions occurring throughout adolescence. It should be noted that developmental changes are characterised by early increases followed by a decrease throughout adolescence and adulthood, and maturity is estimated by the following reduction is complete. Cortical thickness of the lateral occipital cortex and lateral orbitofrontal cortex reached minimal thickness before 25. For other most regions the estimated time to reach 90% of minimum cortical thickness exceeded the age range of the sample. Although there is some variability between measures/studies relating to the absolute time-frame of brain development, recent reviews provide support for regional differences indicating prolonged developmental change in the prefrontal cortex relative to other brain regions (Caballero, Granberg, & Tseng, 2016).

**Development of brain systems implicated in reward processing**

With respect to the development of reward processing system, evidence for developmental change throughout adolescence are largely derived from functional neuroimaging studies (Ernst et al., 2005; Galvan et al., 2006; Van Leijenhorst et al., 2010).
These studies showed significant differences in activation of the striatum during the presentation, omission or anticipation of reward outcomes which were thought to reflect the development of the reward processing system. For example, Ernst et al. (2005) reported greater activation of the left nucleus accumbens in response to reward feedback in adolescents compared to adults. Furthermore, they showed a positive correlation between positive emotion (i.e. how they felt on a 5-point scale after rewarding feedback was presented) and activity nucleus accumbens in adolescents.

Galvan et al. (2006) who investigated the effect of age and reward size on reward processing reported greater activation of the nucleus accumbens in response to reward feedback in adolescents compared to children and adults. They observed that extent of activity in the nucleus accumbens in adolescents was more like adults than in children. They also examined the volume of activation which was more similar to children than adults. Greater magnitude of activation in adolescents was interpreted as reflecting a hyperactive reward-processing system in adolescents, whereas similar volume of activation of the nucleus accumbens in adolescents and adults was thought to reflect the relatively mature reward-related processing system. They also looked at the orbitofrontal cortex and found that the volume of activation was more like children than adults indicating the relative immaturity of frontal regions.

Similarly, Van Leijenhorst et al. (2010) reported greater activation of the anterior insula during the anticipation of uncertain outcomes in adolescents compared to young adults. During reward presentation, greater activation of the ventral striatum was observed in adolescents compared to other age groups and young adults showed greater activation of the orbitofrontal cortex during reward omission. These were thought to reflect the hypersensitivity of the reward processing system and the relative immaturity of prefrontal regions.

Although gross differences in activation has implicated the development of structures and functions, poor temporal resolution limits the ability to closely examine specific processes, how they change with development and ultimately, how they impact risk-taking. As responses in cognitive tasks and decision-making in the real-world often occur rapidly
and the successful responses depend on time-sensitive processes, examining activity with high temporal resolution is important for deeper understanding the role of brain development in risk-taking. For example, it can provide insight into whether these are related to orienting towards outcomes, processing of feedback information, or the deeper evaluation of this information.

**Electroencephalography and Event-Related Potentials**

Electroencephalography (EEG) refers to a neuroimaging technique that measures electrical activity at the surface of the scalp and has a temporal resolution in the order of milliseconds, making it ideal for examining time-course of activation with great precision (Luck, 2014). Many researchers use the event-related potential (ERP) technique to examine changes in EEG activity associated with specific events such as the presentation of stimulus or the execution of a response. Several studies have used the ERP technique to examine the development neurocognitive processes associated with response inhibition and feedback monitoring. The following sections will provide background relating to the functional significance of ERPs involved in inhibition and feedback processing, and review studies examining developmental changes in these ERPs from child- to adulthood.

*Response inhibition and functional significance of the nogo N2 and no go P3*

Developmental inhibition studies tend to focus on the nogo N2 and nogo P3 elicited during the go/no-go task, where participants respond to frequently presented go stimuli while withholding responses to infrequent nogo stimuli (Ciesielski, Harris, & Cofer, 2004; Cragg, Fox, Nation, Reid, & Anderson, 2009; Cragg & Nation, 2008; Hammerer, Li, Muller, & Lindenberger, 2010; Johnstone et al., 2007; Johnstone, Pleffer, Barry, Clarke, & Smith, 2005; Jonkman et al., 2003; Lamm, Zelazo, & Lewis, 2006; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006; Okazaki et al., 2004). Event-related potentials to response inhibition is also widely investigated using the stop-signal task in adults, however few studies have used this task in a developmental context (Johnstone et al., 2007; Lo et al., 2013) and there is some evidence for task-related differences in brain activation between the go/nogo and stop-signal task (Enriquez-Geppert, Konrad, Pantev, & Huster, 2010; Swick et al., 2011). Developmental studies have also looked at other cognitive control tasks such as the Eriksen flanker task.
(Clawson, Clayson, Keith, Catron, & Larson, 2017; Friedman, Nessler, Cycowicz, & Horton, 2009; Ladouceur, Dahl, & Carter, 2004), however interference suppression processes involved in the flanker task have been theorised to be different from response inhibition (Nigg, 2000) with some evidence of underlying differences (Brydges et al., 2012; Brydges, Anderson, Reid, & Fox, 2013). Although it is likely that these findings across tasks are related, the degree to which they can be directly compared is unclear. Therefore, the current thesis will primarily focus on developmental studies using variations of the go/nogo task.

The nogo N2 refers to a frontocentrally distributed negativity occurring around 200-350 ms post-stimulus onset and the nogo P3 refers to a frontocentral-to-central positivity occurring 300-600 ms. The amplitude of these peaks is typically enhanced on nogo compared to go trials and these enhancements are thought to reflect inhibition-related processing. Throughout this thesis, the nogo N2 and nogo P3 will be simply referred to as the N2 and P3, and the change in N2 amplitude elicited on go and nogo trials will be referred to as the nogo N2 and P3 effects. Specific descriptions to distinguish the N2/P3 elicited on go or nogo trials, and different N2/P3 sub-components will be used when necessary. Initial observations suggested that the N2 enhancement reflected inhibitory processing (Falkenstein, Hoormann, & Hohnsbein, 1999; Jodo & Kayama, 1992; Nakata et al., 2006), However, based on subsequent studies showing modulation in N2 amplitude to stimulus-characteristics such as probability on both go and nogo trials, it was argued that its enhancement wasn’t exclusive to trials that required inhibition (Donkers & Van Boxtel, 2004; Fox, Michie, Wynne, & Maybery, 2000; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). It was alternatively proposed that the N2 may reflect a conflict monitoring process which detects when multiple incompatible response representations are activated simultaneously (Botvinick, Braver, Barch, Carter, & Cohen, 2001). Although N2 is sensitive to stimulus probability, it has been argued that alternative accounts such as conflict monitoring and novelty detection account cannot completely account for the entire nogo N2 effect and it has been suggested that N2, in some part, reflects response inhibition (Folstein & Van Petten, 2008).

Recent evidence using cued go/nogo tasks provides support for the role of P3 in response inhibition. These studies showed that P3 amplitude was sensitive to trials requiring
response inhibition. For example, Donkers and Van Boxtel (2004) found that P3 was only enhanced to nogo compared to go trials but was not enhanced to infrequent ‘GO’ trials which didn’t require inhibition. In studies using a cued go/nogo task, it was shown that P3 was not enhanced when there was no planned response to inhibit or when an unplanned response was executed. P3 enhancement was only observed when participants had to change responses or suppress an unplanned response (Randall & Smith, 2011; Smith, Johnstone, & Barry, 2007).

**Development of N2 and P3 from childhood to adulthood**

Numerous studies have examined developmental changes in N2 and P3 from child- to adulthood (Brydges et al., 2013; Ciesielski et al., 2004; Davis, Bruce, Snyder, & Nelson, 2003; Hämerer et al., 2010; Johnstone et al., 2005; Jonkman et al., 2003; Jonkman, 2006; Lamm et al., 2006; Lewis et al., 2006; Okazaki et al., 2004). These studies mainly focus on the N2 with fewer studies examining changes in P3.

For the N2, child-to-adult reductions in overall amplitude, latency and the nogo N2 effect are typically reported (Hämerer et al., 2010; Johnstone et al., 2005; Jonkman et al., 2003; Jonkman, 2006; Lamm et al., 2006). These reductions are thought to indicate increases in the efficiency of conflict monitoring or inhibitory processes, although there are also studies reporting no change and developmental increases in the nogo N2 effect. For the P3, developmental increases in the overall amplitude and the nogo P3 effect have been reported (Johnstone et al., 2005; Jonkman et al., 2003; Okazaki et al., 2004). Notably, the nogo P3 effect is absent in young children but is present in older groups with increasing magnitude, also manifesting as a posterior-to-anterior topographical shift on nogo trials. These increases in the nogo P3 effect are thought to indicate improvements in the ability to suppress responses.

However, these findings are largely derived from child-adult comparisons with few studies examining changes throughout adolescence into adulthood (Lamm et al., 2006; Lewis et al., 2006; Hämerer et al., 2010). A study by Lamm et al. (2006) examined changes in executive functioning and N2 in a single sample of children and adolescents (n = 33, 6-16 years). They reported linear age-related decreases in N2 amplitude that were associated with improvements in executive functioning. However, no changes in latency were reported and
the P3 was not examined. In a similar but larger study by Lewis et al. (2006) \((n = 58, 5-16\) years), a reduction in overall N2 amplitude was also observed, but it showed a curvilinear trajectory which reached a plateau by mid-adolescence (differences between 15-16-year-olds compared to 11-12 and 13-14-year old groups were non-significant) and no changes in nogo N2 effect were reported. This study examined P3 but reported a developmental reduction in the nogo P3 effect driven by a decrease to P3 on nogo trials but not go trials. Despite inconsistency with the child-adult literature, this effect was interpreted as an improvement the efficiency of the inhibitory process.

More consistent with the broader literature, Hämmerer et al., (2010) compared children \((n = 45, M = 10.15\) years), adolescents \((n = 44, M = 14.42\) years), younger \((n = 46, M = 24.27\) years) and older adults \((n = 46, M = 71.24\) years) using the Continuous Performance Task. They reported significant linear decreases in overall N2 amplitude and the nogo N2 effect from children to older adults. They reported curvilinear changes in P3 on go trials with smaller amplitudes in children and older adults (no difference between adolescents and adults), and a linear increase in P3 amplitudes on nogo trials. Collectively, general reductions in the N2 and increases in P3 are consistent with child-adult studies. However, there is considerable variability and little evidence documenting changes in N2 and P3 later in development, calling for more research to focus on the transition from adolescence to adulthood. For example, there is not enough evidence commenting on whether decreases in N2 followed a linear or curvilinear trajectory and direction of change in P3 appears more inconsistent due to the existence of very few studies.

**Reward processing and the feedback-related negativity**

Unlike response inhibition, there has been a greater focus on adolescence and the adolescent-adulthood transition in reward processing research using the ERP technique (Crowley et al., 2013; Grose-Fifer, Migliaccio, & Zottoli, 2014; Hämmerer, Li, Müller, & Lindenberger, 2011; Martínez-Velázquez, Ramos-Loyo, González-Garrido, & Sequeira, 2015; Santesso, Dzyundzyak, & Segalowitz, 2011; Zottoli & Grose-Fifer, 2012). These studies focus on examining modulations the feedback-related negativity (FRN) elicited to
different outcomes (e.g. the presentation and omission of reward or punishment) on risky decision-making tasks used in behavioural risk-taking studies.

The FRN refers to a frontocentrally distributed negativity that peaks around 200-350 ms following feedback presentation on tasks with uncertain outcomes. The FRN has been shown to be sensitive to feedback valence where amplitudes are enhanced following negative feedback. This will be referred to in the thesis as the FRN valence effect. Its amplitude is also sensitive to other feedback characteristics such as outcome likelihood and magnitude (Eppinger, Mock, & Kray, 2009; Sambrook & Goslin, 2015; Yi et al., 2012). The anterior cingulate cortex has also been estimated as its neural generator, like the N2 which has been implicated in conflict and performance monitoring (Crowley et al., 2013). Due to its sensitivity to several outcome characteristics and its association with the anterior cingulate cortex, the FRN is thought to be involved in performance monitoring based on external feedback, although its specific function is still debated.

According to the reinforcement-learning theory, the FRN reflects encoding of a reward prediction error signal by the anterior cingulate cortex (Holyord & Coles, 2002). The reward prediction error is the output of ongoing monitoring processes in the basal ganglia that signal differences between outcomes predicted by the system and actual outcomes. When outcomes are better than expected, a phasic increase in dopaminergic activity is elicited. Conversely, a phasic decrease in dopaminergic activity is elicited when the outcome is worse than predicted. It is proposed that these phasic changes inhibit/disinhibit neurons in the anterior cingulate cortex, giving rise to the FRN. Therefore, developmental changes to the dopaminergic system and functional changes in dopamine activity are thought to also be reflected in the FRN.

Development of the FRN

Developmental FRN studies mainly use two types of tasks, an unpredictable gambling task and a probabilistic learning task. In unpredictable gambling tasks, the likelihood of all outcomes is equal and are therefore unpredictable (e.g. Crowley et al., 2013; Martinez-Velaquez et al., 2015; Zottoli & Grose-Fifer, 2012; Santesso et al., 2011). In probabilistic learning tasks, responses are associated with specific outcomes with a degree of
uncertainty and participants are required to learn these response-outcome associations from external feedback provided on the trial (e.g. Eppinger et al., 2009; Grose-Fifer et al., 2014; Hämmerer et al., 2011). Across both tasks, studies typically report reductions in overall FRN amplitude from child- to adulthood, which were interpreted as reflecting a decrease in responsiveness to external feedback. Although these reductions are not always observed (Santesso et al., 2011).

Santesso et al., (2011) compared FRN in mid-to-late adolescents ($n = 20, M(age) = 16.4$ years) and young adults ($n = 20, M(age) = 21.7$ years) on an unpredictable gambling task. They reported a significant FRN valence effect but did not report any main effect or interactions with age group. Similarities in FRN were suggested to reflect maturation of feedback-related processes, although it was also argued that feedback in unpredictable gambling tasks is uninformative, contributing to absence of group differences. Although outcomes are exclusively revealed by the feedback stimulus, the unpredictable nature of the task means that the information expressed in feedback cannot be used to guide future responses meaningfully. Studies using probabilistic learning tasks have been used to examine FRN modulations to meaningful feedback and its association with risky decision-making behaviour. In line with findings from unpredictable gambling tasks, these studies report reductions in FRN reported, as well as developmental increases in the FRN valence effect during adolescence (Eppinger et al., 2009; Grose-Fifer et al., 2014; Hämmerer et al., 2011). Reductions in FRN amplitude were thought to reflect a shift in reliance on external to internal monitoring processes, and the increase in the FRN valence effect was interpreted as an increase in the ability to discriminate between feedback conditions.

In relation to personality and behaviour, significant associations with FRN have been reported in developmental studies. For personality, Santesso et al., (2011) observed relationships between FRN amplitude elicited in unpredictable gambling tasks and with reward and punishment sensitivity, where sensitivity to punishment was associated with overall enhanced FRN, and sensitivity to rewards was associated with an attenuation. However, they did not examine changes in these associations across age groups. In probabilistic learning tasks, For behaviour, Eppinger et al., (2009) has reported amplitude reduction in FRN to positive feedback with learning and reported that this decrease was less
pronounced in children. However, evidence from a recent study suggests that the learning component did not modulate developmental changes in FRN (Crowley et al., 2013). Although some developmental studies report associations between FRN and behaviour, these findings are infrequent and inconsistent.

Although evidence from unpredictable gambling tasks and probabilistic learning tasks have reported consistent developmental change in FRN, it is argued that these tasks lack ecological validity and lacks affective engagement, which may contribute to the lack of reported associations between the FRN and behaviour, attitudes and other personality factors. In risky decision-making studies have examined individual and developmental changes in behaviour on dynamic risk-taking tasks (Figner et al., 2009; Lejuez et al., 2002, 2007; Lejuez, Simmons, Aklin, Daughters, & Dvir, 2004). In dynamic risk-taking tasks such as the Balloon Analogue Risk Task (BART) and the Cambridge Card Task, participants make decisions where the magnitude and probability of outcomes change from trial-to-trial, such that magnitude and probability of negative outcomes accumulates across trials. These tasks are argued to better reflect the novelty of risk-taking situations in the real-world and are thought to be more affectively engaging. Supporting the validity of dynamic tasks, several studies have measures of risky decision-making to differentiate between risk and non-risk groups (Aklin, Lejuez, Zvolensky, Kahler, & Gwadz, 2005; Lejuez et al., 2002, 2003; Lejuez et al., 2004). Supporting its use in a developmental context, behavioural study by Figner et al., (2009) found that risky decision-making in adolescents were greater than adults only in the dynamic version of the task but not the static version. It was suggested that affective engagement of the task differentially engages motivational systems involved in decision-making manifesting as behavioural differences in risky decision-making.

Recent ERP studies in adults and at-risk populations have also examined changes in FRN using dynamic tasks (Fein & Chang, 2008; Kardos et al., 2016; Kiat, Straley, & Cheadle, 2016; Takács et al., 2015; Yau, Potenza, Mayes, & Crowley, 2015). In actively-drinking, treatment-naïve alcoholics, smaller FRN amplitudes were associated with greater family history of alcohol problems (Fein & Chang, 2008). In an anxious sample, risk-aversive behaviour was observed as well as reduced FRN amplitudes (Takács et al., 2015). In problematic internet users, overall FRN amplitudes were attenuated suggesting decreased
sensitivity to external feedback (Yau et al., 2015). However, current research has yet to examine changes in FRN during dynamic risk-taking during the adolescent-adult transition.

**Summary, Aims and Thesis Outline**

The neurodevelopmental model of risk-taking proposes that developmental differences in cognitive control and reward processing contributes to heightened risk-taking in adolescents, this model is supported by evidence of regional differences in the structural development of the brain and gross temporal changes in functional neuroimaging studies. Further investigation into specific neurocognitive processes using EEG and the ERP technique also provide evidence for improvement of inhibition and feedback processing. However, there are some gaps in developmental research and our understanding of the relationship between ERPs and behaviour is still unclear. With respect to response inhibition, there are many child-adult studies however there are limited studies examining the development of inhibition in adolescence and during the transition into adulthood. One reason could be because the response inhibition tasks are relatively simple and performance reaches ceiling before adulthood, making it difficult to examine changes in brain response. With respect to feedback processing, the association between FRN and behaviour hasn’t been observed consistently. The absence of significant brain-behaviour relationships could due to the predominant use of static risky decision-making tasks.

Using EEG and the ERP technique, this thesis aims to provide insight into the development of response inhibition and feedback monitoring, during the transition from adolescence to adulthood, and examine their role in risk-taking behaviour. The first study focuses on response inhibition and exploring of the use of a modified go/nogo task that detects partial inhibition in adults, and its potential utility for detecting developmental change during the adolescent-adult transition. Using this paradigm, the second study examines the development of response inhibition during the adolescent-adult transition using a cross-sectional design. The third study explores the use of a dynamic decision-making task to examine the development of reward processing during the adolescent-adult transition also using a cross-sectional design. While cross-sectional designs are less powerful than longitudinal designs, as there is additional between-subject variance, it is a cost-effective
method of providing insight into the development of these two systems which could be used to provide further support for a refined longitudinal investigation of the dual-systems model. The fourth study examines whether different levels of alcohol consumption are associated with differences in risky decision-making and underlying inhibition and feedback processing indices. Lastly, the general discussion will conclude the thesis with a summary and discussion of key findings providing suggestions for future research.
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Chapter 2: N2 and P3 Modulation during Partial Inhibition in a Modified Go/Nogo Task

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**Abstract**

The neural response following the partial inhibition of responses can provide insight into the processes underlying response inhibition. We examined the N2 and P3 on trials where participants correctly responded to go stimuli, successfully inhibited their response to nogo stimuli, and nogo trials where they initiated but did not complete their response (partial inhibitions) in an adult sample (N = 24, M(age) = 21.17, SD(age) = 3.52). An enhanced and delayed N2 was observed on partially inhibited compared to successfully inhibited nogo trials. Further analysis showed that this modulation was error-related. An enhanced central P3 was observed following successful inhibitions compared to correct go trials, but not following partial inhibitions. The results suggest that the central P3 enhancement is specific to the complete and successful inhibition of responses. Therefore, the absence of a central P3 on partial inhibitions could reflect insufficient inhibition or a monitored failure in inhibiting the response. Although, our findings provide support for the role of P3 in response inhibition, it raises questions about the processes involved in the subsequent inhibition or correction of the erroneous response. Further research examining the neural response following both partial and unsuccessful inhibitions could provide insight regarding these processes.
Introduction

Response inhibition refers to the capacity to suppress inappropriate but pre-potent responses, and is commonly studied using the go/nogo task. In the go/nogo task, participants rapidly respond to frequently presented go stimuli while withholding responses to infrequently presented nogo stimuli. Neuroimaging techniques such as functional magnetic resonance imaging (fMRI) and event-related potentials (ERPs) have been used to study the functional networks and processes underlying response inhibition. In fMRI studies, several brain regions have been implicated for response inhibition, such as the ventrolateral prefrontal cortex, the dorsolateral prefrontal cortex and the pre-supplementary motor area (Chikazoe, 2010; Crews, Braun, Hoplight, Switzer, & Knapp, 2000; Garavan, Ross, & Stein, 1999; Swick, Ashley, & Turken, 2011). The ERP technique has also been used to study response inhibition by examining the electrophysiological response during go and nogo trials, and its high temporal resolution allows researchers to explore the time-course of inhibitory processes. Typically, an enhanced frontocentral negativity occurring around 200–300 ms, as well as an enhanced central positivity occurring around 300–600 ms are observed following the presentation of a nogo stimulus (Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003; Van Veen & Carter, 2002). These peaks are referred to as the N2 and P3 respectively. The enhancements of these peaks are thought to be related to response inhibition, although the relationship between the two is still debated (Pires, Leitão, Guerrini, & Simões, 2014).

Initial observations lead to the conclusion that the N2 enhancement on nogo trials reflected inhibitory processing (Falkenstein, Hoormann, & Hohnsbein, 1999; Jodo & Kayama, 1992; Nakata et al., 2006). However, subsequent findings showed that N2 amplitude was sensitive to stimulus-related changes such as probability, and its enhancement was not exclusive to trials that required inhibition (Donkers & Van Boxtel, 2004; Fox, Michie, Wynne, & Maybery, 2000; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). It was proposed that the N2 reflects a conflict monitoring process which elicits a response when multiple incompatible representations are activated at the same time (Botvinick, Braver, Barch, Carter, & Cohen, 2001).
Using inverse modelling techniques, the anterior cingulate cortex has been identified as the main generator of the N2, and it is also recruited during interference suppression tasks where conflicting information is presented (Laird et al., 2005; Van Veen & Carter, 2002). The activity within the anterior cingulate cortex has also been estimated as the generator of the error-related negativity (ERN) which has been implicated in error monitoring (Dehaene, Posner, & Tucker, 1994). The involvement of the anterior cingulate cortex in conflict and error monitoring has also been confirmed in functional neuroimaging studies, and the co-localisation of the N2 and ERN to this region supported the role of N2 in conflict monitoring (Botvinick et al., 2001; Botvinick, Cohen, & Carter, 2004; Nieuwenhuis et al., 2003). However, additional generators of the N2 have also been identified, including the right ventral prefrontal cortex, the dorsolateral prefrontal cortex and the pre-supplementary motor area which have been implicated in response inhibition (Gonzalez-Rosa et al., 2013; Lavric, Pizzagalli, & Forstmeier, 2004). These findings were argued to support that the N2, in part, reflects response inhibition.

The centrally distributed P3 is an additional component that has also been implicated in response inhibition due to observed enhancements on nogo compared to go trials. The associations between the P3 and response inhibition were derived from investigations using modified tasks (such as the cued go/nogo task) which have observed that P3 amplitude was sensitive to trials which required inhibition (Randall & Smith, 2011; Smith, Johnstone, & Barry, 2007). In these studies, it was found that P3 was only enhanced when there was a planned response to be suppressed. In a comparison between go/nogo and go/GO tasks (where ‘GO’ refers to an infrequent condition which requires a response with maximal force), P3 amplitude was enhanced on nogo compared to go trials, but was not enhanced on GO trials which did not require inhibition (Donkers & Van Boxtel, 2004). Similarly, in a cued go/nogo task, P3 was not enhanced when there was no planned response to inhibit (nogo cue followed by a nogo stimulus) or when an unplanned response was executed (nogo cue followed by a go stimulus). P3 enhancements were only observed when participants were required to change responses or suppress a planned response (Randall & Smith, 2011; Smith et al., 2007).
To further explore the functional significance of the P3, researchers have investigated whether the P3 effect could be attributed to stimulus or response factors, and whether the P3 effect could be explained by a lack of motor potentials on nogo trials (Smith, 2011; Smith, Jamadar, Provost, & Michie, 2013; Smith, Johnstone, & Barry, 2008). In a cued go/nogo task, it was found that P3 was sensitive to both stimulus and response-related changes, but modulations were larger for response-related changes (Smith, 2011). In a comparison between motor and non-motor versions of the go/nogo task, differences between nogo but not go trials suggest that changes in P3 were related to inhibition rather than movement-related potentials (Smith et al., 2008).

Unsuccessful inhibition

Previous studies have focused on ERPs following the successful inhibition of responses as opposed to failures in inhibiting the response. Examining the neural response to the unsuccessful relative to successful inhibitions can provide additional insight into the functional significance of the N2 and P3. A number of studies have investigated the ERPs following successful and unsuccessful inhibitions, however there is a large focus on the stop-signal task (Bekker, Kenemans, Hoeksma, Talsma, & Verbaten, 2005; Chevalier, Kelsey, Wiebe, & Espy, 2014; Cragg, Fox, Nation, Reid, & Anderson, 2009; Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Ramautar, Kok, & Ridderinkhof, 2004, 2006; Roche, Garavan, Foxe, & O’Mara, 2005; Van Boxtel, Van der Molen, Jennings, & Brunia, 2001). In the stop-signal task, participants are asked to respond rapidly to frequently presented go stimuli, and withhold their responses on a small number of trials where go stimuli were followed by a stop-signal. Both the stop-signal and go/nogo tasks require response inhibition. However, the fundamental difference is that a go stimulus is presented prior to the stop-signal on stop trials, but is not presented at all on nogo trials. Although similar ERP patterns were found in a study comparing the go/nogo and stop-signal tasks (Van Boxtel et al., 2001), fMRI studies have reported differences in the involvement of common brain regions between the two tasks (Chikazoe et al., 2009; Chikazoe, Konishi, Asari, Jimura, & Miyashita, 2007). A meta-analysis comparing fMRI activation between tasks found that although there were common clusters, the two tasks engaged distinct neural circuits (Swick et al., 2011).
conceptual similarities between the go/nogo and stop-signal tasks, there is evidence that specific underlying processes differ between the two tasks.

In the go/nogo task, only one study has examined the ERPs following the successful and unsuccessful inhibition of responses (Roche et al., 2005). Compared to the successful inhibition of responses, they observed delayed N2 and P3 when inhibition was unsuccessful but did not observe any differences in amplitude. It was argued that the success of inhibition is largely determined by timing, where the failure to suppress a response is likely due to the delayed onset of inhibitory processes. It was thought that inhibitory processes must begin in key structures during a critical time period, if an attempt to suppress the motor response is to be successful (Garavan, Ross, Murphy, Roche, & Stein, 2002; Roche et al., 2005). The lack of research investigating ERPs following the unsuccessful inhibition of responses using the go/nogo task can be attributed to overall high accuracy, resulting in an insufficient number of unsuccessful trials required for ERP analysis.

Partial inhibition

As an alternative to examining unsuccessful inhibitions, researchers have modified the go/nogo task and examined ERPs following the partial inhibition of responses. In standard go/nogo tasks, only two outcomes are represented on nogo trials (successful or unsuccessful inhibitions). However, the process of suppressing a response is not an all-or-none process (Rubia et al., 2001). For instance, participants may have initiated a motor response following a nogo stimulus but were able to suppress the response before completion. Although these trials are categorised as successful inhibitions, they are not necessarily equivalent to nogo trials where the response was completely suppressed. These trials are referred to as partial inhibitions. Of interest to the current study, it has previously been shown that partial inhibitions accounted for a larger proportion of errors than unsuccessful inhibitions (Amieva et al., 2002; Chevalier et al., 2014; Cragg et al., 2009). It was suggested that taking partial inhibitions into account could improve the sensitivity of accuracy as index of performance, as well as provide insight into the processes underlying response inhibition (Cragg & Nation, 2008).
Partial inhibitions can be measured using a modified task with a release- and-respond mechanism, where responses are made by releasing the home key before pressing the target key. Nogo trials are categorised as partial inhibitions if the home key is released but the target key was not pressed. If the target key was also pressed, then the nogo trial would be categorised as an unsuccessful inhibition. Two studies in children have examined the neural response following the partial inhibitions using this paradigm (Chevalier et al., 2014; Cragg et al., 2009). In children aged 7 and 9 years, Cragg et al. (2009) observed delayed N2 following partial inhibition compared to successful inhibition, but did not observe differences in amplitude. Using N2 as an index of inhibitory processing, it was suggested that the processes involved in the partial and successful inhibition of responses were similar but were initiated earlier on successfully inhibited trials. With regards to P3, differences were difficult to interpret as the typical P3 effect was not elicited.

Chevalier et al. (2014) studied partial inhibitions in 5 year old children but did not measure the N2 and P3. They examined the lateral frontal negativity, and observed significant differences in latency which were thought to indicate differences in the timing of the action decision. Specifically, the lateral frontal negativity was delayed on partial inhibitions and they also found that response onset on unsuccessful inhibitions was earlier than standard go trials. It was suggested that the success of inhibition was influenced by the interaction between response inhibition and timing of the action decision, where early response initiation coupled with delayed action decisions is likely to result in the unsuccessful inhibition of the response. Although differences in the neural response following partial inhibitions have been reported in child samples, research has yet to examine ERPs following the partial inhibition of responses in adults where response inhibition processes have matured.

The current study aims to examine ERP components following the successful and partial inhibition of responses in a sample of adults. If the success of inhibition of responses is solely dependent on timing as suggested in previous studies using the go/nogo task (Chevalier et al., 2014; Cragg et al., 2009; Roche et al., 2005), on partially inhibited trials, we expect that components reflecting inhibition to be delayed but not differ in amplitude.
Methods

Participants

Undergraduate psychology students from the University of Western Australia and volunteers from the local community were recruited to participate in the study. The sample consisted of 24 participants aged between 17 and 29 years ($M = 21.17$, $SD = 3.52$, 11 males, 19 right handed). All participants provided informed written consent prior to participation and the protocol was approved by the human research ethics committee of the University of Western Australia.

Modified go/nogo task (Cragg & Nation, 2008)

The task was similar to the task used in Cragg and Nation (2008) and Cragg et al. (2009). The task was designed to be child-friendly, appearing as a game of soccer. A background consisting of green grass, blue sky and a soccer goal was constantly displayed on the screen. On each trial, either a soccer ball or a rugby ball was presented (Figure 2.1). For half of the participants, the soccer ball and the rugby ball represented the go and nogo stimulus respectively, and these pairings were reversed for the other half. Participants were asked to respond to go stimuli and withhold their responses to nogo stimuli.

The task had a release-and-respond mechanism where participants responded to go stimuli by releasing the home key then pressing the target key. On nogo trials, participants withheld their responses by keeping the home key held down. The left and right mouse buttons were assigned as the home and target keys respectively. Participants only used their right index finger to control both the home and target keys and were instructed to respond as quickly and as accurately as possible.

Correct responses to go stimuli (correct go) were identified when the participant released the home key and pressed the target key within 2000 ms of the stimulus onset. Successfully inhibited trials (successful inhibitions) were identified as nogo trials where participant did not release the home key. Partially inhibited trials (partial inhibitions) were identified as nogo trials where the participant released the home key but did not press the
target key. Lastly, unsuccessfully inhibited trials (unsuccessful inhibitions) were identified as nogo trials where the participant released the home key and pressed the target key.

Figure 2.1 Example of background stimulus (left), soccer ball (middle) and rugby ball (right).

Procedure

Instructions were presented on-screen and participants completed two practice blocks of 30 go trials to encourage the development of a pre-potent response. Each go and nogo stimulus was presented for 200ms. For these first two practice blocks, variable inter-stimulus intervals were used to prevent the prediction of stimulus onset and to discourage anticipatory responding. The inter-stimulus intervals varied randomly between 1600, 1800, 2200 and 2400 ms. Following the completion of the practice blocks, participants completed an additional two blocks, containing 75 go and 25 nogo trials in each block. To encourage a regular pattern of responding, the inter-stimulus interval between each stimulus presentation was fixed at 2000 ms. Throughout the task, participants were presented with encouraging feedback after each block. Participants also participated in an additional gambling task lasting for approximately 15 minutes which reported in Chapter 4.

Data acquisition

Electrophysiological data were acquired using SCAN 4™ and were processed offline using EEGLAB 13.3.2 (Delorme & Makeig, 2004) and ERPLAB 5.0.0 (Lopez-Calderon & Luck, 2014). The electrophysiological activity was continuously recorded using Ag/AgCl electrodes at 33 scalp locations (FP1, FP2, F3, F4, F7, F8, Fz, FC1, FC2, FC5, FC6, FCz, FT9, FT10, C3, C4, Cz, T7, T8, CP1, CP2, CP5, CP6, P3, P4, P7, P8, Pz, PO9, PO10, O1, O2).
O2, Iz). The online EEG was amplified using a NuAmps 40-channel amplifier, digitised at a sampling rate of 250 Hz and filtered online using a 0.05–30 Hz bandpass filter. The right mastoid electrode was set as the online reference and AFz was set as the ground. Electrodes were placed 2 cm above and below the left eye to record ocular movement.

The EEG data were filtered offline using a 0.5 Hz high-pass Butterworth filter (12 dB roll off). Ocular artifacts were corrected using independent components analysis and guided by SASICA to identify and remove components associated with ocular muscle activity (Chaumon, Bishop, & Busch, 2015). Channels with excessive noise were spherically interpolated, one channel was interpolated on the data of six participants, and two channels were interpolated on the data of two participants. EEG data were re-referenced offline to a common averaged reference.

Based on the PCA findings, both stimulus and response-locked data were analysed. Stimulus-locked epochs were extracted for correct go, successful inhibitions and partial inhibitions. Stimulus-locked epochs were segmented from 100 ms pre-stimulus onset to 1000 ms post-stimulus onset. Response-locked epochs were extracted for correct go and partial inhibitions. Response-locked epochs were time-locked to the release of the home key and were segmented from 400 ms pre-response to 700 ms post-response. For both stimulus and response-locked waveforms, the individual averaged waveforms were baseline-corrected around amplitude calculated over the pre-stimulus interval, as recommended in Luck (2014). Individual average waveforms were computed for each condition, and epochs with activity exceeding ±150 μV were excluded from these averages. The minimum number of epochs accepted into each participant's condition averages was five, and the average numbers of epochs included in the ERPs across participants were 145 (SD = 10) for correct go, 38 (SD = 6) for successful inhibitions and 9 (SD = 5) for partial inhibitions. Due to differences in trial numbers between conditions, the signal-to-noise ratio was largest on correct go followed by successful and partial inhibitions.

Measurement of event-related potentials

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1 Despite to differences in signal-to-noise ratio between correct go, successful inhibition and partial inhibitions, condition effects remained significant when matching on trial number.
On stimulus-locked waveforms, the N2 was measured at Fz, FCz, Cz, and P3 was measured at Fz, FCz, Cz, Pz. On response-locked waveforms, the CRN and ERN was measured at Fz, FCz and Cz. As peak detection methods are sensitive to noise artifacts, particularly for smaller peaks, mean amplitudes were used as the measure of amplitude. Mean amplitudes were calculated over a 40 ms window centred on the peak latencies of the grand-averaged waveforms. A short interval was used to minimise the interference of adjacent components. Due to latency jitter between conditions, separate latency windows were identified for each condition by averaging across the midline sites. This allowed for the comparison of amplitude across site while accounting for shifts in latency between conditions.

N2 mean amplitudes were measured over 240–280 ms on correct go, 228–268 ms on successful inhibitions, and 264–304 ms on partial inhibitions. P3 mean amplitudes were measured over 360–400 ms on correct go, 340–380 ms on successful inhibitions, and 520–560 ms on partial inhibitions. Peak latencies were only measured for N2 and were identified as the time-point at which the amplitude was most negative between 200 and 350 ms.

For response-locked waveforms, the ERN and correct-related negativity (CRN) were measured from the partial inhibition and correct go waveforms respectively at Fz, FCz, Cz. ERN and CRN mean amplitudes were computed over separate 40 ms window centred on the peak of the grand-averaged waveforms with intervals averaged across midline. ERN mean amplitudes were measured over 48–88 ms, and CRN was measured over 0–40 ms.

Data analysis

Preliminary analyses were conducted to ensure the data was appropriate for analysis. Four outliers were winsorized to 2.1 times the interquartile range (Hoaglin & Iglewicz, 1987). Skew and kurtosis coefficients were used to assess the normality of the data as outlined by Tabachnick and Fidell (2001). Skew and kurtosis coefficients were compared to a critical threshold of ±3.29 and these coefficients were derived by dividing skew and kurtosis values with their respective standard error values. The proportion of error on go trials was not normally distributed.
For behavioural data, a non-parametric Wilcoxon signed-rank test was used to compare the proportion of errors between go and nogo trials. A paired-sample t-test was used to compare the average median release time on correct go trials and partially inhibited nogo trials. For stimulus-locked components, N2 mean amplitude and peak latency were analysed using 3 × 3 repeated-measures ANOVAs with site (Fz, FCz, Cz) and condition (correct go, successful inhibitions, partial inhibitions) as within-subjects factors. P3 mean amplitude was analysed using a 4 × 3 repeated-measures ANOVA with site (Fz, FCz, Cz, Pz) and condition (correct go, successful inhibitions, partial inhibitions) as within-subjects factors. Greenhouse-Geisser corrected p-values were reported for effects where the assumption of sphericity was violated. Significant main effects were followed by paired-sample t-tests, and the sequential Holm-Bonferroni method was used to adjust for multiple comparisons (Holm, 1979). This method is more powerful than the Bonferroni approach while accounting for inflation of familywise type-1 error. Significant site by condition interactions, were followed by paired-sample t-tests or one-way ANOVAs and examining the within-subjects contrasts of site for each condition separately.

For response-locked components, CRN/ERN mean amplitude were analysed using a 2 × 3 repeated-measures ANOVA with condition (correct go, partial inhibition) and site (Fz, FCz, Cz) as within-subjects factors. To compare stimulus and response-locked ERPs on partial inhibitions, a 2 × 3 repeated-measures ANOVA was conducted with site (Fz, FCz, Cz) and component (ERN, N2) as within-subjects factors. ERN and N2 mean amplitude on partial inhibitions were also compared at Fz, FCz, Cz, using three Pearson's correlations.

Principal components analysis

To explore whether the observed N2 differences elicited in response to partial inhibitions could be due to additional error-related activity, we conducted temporal principal components analysis (PCA) using the ERP PCA toolkit (version 2.54, Dien, 2010). Due to latency shifts between conditions, separate PCAs were conducted for successful and partial inhibitions. Data from 100ms pre-stimulus to 750ms post-stimulus (213 data points) for 24 participants, at 33 scalp locations were included in the analysis (792 cases). The resulting case-to-variable ratio for each PCA was 3.71. Temporal PCA was conducted using the
covariance matrix with Kaiser normalisation, and the number of factors retained was determined by examining plots derived from the ERP PCA toolkit showing parallel tests comparing the scree of the dataset to the scree derived from a fully random dataset. For successful inhibitions, 10 factors were retained, explaining 92% of the total variance. For partial inhibitions, 14 factors were retained, explaining 88.12% of the total variance. N2 and P3 components were identified by inspecting microvolt-rescaled, factor loading waveforms of each factor derived from the ERP PCA toolkit, and the scalp distribution of these factors. Specifically, relevant factors were identified based on the peak latency, polarity and scalp distribution of the factors with respect to the ERP waveforms. Factors explaining 10% of the total variance were not interpreted. Interpreted factors are depicted in Figure 2.3.

**Results**

**Behavioural results**

Participants made errors on 1% of go trials and 21% of nogo trials. Unsuccessful and partial inhibitions accounted for 24% and 86% of nogo error trials respectively. The proportion of error on nogo trials (including both unsuccessful and partial inhibitions) was significantly larger than the proportion of error on go trials (nogo, $M = 0.21$, $SD = 0.10$; go, $M = 0.01$, $SD = 0.02$; $Z = -4.29$, $p < 0.001$). The home key was released significantly earlier on partial inhibitions than correct go trials, indicating that partial responses were initiated earlier (partial inhibitions, $M = 243$, $SD = 34$; correct go, $M = 270$, $SD = 32$; $t(23) = 3.70$, $p < 0.001$, $d = 0.75$).

**Stimulus-locked ERPs**

As can be seen on the scalp maps and waveforms depicted in Figures 2.2, the N2 peak occurring between 200 and 350 ms was frontocentrally distributed and P3 (300–700 ms) had a central to parietal distribution. Descriptive statistics for N2 and P3 mean amplitude, and N2 peak latency are represented in Table 2.1.
Table 2.1
Mean and standard deviation of N2, P3, CRN/ERN mean amplitude (μV), N2 peak latency (ms) at each site (Fz, FCz, Cz, Pz) for all conditions (correct go, successful inhibitions, partial inhibitions).

<table>
<thead>
<tr>
<th>Component</th>
<th>Site</th>
<th>Correct go</th>
<th>Nogo</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N2 mean amplitude</td>
<td>Fz</td>
<td>-3.09(2.49)</td>
<td>-4.07(3.25)</td>
<td>-6.06(3.61)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>-2.96(3.11)</td>
<td>-4.57(4.28)</td>
<td>-6.46(5.23)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>-1.09(3.34)</td>
<td>-2.78(4.60)</td>
<td>-4.31(5.80)</td>
<td></td>
</tr>
<tr>
<td>N2 peak latency</td>
<td>Fz</td>
<td>277(44)</td>
<td>262(33)</td>
<td>283(36)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>269(44)</td>
<td>249(26)</td>
<td>272(41)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>259(50)</td>
<td>242(24)</td>
<td>271(43)</td>
<td></td>
</tr>
<tr>
<td>P3 mean amplitude</td>
<td>Fz</td>
<td>-0.13(2.69)</td>
<td>3.00(3.55)</td>
<td>-0.70(2.87)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>0.34(2.41)</td>
<td>7.32(4.30)</td>
<td>1.51(2.91)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>0.72(2.22)</td>
<td>7.45(4.18)</td>
<td>3.45(2.89)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pz</td>
<td>1.98(2.87)</td>
<td>4.99(2.53)</td>
<td>3.69(2.88)</td>
<td></td>
</tr>
<tr>
<td>CRN/ERN mean amplitude</td>
<td>Fz</td>
<td>-3.60 (2.51)</td>
<td>-</td>
<td>-6.33 (3.06)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>-3.06 (2.91)</td>
<td>-</td>
<td>-7.89 (4.69)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>-0.95 (3.15)</td>
<td>-</td>
<td>-6.12 (5.14)</td>
<td></td>
</tr>
</tbody>
</table>
Figure 2.2 LEFT: Stimulus-locked and MIDDLE: response-locked grand-averaged waveforms of correct go (solid green line), successful inhibitions (dotted red line) and partial inhibitions (dashed purple line). Voltage (μV) is represented on the vertical axis and time (ms) is represented on the horizontal axis. RIGHT: Scalp distributions of the N2 and P3 on correct go, successful inhibition and partial inhibition, and CRN/ERN on correct go and partial inhibition (latency windows in Measurement of event-related potentials section).
**N2 mean amplitude**

Significant main effects were observed for site and condition (site, $F(2,46) = 8.79$, $p = .005, \eta^2_p = .276$; condition, $F(2,46) = 11.62$, $p < .001, \eta^2_p = .336$), but there was no statistically significant site by condition interaction ($F(4,92) = 0.73$, $p = .505, \eta^2_p = .031$). Follow-up $t$-tests for the main effect of site showed that N2 amplitude was maximal at frontal and frontocentral sites. Specifically, N2 amplitude at Fz and FCz were larger than Cz (Fz, $t(23) = -2.47$, $p = .021$, $d = 0.50$; FCz, $t(23) = -5.82$, $p < .001$, $d = 1.19$) but did not differ significantly between Fz and FCz ($t(23) = 0.60$, $p = .556$, $d = 0.12$).

Follow-up $t$-tests for the main effect of condition showed a typical nogo effect, where N2 amplitude was larger (more negative) on successful inhibitions compared to correct go trials ($t(23) = 3.08$, $p = .005$, $d = 0.63$). N2 amplitude on partial inhibitions was larger compared to correct go and successful inhibitions (correct go, $t(23) = 4.39$, $p < .001$, $d = 0.89$; successful inhibition, $t(23) = 2.34$, $p = .028$, $d = 0.48$).

**N2 peak latency**

Significant main effects were observed for site and condition (site, $F(2,46) = 9.26$, $p = .002, \eta^2_p = .287$; condition; $F(2,46) = 5.34$, $p = .008, \eta^2_p = .198$), but there was no statistically significant site by condition interaction ($F(4,92) = .37$, $p = .743, \eta^2_p = .016$). Follow-up $t$-tests for the main effect of site showed that N2 peak latency at Fz was later than FCz and Cz (FCz, $t(23) = 4.00$, $p = .001$, $d = 0.82$; Cz, $t(23) = 3.29$, $p = .003$, $d = 0.67$), but did not differ significantly between FCz and Cz ($t(23) = 1.67$, $p = .103$, $d = 0.34$).

Follow-up $t$-tests for the main effect of condition showed that N2 was delayed on partial inhibitions compared to successful inhibitions ($t(23) = 3.51$, $p = .002$, $d = 0.67$). However, N2 latency on correct go trials did not differ significantly from both successful and partial inhibitions after correcting for multiple comparisons (successful inhibitions, $t(23) = -0.87$, $p = .394$, $d = 0.18$; partial inhibitions, $t(23) = 2.21$, $p = .037$, $d = 0.45$).
**P3 mean amplitude**

Significant main effects of site and condition were observed (site, $F(3,69) = 14.64$, $p < .001$, $\eta_p^2 = .389$; condition, $F(2,46) = 44.29$, $p < .001$, $\eta_p^2 = .658$), as well as a significant site by condition interaction ($F(6,138) = 12.40$, $p < .001$, $\eta_p^2 = .350$). The site by condition interaction was followed by three one-way ANOVAs examining the effect of site for each condition separately. The pattern of results showed that P3 on successful inhibitions were centrally distributed. This was indicated by significant quadratic and cubic trends in the within-subjects contrasts (quadratic, $F(1,23) = 46.71$, $p < .001$, $\eta_p^2 = .670$; cubic, $F(1,23) = 8.41$, $p = .008$, $\eta_p^2 = .268$) and mean values which showed that P3 amplitudes were largest (most positive) at FCz and Cz. P3 on partial inhibitions were more posteriorly distributed. This was indicated by significant linear and quadratic trends in the within-subjects contrasts (linear, $F(1,23) = 19.75$, $p < .001$, $\eta_p^2 = .462$; quadratic, $F(1,23) = 4.03$, $p = .057$, $\eta_p^2 = .149$) and mean values which showed increasing P3 amplitude from Fz to Pz. P3 on correct go trials was also posteriorly distributed. Within-subjects contrasts revealed a significant linear trend ($F(1,23) = 4.50$, $p = .045$, $\eta_p^2 = .164$) and the mean values showed increasing amplitude from Fz to Pz. The results of these analyses are all consistent with the scalp distributions depicted in Figure 2.2.

**PCA results**

The microvolt-scaled time-course, distribution and the associated variance of PCA factors for successful and partial inhibitions are depicted in Figure 2.3. With regards to the N2, a single factor corresponding to the N2 was identified on successful inhibitions and two factors were identified on partial inhibitions. With regards to P3, a centrally distributed factor corresponding to the P3 was identified on successful inhibitions, whereas all factors corresponding to the P3 on partial inhibitions were parietally distributed. There was no centrally distributed factor on partial inhibitions.
Figure 2.3 Stimulus-locked, grand-averaged waveforms and waveforms of microvolt-scaled factor loadings on successful and partial inhibitions, as well as the scalp topography, percentage of total variance explained, and the peak latency of loadings for each factor.

Response-locked ERPs

To explore the suggestion that the additional factor identified in the temporal PCA of the partial inhibition waveforms (See Principal components analysis section) reflects the superimposition of an ERN peak coinciding with the detection and interruption of the response in this condition, further analyses were conducted on the response-locked ERPs. Response-locked waveforms are depicted Figure 2.2 where an enhanced ERN is elicited following a partial response.

CRN/ERN mean amplitude

A significant main effect of condition was observed, indicating an enhanced negativity was elicited following partial responses compared to correct go responses ($F(1,23) = 23.11, p < .001, \eta_p^2 = .501$). A significant main effect of site and significant site by condition interaction were also observed, suggesting differences in distribution across
conditions (site, $F(2,46) = 8.73, p = .004, \eta^2_p = .275$; site by condition; $F(2,46) = 9.93, p = .002, \eta^2_p = .302$).

The site by condition interaction was followed by two one-way ANOVAs, examining the effect of site for correct go and partial inhibitions separately. The pattern of results showed that ERN on partial inhibitions was frontocentrally distributed. This was indicated by a non-significant linear trend and a significant quadratic trend in the within-subjects contrasts (linear, $F(1,23) = .05, p = .820, \eta^2_p = .002$; quadratic, $F(1,23) = 34.55, p < .001, \eta^2_p = .600$) and mean values which showed that ERN amplitude was largest (more negative) at FCz. On the other hand, CRN was more frontally distributed. This was indicated by significant linear and quadratic trends (linear, $F(1,23) = 22.47, p < .001, \eta^2_p = .494$; quadratic, $F(1,23) = 30.97, p < .001, \eta^2_p = .574$), and mean values showing larger CRN amplitudes at Fz and FCz. The results of these analyses are all consistent with the scalp distributions depicted in Figure 2.2.

**Stimulus-locked vs. response-locked ERPs**

Stimulus and response-locked ERPs were compared to investigate whether the observed N2 differences on partial inhibitions are response-related. If this activity is response-related, it should be more synchronised to the response and manifest as a larger peak on the response-locked ERP waveforms. The results showed a significant main effect of component, indicating that ERN amplitude was larger (more negative) than N2 amplitude ($F(1,23) = 7.63, p = .011, \eta^2_p = .249$).

A significant main effect of site and a significant site by condition interaction were also observed, suggesting some differences in distribution of the N2 and ERN (site, $F(2,46) = 4.12, p = .046, \eta^2_p = .152$; site by condition, $F(2,46) = 10.58, p = .001, \eta^2_p = .315$). Follow-up $t$-tests showed that ERN had a frontocentral maximum, where ERN amplitude at FCz was larger than Fz and Cz (Fz, $t(23) = -2.65, p = .014, d = 0.54$; Cz, $t(23) = -3.68, p = .001, d = 0.75$). On the other hand, N2 on partial inhibitions had a frontal to frontocentral maximum, where N2 amplitude at FCz was larger than Cz but did not differ significantly from Fz (Cz, $t(23) = -4.55, p < .001, d = 0.93$; Fz, $t(23) = 0.64, p = .530, d = 0.13$).
To investigate whether N2 activity on partial inhibitions can be attributed to the ERN, we conducted three Pearson’s correlations comparing ERN and N2 amplitude on partial inhibitions at Fz, FCz, Cz. We observed significant and strong correlations at all sites (Fz, \( r(22) = .85, p < .001 \); FCz, \( r(22) = .884, p < .001 \); Cz, \( r(22) = .90, p < .001 \)).

**Discussion**

We investigated the functional significance of the N2 and P3 by examining the ERPs following the partial inhibition of responses in a modified go/nogo task. We hypothesised that if response inhibition was dependent solely on timing (Cragg et al., 2009; Roche et al., 2005), then ERP components related to inhibition would be delayed on partial inhibitions, but would not differ in amplitude. However, we observed substantial differences in the neural response following successful and partial inhibitions, indicating that the inhibitory processes underlying each condition may not be equivalent processes that were delayed in onset. Both the N2 and P3 components showed significant differences between successful and partial inhibitions, and changes in the N2 were distinct from changes in the P3.

*N2 on partial inhibitions reflects error-monitoring*

Our results showed a delayed and enhanced N2 following partial inhibitions compared to successful inhibitions. As the N2 in both conditions were elicited by the same stimulus, it is likely that these modulations are response-related. From a response inhibition perspective, this could reflect a delayed onset and increased engagement of inhibitory processes when inhibiting an ongoing response, compared to an unexecuted response. From a conflict monitoring perspective, response conflict generated on partial inhibitions could be greater than successful inhibitions. A prepared motor response could be harder to suppress and generate more conflict than a less-prepared response. Consistent with this idea, we observed that participants released the home key earlier on partial responses compared to correct go trials, suggesting the partial responses were prepared earlier. Although these hypotheses are plausible, the analysis of response-locked ERPs suggests that N2 modulation on partial inhibitions reflects error-related processing of the partial response.
The involvement of error-related processes on partial inhibitions were revealed by the temporal PCA which identified two factors corresponding to the N2 for partial inhibitions but only a single factor on successful inhibitions. To investigate whether this additional factor was related to error-monitoring, we compared the ERN to the PCA factor loadings as well as the N2. First, we observed a significant ERN following partial inhibitions, indicating that participants detected partial responses as errors. Second, we found that the timing of the ERN coincides with the peak latency of the additional PCA factor, when considering both the peak latency of the ERN and the average release time. Third, we compared ERN and N2 mean amplitude and observed that ERN amplitude was larger than the N2, indicating that the negativity was more synchronised to the response. Finally, Pearson’s correlations showed a very strong and significant positive correlation between ERN and N2 amplitude across the frontocentral midline sites. Collectively, these findings suggest that the modulation of N2 on partial inhibitions reflects error monitoring.

P3 enhancement is specific to completely suppressed responses

The P3 has been implicated in response inhibition based on observations of specific enhancements on trials which required inhibition (Donkers & Van Boxtel, 2004; Randall & Smith, 2011; Smith, 2011; Smith et al., 2007; Smith et al., 2008). We observed enhanced central P3 amplitudes following successful inhibitions but not following the partial inhibitions. Given that successful and partial inhibitions were elicited by the same stimulus, it is unlikely that observed P3 differences were due to stimulus-related factors such as novelty. We argue that the specific enhancement of central P3 on successful inhibitions reflects the complete inhibition of the response. Therefore, the absence of a central P3 on partial inhibitions could indicate the absence of inhibition-related processes. However, this interpretation raises questions about the subsequent suppression or correction of the ongoing responses, and if these processes are reflected in the P3. Alternatively, the absence of central P3 could reflect the monitored failure in inhibiting the response, which is in line with the observed ERN.

There were some discrepancies in the P3 results between our study and Roche et al. (2005) which examined the neural response to successful and unsuccessful inhibitions. We
observed substantial differences in the P3 amplitude and distribution, whereas they only reported differences in latency. The discrepancy between studies could be due to differences in processes underlying partial and unsuccessful inhibitions, as well as task differences. In our study, the stimulus and response pairs were pre-determined (i.e. the go stimulus was always associated with a specific stimulus) but they were not pre-determined in Roche et al. (2005). In the X-Y go/nogo task used in Roche et al. (2005), go and nogo trials were indicated based on context: go trials were indicated by a change in stimulus, and nogo trials were indicated by a repeating stimulus. The discrepancy between studies could be related to differences in context-updating processes, as the P3 has also been implicated in the orienting of attention and context-updating processes (Polich, 2007).

Motor-related activity is another potential factor which could account for some P3 differences. Numerous researchers have investigated the impact of motor-related activity by comparing ERPs elicited during motor and non-motor versions of the go/nogo task. Although some differences in the P3 effect between motor and non-motor tasks have been reported, overall, the findings indicate that motor-related activity cannot completely account for the P3 effect (Bruin & Wijers, 2002; Burle, Vidal, & Bonnet, 2004; Nakata et al., 2004; Smith et al., 2013, 2008; Van’t Ent & Apkarian, 1999). Although we cannot completely discount the role of motor-related activity, based on previous findings, it is unlikely the absence of central P3 is entirely explained by motor-related activity.

Partial inhibition and the stop-signal task

Only a few studies have examined partial and unsuccessful inhibitions using the go/nogo task, whereas numerous studies have investigated this using stop-signal task (e.g. De Jong, Coles, Logan, & Gratton, 1990; Kok et al., 2004; Ramautar et al., 2004, 2006). Although both tasks have been used to study response inhibition, there are key differences between the two tasks and their engagement of common brain regions (Swick et al., 2011). Due to these differences, the degree to which the findings can be compared is unclear. Nevertheless, we attempt to compare our findings with stop-signal studies.

One of the initial investigations exploring partial responses in the stop-signal task used ERPs, electromyography (EMG), and force transducers to measure cognitive, muscular
and behavioural aspects of the response (De Jong et al., 1990). Contrary to our findings, partial responses only accounted for a minor proportion of errors on stop-trials, and response onset did not differ between partial and standard go responses (derived from EMG and transducer activity). With regards to the electrophysiological response, a similar central P3 enhancement was observed on successfully inhibited stop-trials. However, they only examined the neural response to successful stop-trials and not partial or unsuccessful stop-trials.

Compared with subsequent stop-signal studies, there were similarities in both behaviour and electrophysiology. Behaviourally, faster response times were observed on unsuccessful stops compared to standard go responses (Kok et al., 2004; Ramautar et al., 2004; 2006). With respect to the N2, an enhanced and delayed N2 was observed following unsuccessful inhibition, and this was attributed to error-related processing (Kok et al., 2004; Ramautar et al., 2004). Although, one study did attribute this to the processing of the stop-signal stimulus, after observing a stronger response on stimulus-locked compared to response-locked waveforms (Ramautar et al., 2004). With respect to the P3, similar changes in the P3 distribution from successful and unsuccessful stop trials were observed. P3 to successfully inhibited stop trials showed a frontocentral distribution, while P3 to unsuccessful inhibitions was more posteriorly distributed (Kok et al., 2004; Ramautar et al., 2004; 2006). Contrary to our findings, P3 amplitudes were enhanced on unsuccessful compared to successful inhibitions, although this enhancement originated from the parietal region. Furthermore, it was found that this enhanced parietal positivity was more prominent when synchronised to the response (Ramautar et al., 2004). It was suggested that the enhanced parietal P3 to unsuccessful trials was response-related and reflected monitoring or evaluation of the erroneous response. However, due to the presence of an overall large parietal P3, it was difficult to observe changes in the central P3. Discrepancies in the relative size of the parietal P3 between studies could be due to task-specific factors, or differences in the processes underlying partial and unsuccessful inhibitions.
Conclusions and future research

The current study is the first to examine ERPs following the partial inhibition of responses in adults, and showed distinct modulation of the N2 and P3 following partial inhibitions. We showed that differences in N2 on partial inhibitions were attributed to an additional error-monitoring process and observed that central P3 enhancement was specific to successful inhibitions, where the response was completely suppressed. Our results provide support for the role of P3 in response inhibition, either as an index of inhibitory processes or the monitoring of its success. The interpretation of P3 as an index of inhibitory processing raises questions about the processes involved in the subsequent suppression or correction of the ongoing response. Further investigation examining N2 and P3 effects following partial inhibitions and unsuccessful inhibitions could provide additional insight regarding these processes, and whether the processes underlying partial and unsuccessful inhibitions are distinct.
References


Chapter 3: Partial inhibition reveals differences during response inhibition between mid-to-late adolescents and young adults

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Abstract

Numerous studies have examined the development of response inhibition using the go/nogo task. However, few investigations have used this task study changes during the adolescent-adult transition. To investigate the development of response inhibition during this period, we compared mid-to-late adolescents \((N = 28, M(SD)_{age} = 15.74(1.06) \text{ years})\) and young adults \((N = 22, M(SD)_{age} = 22.02(3.17) \text{ years})\) on an error-sensitive modification of the go/nogo task which can detect partial inhibitions - when participants initiate, but do not complete, responses to nogo trials. The groups were compared on measures of task performance (proportion of error, response time) and event-related potentials (ERPs) elicited to go and nogo stimuli (P2, N2, P3). The results showed a higher proportion of false alarm errors in adolescents, which reduced with age only in the adolescent group. Although similarities in N2 and P3 amplitude suggest that conflict monitoring and inhibitory processes are relatively mature by mid-to-late adolescence, delayed P2 latencies in the adolescent group indicated that there were differences in early attentional processing. As opposed to an immaturity of inhibitory processing, our data suggests that poorer accuracy in adolescents may be driven by an inability to compensate for slower stimulus processing when responding.
Introduction

Adolescence marks a critical period of development where substantial biological, behavioural and cognitive changes occur. This period has been characterised by an increase in risk-taking, with several studies reporting elevated levels of risk-taking in adolescents compared to adults (Defoe, Dubas, Figner, & Aken, 2015). Several neurodevelopmental models of risk-taking suggest that this is partly attributed to the prolonged development of the ability to suppress rewarding but potentially hazardous impulses (Shulman et al., 2016). The capacity to suppress pre-potent but inappropriate responses is referred to as response inhibition and it is commonly studied using the go/nogo task, where participants rapidly respond to frequently presented go stimuli while withholding responses to infrequently presented nogo stimuli. Response inhibition is primarily associated with the functioning of the prefrontal cortex, but is also associated with a distributed network involving the striatum, parietal and temporal lobes, and other structures such as the pre-supplementary motor area (Chikazoe, 2010; Garavan, Ross, & Stein, 1999; Liddle, Kiehl, & Smith, 2001; Swick, Ashley, & Turken, 2011).

With respect to brain development, significant changes to the structure of the prefrontal cortex occurs during adolescence (Giedd, 2004; Gogtay et al., 2004; Huttenlocher & Dabholkar, 1997; Paus et al., 1999; Sowell, Thompson, Holmes, Jernigan, & Toga, 1999; Sowell, Thompson, & Toga, 2004), and as these changes continue, further development of the associated cognitive functions are also expected. Using the event-related potential (ERP) technique and the go/nogo task, numerous studies have examined the development of response inhibition from childhood to adulthood, reporting significant changes in task performance and the neural response. However, these studies largely focus on contrasts between children to adult groups, and finer changes throughout childhood (Ciesielski, Harris, & Cofer, 2004; Cragg, Fox, Nation, Reid, & Anderson, 2009; Cragg & Nation, 2008; Hammerer, Li, Muller, & Lindenberger, 2010; Johnstone et al., 2007; Johnstone, Pleffer, Barry, Clarke, & Smith, 2005; Jonkman et al., 2003; Jonkman, 2006; Lamm, Zelazo, & Lewis, 2006; Lewis, Lamm, Segalowitz, Stieben, & Zelazo, 2006; Okazaki et al., 2004); and relatively few studies have used the go/nogo task to examine the development of response
inhibition during adolescence, particularly during the transition from adolescence to adulthood (Hämmerer et al. 2010; Lewis et al. 2006).

*The development of event-related potentials in the go/nogo task*

Developmental studies examining changes in ERPs elicited in the go/nogo task typically focus on the frontocentral N2 and central P3 peaks. The amplitudes of these peaks are typically enhanced on nogo compared to go trials, and these enhancements (referred to as the nogo N2 and P3 effects) were thought to reflect inhibition-related processing. Although the N2 was initially implicated in response inhibition, subsequent studies have shown that the enhancement of the N2 was not exclusive to trials that required inhibition (Donkers & Van Boxtel, 2004; Fox, Michie, Wynne, & Maybery, 2000; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). Alternatively, it was proposed that N2 could reflect conflict monitoring, a process that detects when multiple incompatible representations are simultaneously activated (Botvinick, Braver, Barch, Carter, & Cohen, 2001). On the other hand, central P3 enhancements were more specific to trials that required response inhibition. Using a cued go/nogo task, P3 was not enhanced when there was no planned response to inhibit (nogo cue followed by a nogo stimulus) or when an unplanned response was executed (nogo cue followed by a go stimulus). Enhancements were only observed when participants were required to change responses or suppress a planned response (Randall & Smith, 2011; Smith, 2011; Smith, Johnstone, & Barry, 2007; Smith, Johnstone, & Barry, 2008). As such, the enhancement of P3 amplitude in inhibition trials was thought to reflect the inhibitory process.

With respect to the development of conflict monitoring, several studies have reported developmental reductions in N2 amplitude from childhood to adulthood, reflecting improvements in the efficiency of conflict monitoring processes (Brydges, Anderson, Reid, & Fox, 2013; Hämmerer et al., 2010; Johnstone et al., 2005; Jonkman, 2006; Lamm et al., 2006; Lewis et al., 2006). During adolescence, studies have reported marginal group differences and linear trends indicating a reduction in overall N2 amplitude and the nogo N2 effect (Hämmerer et al., 2010; Lewis et al., 2006). The reduction in the nogo N2 effect was interpreted as a developmental decrease in conflict experienced.
Similarly, developmental reductions in N2 peak latency have also been reported (Brydges et al., 2013; Johnstone et al., 2005; Lamm et al., 2006; Lewis et al., 2006). Brydges et al. (2013) and Johnstone et al. (2005) reported reduced N2 latency in adults compared to children, and Lamm et al. (2006) and Lewis et al. (2006) also observed age-related reductions in children and adolescents. These reductions were also interpreted as reflecting improvements in the efficiency of conflict monitoring processes, possibly due to the continued myelination occurring throughout childhood and adolescence (Cardenas et al., 2005; Picton & Taylor, 2007).

With respect to the development of response inhibition reflected by the P3, developmental increases in the nogo P3 effect from childhood to adulthood have been reported. A few studies have reported that the nogo P3 effect was absent in young children but was observed in older age groups with increasing magnitude, and this increase was thought to reflect an improvement in the ability to suppress responses (Hämmerer et al., 2010; Jonkman et al., 2003; Jonkman, 2006). In adolescents, Hämmerer et al. (2010) reported a significant linear trend showing an increase in P3 amplitude to nogo trials across the lifespan (children, adolescents, adults and older adults). Contrasting with other findings, Lewis et al. (2006) reported developmental decreases in P3 amplitude throughout childhood and adolescence.

In addition to the N2 and P3, ERP studies using the go/nogo task have also examined developmental changes in the P2 (Johnstone et al., 2007, 2005). Similar to changes in the N2, developmental reductions in P2 amplitude from childhood to adulthood have been reported (Johnstone et al., 2005). The P2 has been extensively examined in the oddball paradigm as well as other cognitive control tasks, and it is generally thought to reflect early exogenous processing of the stimulus and the orientation of attention to the stimulus. It has been reported that P2 amplitude to infrequent stimuli in the oddball task were modulated depending on whether it was described as the target or non-target (Potts, Liotti, Tucker, & Posner, 1996). Similarly, changes in P2 amplitude were observed to various features such as colour, size and orientation of stimuli when they were specified by task instructions as being significant (Luck & Hillyard, 1994). As such, it has been hypothesised that modulations in P2 may reflect top-down processes that facilitate the orientation of attention and processing.
of certain features which have been identified as having potential significance (Daffner, Alperin, Mott, Tusch, & Holcomb, 2015). Reported developmental reductions in P2 latency could reflect faster orienting towards the stimulus and reductions in amplitude may indicate improvements in the efficiency of early stimulus-related processes.

Overall, studies examining the development of response inhibition using ERPs have reported significant differences between children and adults, as well as significant age-related changes on several indices supporting the ongoing maturation of conflict monitoring, inhibitory and early stimulus processing. However, there is little evidence regarding the development of these processes during adolescence and during the adolescent-adult transition.

Current Study

The current study focused on examining response inhibition during the transition from adolescence to adulthood. To detect subtle changes in task performance during this period, an error-sensitive modification of the go/nogo task was used. This task detects partial inhibitions, which refers to an initiated, but incomplete response to a nogo trial. To examine the underlying processes ERPs elicited following correctly identified go trials and successfully inhibited nogo trials were measured. If response inhibition continues to develop throughout this adolescent period, we expected to observe developmental improvements in accuracy and/or a reduction in the speed of responses. If the underlying attentional, conflict monitoring or inhibitory processes continue to mature, we expect these maturational changes to be reflected by the P2, N2 and P3 respectively. In line with previous research, we predicted that a developmental reduction in the amplitude and latency of the P2 and N2 would be observed. We also predicted that an increase in the nogo P3 effect would be observed, indicating an improvement in the ability to suppress the response.

Methods

Participants

The sample consisted of 28 adolescents aged between 14 and 17 years (\(M(SD)_{age} = 15.74 \ (1.06) \) years, 16 males, 26 right-handed) and 22 adults aged between 18 and 28 years
The data of adult participants were also reported in Chapter 2. Participants were recruited from local high schools, psychology undergraduates from the University of Western Australia, and volunteers from the local community. Adult participants and a parent/legal guardian of adolescent participants provided informed consent prior to participation and the protocol was approved by the human research ethics committee of the University of Western Australia.

**Modified Go/Nogo Task (Cragg & Nation, 2008).**

This task was originally designed by Cragg and Nation (2008) and a modification by Nguyen, Moyle and Fox (2016) was used in the current study to counterbalance the specific stimuli associated with go and nogo responses. Throughout the task, a soccer goal was constantly displayed on a green and blue background. On each trial, either soccer ball or a rugby ball was presented (Figure 3.1). For half of the participants, the soccer ball and the rugby ball represented the go and nogo stimulus respectively, and these pairings were reversed for the other half. Participants were instructed to respond to go stimuli and withhold their responses to nogo stimuli.

The response device (a mouse) incorporated a release-and-respond mechanism where participants responded to go stimuli by releasing the left-mouse button then pressing the right-mouse button. Participants were instructed to use their right index finger to control both the buttons and to respond as quickly and as accurately as possible. On nogo trials, participants withheld their responses by keeping the left-mouse button held down.

Correct responses to go stimuli (correct go) were identified when the participant released the left-mouse button and pressed the right-mouse button within 2000 ms of stimulus onset. Although participants had 2000 ms to respond, all response times were below 1000 ms. Successfully inhibited nogo trials (successful inhibitions) were identified as nogo trials where the participant did not release the left-mouse button. Partially inhibited nogo trials (partial inhibitions) were identified as nogo trials where the participant released the left-mouse button but did not press the right-mouse button. Lastly, unsuccessfully inhibited nogo trials (unsuccessful inhibitions) were identified as nogo trials where the participant released the left-mouse button and pressed the right-mouse button.
Procedure

Instructions were presented on screen and participants completed two practice blocks of 30 go trials to encourage the development of a pre-potent response. Each go and nogo stimulus was presented for 200 ms. For these first two practice blocks, variable inter-stimulus intervals were used to prevent the prediction of stimulus onset and to discourage anticipatory responding. The inter-stimulus intervals were selected randomly between 1600, 1800, 2200 and 2400 ms. Following the completion of the practice blocks, participants completed an additional two blocks, containing 75 go and 25 nogo trials in each block. To encourage a regular pattern of responding, the inter-stimulus interval was fixed at 2000 ms. Throughout the task, participants were presented with encouraging feedback after each block. Participants also participated in an additional gambling task lasting for approximately 15 minutes which reported in Chapter 4.

Data acquisition

EEG data were acquired using SCAN 4™ and processed offline using EEGLAB 13.3.2 (Delorme & Makeig, 2004) and ERPLAB 5.0.0 (Lopez-Calderon & Luck, 2014). The data were recorded using Ag/AgCl electrodes at 33 scalp locations (FP1, FP2, F3, F4, F7, F8, Fz, FC1, FC2, FC5, FC6, FCz, FT9, FT10, C3, C4, Cz, T7, T8, CP1, CP2, CP5, CP6, P3, P4, P7, P8, Pz, PO9, PO10, O1, O2, Iz). Additional electrodes were placed at the left and right mastoid regions, where the right mastoid electrode was set as the online reference. Electrodes were also placed 2 cm above and below the left eye to record ocular movement, and an electrode placed at AFz was set as the ground. The online EEG was amplified using a
NuAmps 40-channel amplifier, digitised at a sampling rate of 250 Hz and filtered online using a 0.05 – 30 Hz bandpass filter.

The EEG data filtered offline using a 0.5 Hz high-pass Butterworth filter (12 dB roll-off). Ocular artifacts were corrected using independent components analysis and guided by SASICA to identify and remove components associated with ocular muscle activity (Chaumon, Bishop, & Busch, 2015). EEG data were re-referenced offline to a common averaged reference. Channels with excessive noise were spherically interpolated. One channel was interpolated for twelve participants, two channels for two participants, and three channels for one participant. Stimulus-locked epochs were extracted for correct go and successful inhibitions, segmented from 100 ms pre-stimulus onset to 1000 ms post-stimulus onset. Epochs with activity exceeding ±150 μV were excluded from the individual grand-averages. Individual averaged waveforms were baseline-corrected to the mean amplitude over the pre-stimulus interval, as recommended in Luck (2014).

Individual average waveforms were computed for correct go and successful inhibition trials. ERP data for partial inhibitions and unsuccessful inhibitions were not extracted due to an insufficient number of trials. On correct go, the average number of epochs included in the grand-averaged ERPs were 145 (SD = 6) and 144 (SD = 10) for adolescents and adults respectively. For successful inhibitions, an average of 29 (SD = 9) and 38 (SD = 6) epochs were included. The number of epochs on successful inhibitions were significantly larger in adults than adolescents \( t(48) = -0.37, p = .001, d = 1.16 \) but did not differ on correct go trials \( (t(48) = 0.15, p = .882, d = 0.04) \)

Measurement of Event-Related Potentials

To examine whether differences in epoch number resulted in greater noise in the adolescent group, we compared noise in the EEG between adolescents and adults, as measured by the standard deviation of EEG amplitude in the baseline interval. A mixed ANOVA was conducted with intended sites of interest (Fz, FCz, Cz, Pz) and age group (adolescents, adults) as within- and between-subject factors. The results showed significant main effect of age group and an age group × site interaction. Follow-up independent samples \( t \)-tests were conducted, controlling for multiple comparisons using the Holms-Bonferroni method. The results only showed a statistically significant difference in noise at Pz where noise was greater in the adolescent group. As such, Pz was excluded from further analyses.
The amplitude of the P2, N2 and P3 were measured at Fz, FCz, and Cz. Mean amplitudes were calculated over a 40 ms window centred on peaks of the grand-averaged waveform. This short interval was used to minimise the interference of adjacent components. The P2 grand-averaged peak was identified as the most positive point between 130-230 ms post-stimulus onset, the N2 peak as the most negative point between 200-350 ms, and the P3 as the most positive point between 250-450 ms. Using the same intervals, P2 and N2 peak latencies were measured for each participant. Peak latencies were not analysed for P3 as peaks on correct go trials were not prominent and as such, are particularly sensitive to noise.

To control for potential latency variability on mean amplitude across conditions as well as age group, we computed separate latency windows for each condition and age group. For each condition and age group, ERP amplitudes at each site were measured using the same interval. This was achieved by averaging the identified windows across the extracted midline sites. This allowed us to capture the distribution of each component while accounting for shifts in latency across conditions and age group. The latency windows used to measure ERP mean amplitudes are specified in Table 3.1.

Table 3.1

<table>
<thead>
<tr>
<th>ERP</th>
<th>Condition</th>
<th>Age group</th>
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<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Adolescent</td>
<td>Adult</td>
</tr>
<tr>
<td>P2</td>
<td>Correct go</td>
<td>160-200 ms</td>
<td>140-180 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Successful inhibition</td>
<td>160-200 ms</td>
<td>140-180 ms</td>
<td></td>
</tr>
<tr>
<td>N2</td>
<td>Correct go</td>
<td>240-280 ms</td>
<td>240-280 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Successful inhibition</td>
<td>244-284 ms</td>
<td>232-272 ms</td>
<td></td>
</tr>
<tr>
<td>P3</td>
<td>Correct go</td>
<td>368-408 ms</td>
<td>360-400 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Successful inhibition</td>
<td>376-416 ms</td>
<td>340-380 ms</td>
<td></td>
</tr>
</tbody>
</table>

*Data analysis*

Preliminary analyses were conducted to ensure the data were appropriate for analysis. Fourteen outliers were identified across all variables and participants, and these outliers were winsorized to 2.2 times the interquartile range (Hoaglin & Iglewicz, 1987). All variables
except for the proportion of error on go trials were normality distributed, as assessed by examining skew and kurtosis coefficients against critical thresholds of |2| and |7| (Curran, West, & Finch, 1996). Due to a ceiling effect, the proportion of errors on go trials was not analysed further.

For behavioural data, the proportion of error on nogo trials (i.e. proportion of partial and unsuccessful inhibitions relative to all nogo trials) in adolescents and adults were compared using an independent samples t-test. To examine whether age group differences in the proportion of error was related to age, Pearson’s correlations between age and the proportion of error were conducted for adolescents and adults separately. Median release times in adolescents and adults were compared using an independent samples t-test.

P2, N2 and P3 mean amplitude, and P2 and N2 peak latency were analysed using 3×2×2 mixed ANOVAs with site (Fz, FCz, Cz) and condition (correct go, successful inhibition) and as within-subject factors, and age group (adolescent, adult) as the between-subjects factor. Greenhouse-Geisser corrected p-values were reported for effects where the assumption of sphericity was violated. Significant main effects of site and condition were followed by paired-sample t-tests. For follow-up t-tests, the sequential Holm-Bonferroni method was used to adjust for the accumulation of type-I error in multiple comparisons (Holm, 1979). Significant site × condition or site × age group interactions were followed by one-way ANOVAs and examining within-subject contrasts of site for each condition/age group separately.

Results

Behavioural results

The proportion of error on nogo trials was significantly larger in adolescents than adults ($t(48) = 3.69, p = .001, d = 1.06$). In addition, increasing age was associated with a reduction in the proportion of error in adolescents ($r(26) = -.73, p < .001$) but not adults ($r(20) = -.19, p = .405$). Regarding response time, there was no significant difference in response time between adolescents and adults ($t(48) = 0.12, p = .904, d = 0.03$).
Table 3.2

*Means and standard deviation of release times (ms) on correct go and partial inhibitions and the proportion of errors on go and nogo trials in adolescents and adults*

<table>
<thead>
<tr>
<th>Measure</th>
<th>Condition</th>
<th>Age group</th>
<th>Age group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Adolescent</td>
<td>Adults</td>
</tr>
<tr>
<td>Proportion of Error</td>
<td>Go trials</td>
<td>.02 (.03)</td>
<td>.01 (.02)</td>
</tr>
<tr>
<td></td>
<td>Nogo trials</td>
<td>.38 (.18)</td>
<td>.22 (.10)</td>
</tr>
<tr>
<td>Response Time</td>
<td>Correct Go</td>
<td>267 (35)</td>
<td>266 (31)</td>
</tr>
</tbody>
</table>
Electrophysiological Results

Figure 3.2 LEFT: Stimulus-locked, grand-averaged waveforms for correctly identified go trials (solid green line) and successfully inhibited nogo trials (dashed red line) at Fz, FCz, Cz, Pz. Voltage (μV) is represented on the vertical axis and time (ms) is represented on the horizontal axis. RIGHT: Scalp distribution of the P2, N2 and P3.
### Table 3.3

*Means and standard deviations for P2, N2, and P3 mean amplitude (μV), and P2 and N2 peak latency (ms).*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Site</th>
<th>P2 mean amplitude</th>
<th>P2 peak latency</th>
<th>P3 mean amplitude</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Adolescent</td>
<td>Adult</td>
<td>Adolescent</td>
</tr>
<tr>
<td>Correct Go</td>
<td>Fz</td>
<td>-0.13 (1.64)</td>
<td>1.50 (1.83)</td>
<td>181 (26)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>1.34 (2.03)</td>
<td>1.86 (1.66)</td>
<td>179 (27)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>2.62 (2.27)</td>
<td>1.84 (1.60)</td>
<td>173 (23)</td>
</tr>
<tr>
<td>Successful Inhibition</td>
<td>Fz</td>
<td>1.33 (2.56)</td>
<td>1.89 (2.56)</td>
<td>178 (23)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>2.73 (2.71)</td>
<td>2.10 (2.64)</td>
<td>177 (24)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>3.85 (2.46)</td>
<td>1.72 (2.29)</td>
<td>179 (21)</td>
</tr>
</tbody>
</table>

|                            |      | N2 mean amplitude | N2 peak latency | P3 mean amplitude |
|                            |      | Adolescent       | Adult           | Adolescent       |
| Correct Go                 | Fz   | -3.39 (2.24)      | -3.12 (2.57)    | 279 (39)          |
|                            | FCz  | -2.39 (2.49)      | -3.11 (3.21)    | 270 (40)          |
|                            | Cz   | 0.51 (2.72)       | -1.37 (3.36)    | 272 (51)          |
| Successful Inhibition      | Fz   | -4.41 (3.53)      | -4.26 (3.33)    | 279 (31)          |
|                            | FCz  | -3.15 (4.13)      | -4.93 (4.29)    | 270 (28)          |
|                            | Cz   | 0.12 (4.11)       | -3.19 (4.57)    | 256 (25)          |

| Successful Inhibition      | Fz   | 2.83 (4.58)       | 3.14 (3.65)     | 244 (23)          |
|                            | FCz  | 7.56 (4.31)       | 7.45 (4.47)     | 244 (23)          |
|                            | Cz   | 8.85 (3.74)       | 7.47 (4.38)     |                  |
Table 3.4
ANOVA effects of age (Adolescent, Adult), site (Fz, FCz, Cz, Pz) and condition (Correct go, Successful inhibition) for P2, N2 and P3 ERP components

<table>
<thead>
<tr>
<th>ANOVA Effects</th>
<th>P2</th>
<th>N2</th>
<th>P3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amplitude</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>Main effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age group</td>
<td>F(1,48) = 0.10, p = .753, ( \eta^2_p = .002 )</td>
<td>F(1,48) = 2.27, p = .138, ( \eta^2_p = .045 )</td>
<td>F(1,48) = 0.02, p = .896, ( \eta^2_p &lt; .001 )</td>
</tr>
<tr>
<td>Site</td>
<td>F(2,96) = 15.83, p &lt; .001, ( \eta^2_p = .248^* )</td>
<td>F(2,96) = 44.12, p &lt; .001, ( \eta^2_p = .479^* )</td>
<td>F(2,96) = 61.12, p &lt; .001, ( \eta^2_p = .560^* )</td>
</tr>
<tr>
<td>Condition</td>
<td>F(1,48) = .7.79, p = .008, ( \eta^2_p = .140^* )</td>
<td>F(1,48) = 9.70, p = .003, ( \eta^2_p = .168^* )</td>
<td>F(1,48) = 206.77, p &lt; .001, ( \eta^2_p = .812^* )</td>
</tr>
<tr>
<td><strong>2-way interactions</strong></td>
<td></td>
<td></td>
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<tr>
<td>Site × Condition</td>
<td>F(2,96) = 1.09, p = .341, ( \eta^2_p = .022 )</td>
<td>F(2,96) = 0.25, p = .678, ( \eta^2_p = .005 )</td>
<td>F(2,96) = 61.41, p &lt; .001, ( \eta^2_p = .561^* )</td>
</tr>
<tr>
<td>Site × Age group</td>
<td>F(2,96) = 13.67, p &lt; .001, ( \eta^2_p = .222^* )</td>
<td>F(2,96) = 9.36, p &lt; .001, ( \eta^2_p = .163^* )</td>
<td>F(2,96) = 2.18, p = .119, ( \eta^2_p = .043 )</td>
</tr>
<tr>
<td>Condition × Age group</td>
<td>F(1,48) = 4.71, p = .035, ( \eta^2_p = .089^* )</td>
<td>F(1,48) = 1.38, p = .246, ( \eta^2_p = .028 )</td>
<td>F(1,48) = 1.42, p = .239, ( \eta^2_p = .029 )</td>
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<tr>
<td><strong>3-way interaction</strong></td>
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<tr>
<td>Site × Condition × Age group</td>
<td>F(2,96) = 0.42, p = .660, ( \eta^2_p = .009 )</td>
<td>F(2,96) = 2.10, p = .128, ( \eta^2_p = .042 )</td>
<td>F(2,96) = 2.08, p = .397, ( \eta^2_p = .019 )</td>
</tr>
<tr>
<td><strong>Latency</strong></td>
<td></td>
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<tr>
<td><strong>Main effects</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Age group</td>
<td>F(1,48) = 4.96, p = .031, ( \eta^2_p = .094^* )</td>
<td>F(1,48) = 2.18, p = .146, ( \eta^2_p = .043 )</td>
<td>F(1,48) = .02, p = .896, ( \eta^2_p &lt; .001 )</td>
</tr>
<tr>
<td>Site</td>
<td>F(2,96) = 0.66, p = .522, ( \eta^2_p = .013 )</td>
<td>F(2,96) = 11.74, p &lt; .001, ( \eta^2_p = .197^* )</td>
<td>F(1,48) = 4.01, p = .051, ( \eta^2_p = .077 )</td>
</tr>
<tr>
<td>Condition</td>
<td>F(1,48) = .13, p = .717, ( \eta^2_p = .003 )</td>
<td>F(1,48) = 4.10, p = .051, ( \eta^2_p = .077 )</td>
<td>F(1,48) = .13, p = .717, ( \eta^2_p = .003 )</td>
</tr>
<tr>
<td><strong>2-way interactions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site × Condition</td>
<td>F(2,96) = 0.23, p = .794, ( \eta^2_p = .005 )</td>
<td>F(2,96) = 0.69, p = .460, ( \eta^2_p = .014 )</td>
<td>F(1,48) = .02, p = .896, ( \eta^2_p &lt; .001 )</td>
</tr>
<tr>
<td>Site × Age group</td>
<td>F(2,96) = 0.29, p = .749, ( \eta^2_p = .006 )</td>
<td>F(2,96) = 0.24, p = .786, ( \eta^2_p = .005 )</td>
<td>F(1,48) = .11, p = .743, ( \eta^2_p = .002 )</td>
</tr>
<tr>
<td>Condition × Age group</td>
<td>F(1,48) = 0.11, p = .743, ( \eta^2_p = .002 )</td>
<td>F(1,48) = 1.00, p = .322, ( \eta^2_p = .020 )</td>
<td>F(1,48) = .11, p = .743, ( \eta^2_p = .002 )</td>
</tr>
<tr>
<td><strong>3-way interaction</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Site × Condition × Age group</td>
<td>F(2,96) = 4.60, p = .012, ( \eta^2_p = .087^* )</td>
<td>F(2,96) = 1.97, p = .145, ( \eta^2_p = .039 )</td>
<td>F(1,48) = .11, p = .743, ( \eta^2_p = .002 )</td>
</tr>
</tbody>
</table>
**P2 mean amplitude**

Overall amplitude did not differ between age groups, however the adolescent (but not the adult) group did show enhanced amplitude to successfully inhibited nogo compared to correct go trials (Adolescents, \( t(27) = -4.26, p < .001, d = .80 \); Adults, \( t(22) = 0.36, p = .720, d = .08 \)). Other interactions between P2 amplitude and age were not statistically significant.

Relating to distribution, significant site and site × age group effects were observed, indicating an overall frontocentral distribution which differed between adolescents and adults (Figure 3.2). Follow-up analyses showed that the P2 was centrally distributed in adolescents and frontocentrally distributed in adults. A significant linear trend was observed in adolescents, showing amplitude increases from Fz to Cz, compared to significant linear and quadratic trends in adults which showed a frontocentral maximum (Adolescents: linear, \( F(1,27) = 31.14, p < .001, \eta^2_p = .536 \); quadratic, \( F(1,27) = 0.25, p = .619, \eta^2_p = .009 \); Adults: linear, \( F(1,21) = 0.05, p = .833, \eta^2_p = .002 \); quadratic, \( F(1,21) = 5.73, p = .026, \eta^2_p = .214 \)).

**P2 peak latency**

A significant effect of age group indicated that P2 latency was delayed in adolescents compared to the adults. No other effects were statistically significant, except for a three-way interaction between site, condition and age group. Follow-up analyses comparing P2 latency in adolescents and adults, at each site and condition, indicated that the observed group effects were largest at Fz on correct go trials and Cz on successful inhibitions (Correct go at Fz, \( t(48) = 2.54, p = .014, d = 0.72 \); Successful inhibition at Cz, \( t(48) = 2.19, p = .034, d = 0.62 \), all other comparisons were non-significant).

**N2 mean amplitude**

A significant condition effect showing enhanced amplitudes on successful inhibitions compared to correctly go trials. However, group differences in overall N2 amplitude and the nogo N2 effect were not observed.

Relating to distribution, significant site and site × age group effects were observed, indicating an overall frontocentral distribution which differed between adolescents and
adults. Follow-up analyses showed a broad frontal-to-frontocentral distribution in adolescents and a more focal frontocentral distribution in adults. Although significant linear and quadratic trends were observed in both groups, large linear and quadratic trends in adolescents indicated similar amplitudes between Fz and FCz followed by a larger reduction (more positive) at Cz, while a small linear but large quadratic trend in adults indicated a frontocentral maximum (Adolescents: linear, $F(1,27) = 54.34, p < .001, \eta^2_p = .669$; quadratic, $F(1,27) = 21.49, p < .001, \eta^2_p = .443$; Adult: $F(1,21) = 4.94, p = .037, \eta^2_p = .190$; quadratic, $F(1,21) = 34.41, p < .001, \eta^2_p = .621$).

**N2 peak latency**

Latencies did not differ significantly between adolescents and adults, but Pearson’s correlations did show within group associations with age. Relating to distribution, a significant effect of site was observed, with follow-up comparisons showing earlier N2 latencies at FCz/Cz compared Fz (FCz, $t(49) = 5.00, p < .001, d = 0.71$; Cz, $t(49) = 3.98, p < .001, d = 0.56$). No other effects were statistically significant.

**P3 mean amplitude**

A significant condition effect was observed, showing enhanced amplitudes on successful inhibitions compared to correctly go trials. However, group differences in overall amplitude and nogo P3 effect were not observed.

A significant site × condition effect indicated differences in P3 distribution across conditions, where P3 was posteriorly distributed on correctly go trials and frontocentrally distributed on successful inhibition trials. Follow-up analyses revealed a stronger linear trend on correct go trials showing increasing amplitudes from Fz to Cz, while a larger quadratic trend was observed on successful inhibitions showing larger amplitudes at FCz and Cz (Correct go, linear, $F(1,49) = 12.26, p < .001, \eta^2_p = .200$; quadratic, $F(1,49) = 3.37, p = .073, \eta^2_p = .064$; Successful inhibition, linear, $F(1,49) = 77.21, p < .001, \eta^2_p = .612$; quadratic, $F(1,49) = 84.62, p < .001, \eta^2_p = .633$). No other effects were statistically significant.
Discussion

Using a modified go/nogo task, this study examined the development of response inhibition during the adolescent-adult transition. The results showed some evidence of ongoing development while also highlighting relative maturity of processes involved in response inhibition. Observed group differences in accuracy suggest that response inhibition continues to improve throughout mid-to-late adolescence; and similarity in response time suggests that this effect cannot be simply accounted for by a different speed-accuracy trade off. Group differences in P2 and the reduction in latency and the distribution of the N2 provides evidence for ongoing improvements in the efficiency of underlying early attention and conflict monitoring processes during mid-to-late adolescence. However, similarities in N2/P3 amplitude and nogo N2/P3 effects suggest that these processes are relatively mature by mid-to-late adolescence.

Development of conflict monitoring and response inhibition

Numerous studies using the go/nogo task have reported developmental reductions in N2 amplitude throughout childhood/adolescence and between children and adults (Brydges et al., 2013; Johnstone et al., 2005; Jonkman, 2006; Lamm et al., 2006; Lewis et al., 2006) as well as a significant reduction in the nogo N2 effect between adolescents to adults (Hämmerer et al., 2010). These reductions in overall amplitude were interpreted as reflecting improvements in the efficiency of conflict monitoring processes, and the reduction in the nogo N2 effect was thought to reflect a reduction in the amount of conflict experienced. However, no differences in N2 amplitude between adolescent and adult groups were observed, indicating that two groups equally responsive to conflict and suggest that conflict monitoring processes are relatively mature by mid-to-late adolescence. Small effect sizes suggested that the absence of a main effect of age and age by condition interaction for N2 amplitude indicate that the observed result is unlikely due to insufficient power. Although the absence of developmental differences in the nogo N2 effect are inconsistent with Hämmerer et al. (2010), they are in line with the observed plateau in N2 amplitude around 15 to 16 years of age (Lewis et al., 2006). Discrepancies with Hämmerer et al. (2010) may be attributed to smaller age differences between adolescent and adult groups. Both investigations compared
adolescents to young adults although, our current study was focused on mid-to-late adolescents whereas Hämmerer et al. (2010) focused on mid-adolescents ($M(SD)_{age} = 14.42 (0.55)$ years). It is possible that the observed reduction in nogo N2 effect in Hämmerer et al. (2010) reflected greater immaturity of conflict monitoring in mid-adolescents relative to young adults.

Although similar N2 latencies between adolescents and adults also supports the maturity of conflict monitoring, the significant reduction in distribution of the N2 provides evidence for the ongoing refinement of this process. Changes in latency are in line with previously reported findings in children and adolescents (Lamm et al. 2006; Lewis et al. 2006) and the developmental focalisation of the N2 distribution is in line with findings in children and adults (Brydges et al., 2013).

With respect to response inhibition and the P3, very few studies have examined developmental changes in the P3 during the go/nogo task. Some studies have reported an absence of the nogo P3 effect in young children compared to older age groups, as well as increases in the nogo P3 effect from childhood to adulthood which were interpreted as an improvement in the ability suppress responses (Hämmerer et al., 2010; Jonkman, 2006, Jonkman et al., 2003). In contrast, one study has reported developmental decreases in P3 amplitude throughout childhood/adolescence, interpreted as an improvement in cortical efficiency (Lewis et al., 2006). The results did not show an increase or decrease in overall P3 or the nogo P3 effect between adolescents and adults which suggest that the ability to suppress responses is also relatively mature in mid-to-late adolescents. Small effect sizes suggested that the absence of a main effect of age and age by condition interaction for P3 amplitude indicate that the observed result is also unlikely due to insufficient power.

*Performance difference due to slower stimulus processing and impulsive responding*

As the N2/P3 results suggest that conflict monitoring and response inhibition are relatively mature by mid-to-late adolescence, developmental differences in accuracy could be due to slower orienting of attention and early processing of the stimulus, reflected by delayed P2 latency and marginal age- and accuracy-related reductions in adolescents. These findings consistent with child-adult comparisons in the go/nogo task but have yet to be reported
during adolescence or during the adolescent-adult transition (Johnstone et al. 2005; 2007). Furthermore, as delays in P2 latency without a concomitant delay in release time, this could indicate that adolescents were responding prior to the completion of stimulus processing, resulting in lower accuracy.

Aside from the two child-adult studies, other ERP studies using the go/nogo task have not reported developmental changes in the anterior P2. Support for the role of attentional processes during response inhibition has been suggested in a recent functional neuroimaging study (O’Connor, Upton, Moore, & Hester, 2015). This study compared inhibitory processing in neutral versus meaningful/motivationally salient contexts, and reported modulation of the right inferior frontal junction and precentral gyrus and not the inferior frontal gyrus and the pre-SMA which have been implicated in response inhibition. It was suggested that the activity inferior frontal gyrus may reflect processing related to orienting attention to goal-relevant stimuli between neutral and salient conditions. While the current findings highlighting the role of the P2 and early attentional processes, the overall functional significance of the P2 and its relationship with response inhibition is still unclear, warranting further investigation.

Conclusions

The current study showed that measuring partial inhibitions in the go/nogo task augmented the sensitivity of the task to nogo errors, allowing for the identification of subtle performance differences between mid-to-late adolescents and young adults. Although improvements in accuracy suggests ongoing development of response inhibition, there processes appeared to be relatively mature by mid-to-late adolescence. We argued that delays in P2 latency without concomitant delays in response time in our data suggested that poorer accuracy in adolescents could be driven by an inability to compensate for slower stimulus processing when responding.
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Chapter 4: Enhanced sensitivity to feedback processing in adolescence: An electrophysiological investigation of dynamic risk-taking

An T. Nguyen, Dr. Jonson J. Moyle, Dr. Allison M. Fox

Abstract

Previous investigations have studied the development of risk-taking and the feedback response in adolescents and adults using unpredictable gambling and probabilistic learning tasks. However, the context of risky decisions in these studies is largely static, whereas the contexts of real-world decisions are often dynamic. To simulate and examine risky decision-making and the engagement of underlying feedback processes in a more realistic context, mid-to-late adolescents ($N = 28$, $M(SD)_{age} = 15.74(1.06)$ years) and young adults ($N = 22$, $M(SD)_{age} = 22.02(3.17)$ years) were compared on an adapted version of the Balloon Analogue Risk Task (BART, Lejuez et al., 2003). Behavioural indices of risk-taking and event-related potentials (ERPs) elicited following feedback presentation were examined. The results showed a heightened responsiveness to external feedback in adolescents compared to adults, reflected by overall enhancements in P2 and Feedback-related negativity (FRN) amplitude. However, similarities in the FRN and parietal feedback P3 valence effects suggest that the ability to discriminate and evaluate different outcomes are relatively mature by mid-to-late adolescence. No behavioural differences were observed between adolescents and adults. Rather than differences in the maturity of feedback processes, the pattern of results indicate that adolescents and adults differ in the attention allocated to process feedback, potentially reflecting differences in task approach.
Introduction

Adolescence marks a critical period of development during which biological, cognitive and behavioural changes occur. This period has been characterised by an increase in risk-taking with several studies reporting elevated levels of risk-taking on behavioural tasks in adolescents compared to adults (Defoe, Dubas, Figner, & Aken, 2015). Several neurodevelopmental models of risk-taking propose that heightened risk-taking during adolescence is partly attributed to differences in the development of brain systems implicated in cognitive control and reward processing (Shulman et al., 2016). Using functional neuroimaging, some studies have reported hyperactivity in the dopaminergic reward processing system in adolescents and this was hypothesised to result in a greater motivation to pursue rewards (Ernst et al., 2005; Galvan et al., 2006; Geier, Terwilliger, Teslovich, Velanova, & Luna, 2010; Hare et al., 2008; Van Leijenhorst et al., 2010). On the other hand, the cognitive control system involved in regulating goal-directed behaviour is not fully developed. It has been proposed that the coupling of an immature cognitive control system with a hyperactive reward processing system leads to a bias towards risk-taking behaviour in adolescents.

We have previously used a modified go/nogo task to examine the development of the cognitive control system during the adolescent-adult transition (reported in Chapter 3). In line with the neurodevelopmental model, significant improvements in task performance suggested the ongoing development of response inhibition throughout adolescence. However, the underlying processes involved in conflict monitoring and response inhibition, reflected by the N2 and P3, appeared to be mature by mid-to-late adolescence. To investigate whether the reward processing system is hyperactive during adolescence, the current study focuses on examining the development of the reward processing system during the adolescent-adult transition.

Feedback-related negativity

Using the event-related potential (ERP) technique, the development of the dopaminergic reward processing system has been examined by measuring the electrophysiological response to feedback on tasks with uncertain outcomes. Research has
focused on the feedback-related negativity (FRN), a frontocentrally distributed negative peak that occurs approximately 200-300 ms following feedback presentation. Using inverse modelling techniques, the anterior cingulate cortex has been identified as the neural generator of this scalp recorded component and this region is also implicated in related functions such as performance monitoring (Crowley et al., 2013). It has been shown that FRN amplitude is sensitive to valence with enhanced amplitudes observed following negative feedback compared to positive feedback. Its amplitude is also sensitive to other characteristics such as outcome likelihood and magnitude (Eppinger, Mock, & Kray, 2009; Sambrook & Goslin, 2015; Yi et al., 2012). Due to its sensitivity to these outcome characteristics and the anterior cingulate cortex source localisation, the FRN is thought to be involved in feedback monitoring, although its specific function is still debated (Sambrook & Goslin, 2015; San Martín, 2012; Walsh & Anderson, 2012).

According to the reinforcement-learning theory, the FRN reflects the encoding of a reward prediction error signal by the anterior cingulate cortex (Holroyd & Coles, 2002). The reward prediction error is the output of an ongoing monitoring process in the basal ganglia that signals differences between the predicted and actual outcomes. When the actual outcome is better than the predicted outcome, a phasic increase in dopaminergic activity is elicited (positive reward prediction error). Conversely, a phasic decrease in dopaminergic activity is elicited when the actual outcome is worse than predicted (negative reward prediction error). It is proposed that these phasic increases and decreases in dopaminergic activity inhibit and disinhibit neurons in the anterior cingulate cortex, giving rise to the FRN. As the anterior cingulate cortex receives input from the dopaminergic reward processing system, changes to the dopaminergic reward processing system should be associated with changes in the FRN.

**Development of the FRN and the context of feedback**

The development of the feedback response is mainly examined using two types of tasks, unpredictable gambling tasks and probabilistic learning tasks (Downes, Bathelt, & De Haan, 2017). Unpredictable gambling tasks refer to tasks where the probabilities of all outcomes are equal and therefore unpredictable. Researchers typically examine the developmental changes in the system’s response to the valence (i.e. positive or negative
feedback) and magnitude of different outcomes. Argued benefits of using unpredictable gambling tasks are that equiprobable outcomes minimise the confounding effects of stimulus probability and differences in learning and performance. Using unpredictable gambling tasks, several studies have reported reductions in the overall FRN amplitude between children/adolescents and adults, interpreted as reflecting a generalised developmental decrease in the responsiveness to external feedback (Crowley et al., 2013; Martínez-Velázquez, Ramos-Loyo, González-Garrido, & Sequeira, 2015; Zottoli & Grose-Fifer, 2012). However, developmental changes in the FRN elicited by unpredictable gambling tasks are not always observed (Santesso, Dzyundzyak, & Segalowitz, 2011). Given that feedback cannot be used to guide future choices and the proposed role of FRN in reinforcement-learning, it was argued that developmental changes in FRN may not be reflected in the response to unpredictable outcomes on choices that do not rely on learning from previous events.

The development of the feedback response has also been examined using probabilistic learning tasks. In these tasks, participants make choices under uncertain conditions where participants learn response-outcome associations from external feedback and predict the outcome of future responses. On a reinforcement-learning task, differences in task performance and learning-related changes in the FRN between children and adults suggested that developmental reductions in FRN could reflect a shift from external to internal performance monitoring processes (Eppinger et al., 2009). In addition, developmental increases in the FRN valence effect (reported as a ratio of change between the response to positive and negative feedback) from adolescence to adulthood have also been reported, suggesting that the ability to discriminate between outcomes also improves during adolescence (Grose-Fifer, Migliaccio, & Zottoli, 2014; Hämmerer, Li, Müller, & Lindenberger, 2011; Zottoli & Grose-Fifer, 2012). In accordance with the reinforcement-learning theory, improvements in the ability to discriminate between different outcomes should result in faster learning of response-outcome associations resulting in improved decision-making throughout task performance.

Although there is evidence for developmental changes in feedback processing during adolescence, there is currently a limited understanding of how these underlying changes
contribute to risk-taking behaviour. One limitation of using unpredictable gambling tasks and probabilistic learning tasks to investigate contributions of the feedback response to risk-taking behaviour is that risk-taking on behavioural tasks differs considerably from risk-taking behaviours in the real world. In most behavioural tasks used to date, the risks are static across trials and each risky decision is independent. However, risks are often dynamic in real-world risk-taking behaviours. To address this limitation, recent studies have examined risk-taking and feedback processing is by using dynamic risk-taking tasks which are focused on simulating the novelty and affective engagement of real-world risk-taking situations.

**Dynamic risk-taking tasks**

In dynamic risk-taking tasks such as the Balloon Analogue Risk Task (BART) and the Cambridge Card Task, participants are required to make risky-decisions on each trial. However, the distinguishing feature of these tasks is that the probability of negative outcomes accumulates across decisions. Highlighting the validity of the BART, differences on risk-taking on the BART differ with self-reported engagement in various real-world risky behaviours (e.g. alcohol use, smoking, illicit substance use, gambling, unprotected sex) in different populations (e.g. inner-city drug users, problem gamblers, undergraduate students, inner-city adolescents) (Aklin, Lejuez, Zvolensky, Kahler, & Gwadz, 2005; Bornovalova, Daughters, Hernandez, Richards, & Lejuez, 2005; Coffey, Schumacher, Baschnagel, Hawk, & Holloman, 2011; Hopko et al., 2006; Ledgerwood, Alessi, Phoenix, & Petry, 2009; Lejuez et al., 2002, 2003; Lejuez, Simmons, Aklin, Daughters, & Dvir, 2004). With respect to reliability, White, Lejuez and de Wit (2008) have demonstrated acceptable rest-retest reliability of the BART across days ($r = +.77, p < .001$). There is also emerging evidence that dynamic tasks may be useful for detecting individual and developmental differences in risk-taking compared to static tasks. Lejuez et al. (2003) compared the BART with the Bechara Gambling Task, a static risk-taking task that was succeeded by the IOWA gambling task and found that performance on the BART was able to differentiate smoking and non-smoking groups but the Bechara Gambling Task did not. Figner, Mackinlay, Wilkening, and Weber (2009) observed significantly increased risk-taking behaviour in adolescents compared to adults using the Cambridge Card Task and argued that developmental differences in risk-
taking may be more pronounced in dynamic tasks compared to static tasks. They attributed their findings to the affective engagement of participants in the task used.

Recently, studies have used the Balloon Analogue Risk Task (BART) to study changes in FRN in various at-risk groups (Fein & Chang, 2008; Takács et al., 2015; Yau, Potenza, Mayes, & Crowley, 2015). In a population of actively drinking, treatment-naïve alcoholics it was found that smaller FRN amplitudes were associated with greater family history density of alcohol problems (Fein & Chang, 2008). In an anxious sample, it was found that participants showed risk-aversive behaviour and had a reduced FRN, suggesting pessimistic expectations (Takács et al., 2015). In problematic internet users, overall FRN amplitudes were attenuated suggesting decreased sensitivity to external feedback (Yau et al., 2015).

In addition to the FRN, some studies using the BART have also examined the anterior P2 and parietal P3 elicited to the presentation of feedback (Kardos et al., 2016; Kiat, Straley, & Cheadle, 2016; Yau et al., 2015). The anterior P2 refers to an anteriorly distributed positivity occurring approximately 150-200ms (Polezzi, Lotto, Daum, Sartori, & Rumiati, 2008). In the oddball paradigm, it was observed that P2 amplitude to the infrequent stimulus in the oddball task was modulated depending on whether it was described as the target or non-target (Potts, Liotti, Tucker, & Posner, 1996). Similarly, changes in P2 amplitude were observed to various features such as colour, size and orientation of stimuli when they were specified by task instructions as being significant (Luck & Hillyard, 1994). It was hypothesised that modulations of P2 amplitude reflect top-down processes facilitating the processing of certain features (or combination of features) identified as having potential significance (Daffner, Alperin, Mott, Tusch, & Holcomb, 2015). With respect to external feedback, it has been reported that the anterior P2 was sensitive to the predictability of outcomes, with enhanced amplitude to unpredictable compared to predictable outcomes irrespective of valence (Polezzi et al., 2008). Similarly, modulations of the anterior P2 during feedback could reflect top-down attentional allocation, facilitating processing of trials with greater uncertainty or motivational significance. In the BART, P2 amplitudes increased as a function of risk which is in line the interpretation that the functional significance of this peak reflects greater orientation of attention to potentially significant events (Kiat et al., 2016).
The feedback P3 refers to a parietally distributed positivity occurring approximately 300-600ms following feedback presentation. This component is thought to reflect more thorough evaluative processing and is sensitive to outcome likelihood and magnitude, with enhanced amplitudes typically elicited by stimuli which are infrequent and have larger outcomes. As such, it is thought to reflect the motivational significance of the outcome. Yau et al. (2015) observed attenuated P3 amplitudes in problematic internet using adolescents thought to reflect their impaired ability to assign attention to motivationally salient events. Kardos et al. (2016) observed an attenuated P3 in older adults compared to younger adults to both positive and negative feedback, suggesting that the motivational salience of these outcomes may be underrated in older adults.

Although numerous studies have examined risk-taking and the underlying feedback processes using dynamic risk-taking tasks, research has yet to examine this within the younger developmental context. In the current study, we used an ERP adapted version of the Balloon Analogue Risk Task (BART) to examine feedback response and risk-taking behaviour to study developmental changes in the reward processing system during transition from adolescence to adulthood. Behaviourally, we expect that adolescents would show heightened risk-taking compared to adults. In line with previous observations, we also expected to observe a general reduction in FRN amplitude indicating a general decrease in the responsiveness to external feedback, and an increase in the FRN effect (i.e. the difference in the response to negative and positive feedback) indicating improvements in discriminating between different outcomes. We also examined the anterior P2 and parietal P3 components, to determine whether the orientation of attention and/or the motivational salience of feedback are driving factors underpinning developmental differences in risk-taking behaviour.
Methods

Participants

The sample consisted of 28 adolescents aged between 14 and 17 years ($M(SD)_{age} = 15.74(1.06)$ years, 16 males, 26 right-handed) and 22 adults aged between 18 and 27 years ($M(SD)_{age} = 22.02(3.17)$ years, 10 males, 17 right-handed). The data of these participants were also reported in Chapter 3. This description of adolescents has been used in previous developmental studies (Zottoli & Grose-Fifer, 2012) and in Australia, 18 years of age corresponds to the age of consent. Furthermore, this age typically marks the critical period of environmental change where individuals transition out of the secondary school environment. Participants were recruited from local high schools, psychology undergraduates from the University of Western Australia, and volunteers from the local community. Adult participants and a parent/legal guardian of adolescent participants provided informed consent prior to participation and the protocol was approved by the human research ethics committee of the University of Western Australia.

Balloon Analogue Risk Task (BART, Lejuez et al., 2002)

The BART is a dynamic risk-taking task in which participants are required to inflate virtual balloons with the goal of maximising the amount of money earned. During the task, a single balloon is presented and participants indicate whether they want to continue pumping the balloon or whether they want to cash-out the points accumulated at that point. Initially, the balloon has no value but the balloon’s value increases incrementally with each successful pump. However, the value of the balloon is only awarded to the participant when they cash-out. Successful inflations are also accompanied by an increase in the probability that the balloon will burst. If the pump is unsuccessful, the balloon bursts and the accumulated value is lost. If the participant opts to cash-out, the value of the balloon is transferred to their total prize earnings. Participants were informed that the probability of the balloon bursting increases with each successive pump but were not informed about the actual probabilities or the total number of balloons to be presented during the task.
The current task was based on Fein and Chang (2008) to permit analysis of ERP data acquired during task performance. The maximum pumps possible for each balloon was shortened to 20 pumps and participants were presented with a total of 50 balloons (i.e. 50 sets of trials). Successful pumps were associated with an increase in value of 5 cents. The probability of the balloon bursting after the first pump is 1/20, 1/19 for the second pump, increasing exponentially until the 20th pump when the balloon burst is certain. The cumulative probability of bursts increases as linear function and the expected value of a specific number of pumps peaks at 10 pumps (Figure 4.1). Expected value is calculated by multiplying the balloons value with the cumulative probability of successful pumps. Balloons were represented on screen as a blue circle presented on a black background, and the circle increased in size as the balloon was pumped. Each stimulus remained on the screen until a response was made by pressing the appropriate key (‘Z’ for pump and ‘/’ for cash-out). Visual feedback was presented 500 ms after the response in white text over a black background for a duration of 500ms, indicating either the updated value of the balloon, or whether it had burst. Following the end of the sequence of trials corresponding to each balloon inflation (i.e. cash-out or burst), additional feedback was presented for 500ms indicating either the total earned or that the accumulated value was lost (Figure 4.2).

**Figure 4.1 LEFT:** The cumulative and individual probabilities of bursts **RIGHT:** The expected value for each number of pumps.

Based on previous studies, the adjusted average number of pumps per balloon and the number of burst were extracted as behavioural indices of risky decision-making. The
adjusted average number of pumps is calculated as the average pumps selected for balloons that did not burst. This is preferred to other measures as it is the only measure not constrained by the pseudo-random popping threshold of the balloon (Lejuez et al., 2007). Participants also participated in an additional response lasting for approximately 12 minutes which reported in Chapter 2 and 3.
Figure 4.2 Diagram depicting trial progression for possible responses (pump, cash-out) and feedback outcomes (successful pump, burst and cash-out) in the Balloon Analogue Risk Task
**Data Acquisition**

Electrophysiological data were acquired using SCAN 4™ and were processed offline using EEGLAB 13.3.2 (Delorme & Makeig, 2004) and ERPLAB 5.0.0 (Lopez-Calderon & Luck, 2014). The electrophysiological activity was continuously recorded using Ag/AgCl electrodes at 33 scalp locations (FP1, FP2, F3, F4, F7, F8, Fz, FC1, FC2, FC5, FC6, FCz, FT9, FT10, C3, C4, CZ, T7, T8, CP1, CP2, CP5, CP6, P3, P4, P7, P8, Pz, PO9, PO10, O1, O2, Iz). The online EEG was amplified using a NuAmps 40-channel amplifier, digitised at a sampling rate of 250 Hz and filtered using a 0.05–30 Hz bandpass filter. Additional electrodes were placed at AFz, the left and right mastoid regions, and 2 cm above and below the left eye. The right mastoid electrode was set as the online reference and AFz was set as the ground. Electrodes around the left eye were used to record ocular movement.

The EEG data were filtered offline using a 0.5 Hz high-pass Butterworth filter (12 dB roll-off). Ocular artifacts were corrected by using independent components analysis to identify guided by SASICA and remove components associated with ocular muscle activity (Chaumon, Bishop, & Busch, 2015). Channels with excessive noise were spherically interpolated. One channel was interpolated for 11 participants, two channels were interpolated for one participant and four channels were interpolated for an additional participant. EEG data were re-referenced offline using a common averaged reference.

Feedback-locked epochs were extracted, synchronised to the presentation of feedback to successful pumps and burst trials. Epochs were segmented from 100 ms pre-feedback onset to 1000 ms post-feedback onset. Individual average waveforms were computed for successful pumps and bursts separately, and epochs with activity exceeding ± 150 μV were excluded from these averages. Individual averaged waveforms were baseline-corrected around the amplitude calculated over the pre-feedback interval. Following artefact rejection, the average number of epochs included in the ERPs across all participants were 275 ($SD = 75$) and 19 ($SD = 5$) for successful pumps and bursts respectively. Within the adolescent group, an average of 259 ($SD = 69$) and 18 ($SD = 4$) epochs were included in the averaged ERPs. Within the adult group, an average of 295 ($SD = 79$) and 19 ($SD = 6$) epochs were included in the averaged ERP. Independent samples $t$-tests showed that the number of epochs
in each valence condition did not differ significantly between groups (successful pumps, \( t(48) = -1.74, p = .088, d = 0.50 \); bursts, \( t(48) = -.65, p = .518, d = 0.05 \)).

**Distinct frontocentral and parietal P3 subcomponents**

Inspection of the grand-averaged waveforms revealed distinct frontocentral and parietal peaks, similar to the subcomponent structure observed in the positivity following the error-related negativity (Arbel & Donchin, 2009). To examine whether these two components could be dissociated statistically, principal components analyses (PCA) were conducted on ERP data elicited to feedback indicating a successful pump and burst using the ERP PCA toolkit (version 2.54, Dien, 2010). Data from 100 ms pre-feedback to 750 ms post-feedback (213 data points) for 28 adolescents and 22 adults, at 33 scalp locations were included in the analysis (924 cases for adolescents, 726 cases for adults). The resulting case-to-variable ratio for each PCA was 4.34 for adolescents and 3.41 for adults. Temporal PCA was conducted using the covariance matrix followed by Kaiser normalisation and a Promax rotation. The number of factors retained was determined by examining plots derived from the ERP PCA toolkit showing parallel tests comparing the scree of the dataset to the scree derived from a fully random dataset. For EEG data on feedback indicating successful pumps, 10 factors were retained explaining 95.51% of the total variance. For EEG data on feedback indicating bursts, 10 factors were retained explaining 90.35% of the total variance. P3 subcomponents were identified by inspecting microvolt-rescaled factor loading waveforms for each factor derived from the ERP PCA toolkit, and the scalp distribution of these factors. Specifically, relevant factors were identified based on the peak latency, polarity and scalp distribution of the factors with respect to the ERP waveforms. Factors explaining < 10% of the total variance were not interpreted. On feedback indicating bursts, distinct frontocentral and parietal factors were identified with factor loadings peaking at 416 ms and 644 ms respectively (See Figure 4.4 in Supplementary Figures section).

**Measurement of event-related potentials**

In the FRN literature, there are varying conceptualisations of the FRN which impact its measurement across studies. Some studies conceptualise the FRN as the negative deflection in the waveform (Crowley et al., 2013; Eppinger et al., 2009; Martínez-Velázquez
et al., 2015; Santesso et al., 2011; Yi et al., 2012), whereas other studies refer to it as the relative negativity between positive and negative feedback occurring at ~200-350 ms (Lukie, Montazer-Hojat, & Holroyd, 2014). Additionally, studies have also represented amplitude changes in the FRN peak as a ratio of change (Grose-Fifer et al., 2014; Hämmerer et al., 2011; Zottoli and Grose-Fifer, 2012). To maintain consistency with previous research, the FRN was measured as the negative deflection (represented both as a voltage difference and as a ratio of change) as well as the relative negativity between different feedback outcomes (reflected on the burst-minus-successful pump difference waveform).

ERP amplitudes were quantified as mean amplitudes calculated over a 40 ms window centred on the peak latency of grand-averaged waveform. FRN, P2 and frontocentral P3 were measured at Fz, FCz and Cz, and the parietal P3 subcomponents was measured at Fz, FCz, Cz and Pz. Short intervals were used to minimise the interference of adjacent components and separate latency windows were computed for each group and feedback condition to control for potential latency variability. The latency windows used to measure mean amplitudes are specified in Table 4.1.

On the grand-averaged waveform, the peak latency of the FRN was identified as the most negative point between 200-350 ms following feedback presentation. Due to amplitude differences in the preceding P2 component, the FRN was measured with respect to mean amplitude of the P2. For the P2, peak latencies were identified as the most positive points between 150-250 ms following feedback presentation. The frontocentral P3 subcomponent was identified as the most positive peak between 300-450 ms on both feedback conditions, and due to preceding differences, the frontocentral P3 was measured with respect to the FRN. The parietal P3 subcomponent was identified as the most positive peak between 600-800 ms following burst feedback and 400-600 ms for feedback indicating a successful pump. These intervals were used to capture the parietal components identified in the PCA.
Table 4.1

Mean amplitude latency windows of FRN, feedback P3 and P2 for each condition and age group. Latencies are with respect to the onset of the feedback stimulus.

<table>
<thead>
<tr>
<th>ERP</th>
<th>Condition</th>
<th>Age group</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>P2</td>
<td>Successful pump</td>
<td>188-228 ms</td>
<td>184-224 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>188-228 ms</td>
<td>184-224 ms</td>
<td></td>
</tr>
<tr>
<td>FRN</td>
<td>Successful pump</td>
<td>284-324 ms</td>
<td>280-320 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>280-320 ms</td>
<td>276-316 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Difference</td>
<td>280-320 ms</td>
<td>276-316 ms</td>
<td></td>
</tr>
<tr>
<td>Frontocentral P3</td>
<td>Successful pump</td>
<td>328-368 ms</td>
<td>332-372 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>364-404 ms</td>
<td>380-420 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Difference</td>
<td>376-416 ms</td>
<td>392-432 ms</td>
<td></td>
</tr>
<tr>
<td>Parietal P3</td>
<td>Successful pump</td>
<td>400-440 ms</td>
<td>452-492 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>688-708 ms</td>
<td>648-688 ms</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Difference</td>
<td>668-708 ms</td>
<td>656-696 ms</td>
<td></td>
</tr>
</tbody>
</table>

Data Analysis

Preliminary analyses were conducted to ensure the data were appropriate for analysis. Across all variables and participants, 31 outliers were identified and winsorized to 2.2 times the interquartile range (Hoaglin & Iglewicz, 1987). All were normally distributed, as assessed by examining skew and kurtosis coefficients against critical thresholds of |2| and |7| (Curran, West, & Finch, 1996).

For behavioural data, adjusted average pumps and number of bursts in adolescents and adults were compared using an independent samples t-test. FRN, P2 and frontocentral P3 mean amplitudes were examined using three 3 × 2 × 2 mixed ANOVAs with site (Fz, FCz, Cz) and feedback valence (successful pump, burst) as the within-subject factor and age group (adolescent, adult) as the between-subjects factor. The parietal P3 was analysed using a 4 × 2 × 2 mixed ANOVA with the same factors and including Pz as an additional site. The FRN and frontocentral P3 valence effects reflected on the difference-waveform and the ratio of change (FRN only) were analysed using three 3 × 2 mixed ANOVA with site (Fz, FCz, Cz) as the within-subjects factor and age group (adolescent, adult) as the between-subjects factor.
The valence effect for the parietal P3, reflected on the difference-waveform, was analysed using a 4 × 2 mixed ANOVAs with the same variables including Pz as an additional site.³

Greenhouse-Geisser corrected p-values were reported for effects where the assumption of sphericity was violated. Significant main effects of site and feedback condition were followed by paired-sample t-tests. Significant site × valence interactions were followed up with one-way ANOVAs and by examining within-subject contrasts of site for each feedback condition separately. The sequential Holm-Bonferroni method was used to adjust for the accumulation of type-I error in multiple comparisons (Holm, 1979).

Results

Behavioural Results

The adjusted average number of pumps and the number of bursts did not differ significantly between adolescents and adults (adjusted average pumps, \( t(48) = -1.77, p = .082, d = 0.51; M(SD)_{adolescents} = 6.49(1.70), M(SD)_{adults} = 7.49(2.28) \); number of bursts, \( t(48) = -0.83, p = .409, d = 0.24; M(SD)_{adolescents} = 19.29(4.58), M(SD)_{adults} = 20.59(6.49) \)).

³ As the final sample did not have an equal number of males and females within each age group, a separate set of ANCOVAs were conducted with gender as a covariate. There were no significant main effects of gender and the pattern of age-related effects did not change for any behavioural or ERP indices.
Electrophysiological Results

Figure 4.3 LEFT: Grand-averaged feedback-locked waveforms for successful pumps (green line), bursts (red line) and burst-minus-successful pumps (black line) in adolescents (dashed line) and adults (solid line) at Fz, FCz, Cz. Voltage (μV) is represented on the vertical axis and time (ms) is represented on the horizontal axis. RIGHT: Scalp maps showing the distribution of the P2, FRN, Frontocentral and Parietal P3 (See Table 4.1. for latency windows)
Table 4.2

Mean and standard deviation of P2, FRN and frontocentral and parietal P3 amplitudes measured on waveforms for feedback indicating successful pumps and bursts, the ‘burst minus successful pump’ difference waveform, as well as a ratio score indicating relative change in amplitude from successful pumps to burst

<table>
<thead>
<tr>
<th>ERP</th>
<th>Site</th>
<th>Age group</th>
<th>Successful Pump</th>
<th>Burst</th>
<th>Difference</th>
<th>Ratio Score</th>
<th>Successful Pump</th>
<th>Burst</th>
<th>Difference</th>
<th>Ratio Score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Adolescent</td>
<td></td>
<td></td>
<td></td>
<td>Adult</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td>Fz</td>
<td>4.31 (2.56)</td>
<td>5.04 (2.79)</td>
<td>-</td>
<td>2.92 (2.19)</td>
<td>3.53 (2.65)</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>4.99 (2.50)</td>
<td>5.48 (2.15)</td>
<td>-</td>
<td>3.03 (1.97)</td>
<td>3.29 (2.24)</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>4.17 (2.05)</td>
<td>4.49 (2.30)</td>
<td>-</td>
<td>2.34 (1.50)</td>
<td>2.30 (1.93)</td>
<td>-</td>
<td>-</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FRN</td>
<td>Fz</td>
<td>4.70 (3.31)</td>
<td>3.85 (2.46)</td>
<td>-0.20 (2.78)</td>
<td>0.15 (0.99)</td>
<td>2.52 (2.68)</td>
<td>2.41 (2.19)</td>
<td>0.48 (1.97)</td>
<td>0.16 (1.33)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>4.86 (3.53)</td>
<td>3.63 (2.28)</td>
<td>-0.82 (2.66)</td>
<td>0.19 (0.85)</td>
<td>2.78 (2.75)</td>
<td>2.27 (1.98)</td>
<td>-0.28 (2.04)</td>
<td>0.24 (1.36)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>3.20 (3.41)</td>
<td>1.77 (1.84)</td>
<td>-1.11 (2.16)</td>
<td>0.30 (1.81)</td>
<td>1.71 (2.91)</td>
<td>1.02 (1.54)</td>
<td>-0.99 (2.27)</td>
<td>1.00 (1.76)</td>
<td></td>
</tr>
<tr>
<td>Frontocentral P3</td>
<td>Fz</td>
<td>0.53 (1.31)</td>
<td>4.29 (4.68)</td>
<td>3.55 (3.46)</td>
<td>-</td>
<td>0.43 (0.97)</td>
<td>3.58 (2.42)</td>
<td>4.01 (2.89)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>0.36 (1.38)</td>
<td>5.77 (5.44)</td>
<td>5.21 (4.50)</td>
<td>-</td>
<td>0.14 (1.08)</td>
<td>5.24 (2.93)</td>
<td>5.41 (3.42)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>0.03 (1.31)</td>
<td>4.44 (4.79)</td>
<td>4.40 (4.15)</td>
<td>-</td>
<td>-0.09 (1.13)</td>
<td>4.41 (2.98)</td>
<td>4.44 (3.17)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Parietal P3</td>
<td>Fz</td>
<td>0.19 (1.91)</td>
<td>0.63 (3.69)</td>
<td>1.35 (3.33)</td>
<td>-</td>
<td>0.03 (1.91)</td>
<td>1.74 (2.80)</td>
<td>1.59 (2.69)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>0.48 (1.57)</td>
<td>2.60 (3.49)</td>
<td>2.82 (3.30)</td>
<td>-</td>
<td>-0.09 (1.47)</td>
<td>3.22 (2.19)</td>
<td>3.05 (2.23)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>0.89 (1.55)</td>
<td>4.34 (3.11)</td>
<td>4.22 (2.81)</td>
<td>-</td>
<td>0.22 (1.18)</td>
<td>4.09 (1.60)</td>
<td>3.87 (1.98)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pz</td>
<td>2.33 (1.41)</td>
<td>4.84 (2.66)</td>
<td>4.52 (2.50)</td>
<td>-</td>
<td>1.27 (1.28)</td>
<td>4.18 (2.60)</td>
<td>3.89 (2.57)</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>
### Table 4.3

**ANOVA effects of age (Adolescent, Adult), site (Fz, FCz, Cz, Pz) and valence (Successful pump, Burst) for P2, FRN, frontocentral and parietal P3 ERP components amplitudes**

<table>
<thead>
<tr>
<th>ANOVA effects</th>
<th>P2</th>
<th>FRN</th>
<th>ERP</th>
<th>Frontocentral P3</th>
<th>Parietal P3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main effects</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age group</td>
<td>F(1,48) = 12.40, p = .001, η² = .205*</td>
<td>F(1,48) = 6.60, p = .013, η² = .121*</td>
<td>F(1,48) = .21, p = .646, η² = .004</td>
<td>F(1,48) = .23, p = .636, η² = .005</td>
<td></td>
</tr>
<tr>
<td>Site</td>
<td>F(2,96) = 9.14, p = .002, η² = .160*</td>
<td>F(2,96) = 22.34, p &lt; .001, η² = .318*</td>
<td>F(2,96) = 9.13, p = .002, η² = .160*</td>
<td>F(3,144) = 10.47, p = .001, η² = .179*</td>
<td></td>
</tr>
<tr>
<td>Valence</td>
<td>F(1,48) = 2.26, p = .139, η² = .045</td>
<td>F(1,48) = 5.69, p = .021, η² = .106*</td>
<td>F(1,48) = 64.12, p &lt; .001, η² = .572*</td>
<td>F(1,48) = 116.50, p &lt; .001, η² = .708*</td>
<td></td>
</tr>
</tbody>
</table>

| **2-way interactions** |    |     |     |                 |            |
| Site × Valence  | F(2,96) = 6.23, p = .009, η² = .115* | F(2,96) = 2.97, p = .079, η² = .058 | F(2,96) = 10.47, p = .001, η² = .179* | F(3,144) = 10.47, p = .001, η² = .179* |
| Site × Age group | F(2,96) = 1.45, p = .240, η² = .029 | F(2,96) = 1.11, p = .333, η² = .023 | F(2,96) = 2.19, p = .092, η² = .044 | F(3,144) = 2.19, p = .092, η² = .044 |
| Valence × Age group | F(1,48) = 0.49, p = .487, η² = .010 | F(1,48) = 1.19, p = .281, η² = .010 | F(1,48) = 3.03, p = .088, η² = .059 | F(1,48) = 3.03, p = .088, η² = .059 |

| **3-way interaction** |    |     |     |                 |            |
| Site × Valence × Age group | F(2,96) = .95, p = .390, η² = .019 | F(2,96) < .01, p = .998, η² < .001 | F(3,144) = .51, p = .674, η² = .011 | F(3,144) = .51, p = .674, η² = .011 |

| **Main effects** |    |     |     |                 |            |
| Age group       | F(1,48) = 0.52, p = .474, η² = .011 | F(1,48) = 0.61, p = .438, η² = .013 | F(1,48) = 0.06, p = .812, η² = .001 | F(1,48) = 0.05, p = .834, η² = .001 |
| Site            | F(2,96) = 13.69, p < .001, η² = .222* | F(2,96) = 4.02, p = .034, η² = .077* | F(2,96) = 10.98, p < .001, η² = .186* | F(3,144) = 18.00, p < .001, η² = .273* |

| **2-way interactions** |    |     |     |                 |            |
| Site × Age group  | F(2,96) = 0.79, p = .456, η² = .016 | F(2,96) = 2.03, p = .138, η² = .040 | F(2,96) = 0.21, p = .810, η² = .004 | F(3,144) = 0.54, p = .659, η² = .011 |
Feedback P2

No significant interactions between the factors of valence and site with age were observed indicating that the pattern and topography of this peak did not differ between adolescents and adults. A significant main effect of age group was observed showing that overall P2 amplitude was larger (more positive) in adolescents compared to adults.

With respect to distribution, a significant effect of site was observed and follow-up analyses showed that the P2 was frontally distributed (Fz vs. FCz – t(49) = -1.64, p = .107, d = 0.23; FCz vs. Cz, t(49) = .507, p < .001, d = 0.72). A significant site × valence interaction showed differences in distribution such that P2 was frontocentrally distributed on successful pump trials whereas on burst feedback trials, the P2 showed a broad frontal-to-frontocentral P2. The frontocentral distribution was indicated by significant quadratic trend, and the frontal-to-frontocentral distribution was indicated by significant linear and quadratic trends showing a reduction in amplitude from Fz/FCz to Cz (Successful pump: linear, F(1,49) = 1.78, p = .188, \( \eta_p^2 = .035 \); quadratic, F(1,49) = 42.19, p < .001, \( \eta_p^2 = .463 \); Burst: linear, F(1,49) = 6.28, p = .016, \( \eta_p^2 = .114 \); quadratic, F(1,49) = 27.99, p < .001, \( \eta_p^2 = .364 \)).

Feedback-related negativity

Significant effects of site and valence were observed showing a frontal-to-frontocentral FRN which was enhanced (more negative) on burst feedback compared to successful pump feedback trials. The frontal-to-frontocentral distribution was indicated by follow-up analyses showing enhanced amplitudes (more negative) at Fz/FCz compared to Cz (Fz vs. Cz, t(49) = -4.60, p < .001, d = 0.65; FCz vs. Cz, t(49) = -8.73, p < .001, d = 1.23).

A significant effect of age group was observed showing that overall FRN amplitude was larger (more negative) in adolescents compared to adults. However, no significant interactions with age were observed, indicating that the pattern of site and valence effects did not differ between adolescents and adults.

When the FRN was measured on the ‘burst minus successful pump’ difference waveform and represented as a ratio of change, there were no group differences in the magnitude and distribution of the valence effect. However, significant effects of site were
observed indicating that the FRN effect was centrally distributed. Follow-up analyses on the difference waveform FRN showed increases in amplitude from Fz to Cz (Fz vs. FCz, $t(49) = 3.36$, $p = .002$, $d = 0.48$; Fz vs. Cz, $t(49) = 3.93$, $p < .001$, $d = 0.56$; FCz vs. Cz, $t(49) = 2.96$, $p = .005$, $d = 0.42$). Follow-up analyses on ratio of change measurements were not statistically significant, but the pattern of effect sizes were indicative of a central maximum (Fz vs. FCz, $t(49) = -0.45$, $p = .657$, $d = 0.07$; FCz vs. Cz, $t(49) = -2.02$, $p = .049$, $d = 0.29$).

**Frontocentral feedback P3**

Significant effects and interactions of site and valence were observed, indicating an enhanced and frontocentrally distributed P3 on burst feedback compared to successful pump feedback trials, which lacked a prominent peak. The frontocentral distribution of frontocentral P3 to burst feedback trials was indicated by a significant quadratic trend showing a frontocentral maximum (linear, $F(1,48) = 1.06$, $p = .309$, $\eta^2_p = .021$; quadratic, $F(1,48) = 58.92$, $p < .001$, $\eta^2_p = .546$). On successful pump feedback trials, positivity in this time window was parietally maximal, as indicated by significant linear trend showing increases in amplitude from Fz to Pz (linear, $F(1,49) = 14.49$, $p < .001$, $\eta^2_p = .232$; quadratic, $F(1,49) = 0.80$, $p = .375$, $\eta^2_p = .016$). No significant effects or interactions with age group were observed, indicating that overall amplitude and the pattern of site and valence effects did not differ significantly between adolescents and adults.

In line with the above findings, the frontocentral feedback P3 on the ‘burst minus successful pump’ difference waveform was frontocentrally distributed and did not differ between adolescents and adults (Fz vs. FCz, $t(49) = -5.15$, $p < .001$, $d = 1.75$; FCz vs. Cz, $t(49) = 3.58$, $p < .001$, $d = 0.51$).

**Parietal feedback P3**

Significant effects and interactions of site and valence were observed, indicating an overall parietally distributed P3 which was enhanced on burst feedback compared to successful feedback trials. The site × valence interaction and follow-up analyses indicated that the positivity was more anteriorly distributed (centro-parietal) on bursts feedback compared to successful pump feedback trials (parietal). The centro-parietal distribution was
indicated by significant linear and quadratic trends (linear, $F(1,49) = 25.51, p < .001, \eta^2_p = .342$; quadratic, $F(1,49) = 12.71, p < .001, \eta^2_p = .306$; cubic, $F(1,49) = 2.54, p = .117, \eta^2_p = .049$). The trends reflected amplitudes increases from Fz to Pz and plateaued at Cz. The parietal distribution on successful pump feedback trials was indicated by significant linear, quadratic and cubic trends indicating a curvilinear and exponential increase in amplitude from Fz to Pz (linear, $F(1,49) = 22.76, p < .001, \eta^2_p = .317$; quadratic, $F(1,49) = 43.00, p < .001, \eta^2_p = .476$; cubic, $F(1,49) = 10.07, p = .003, \eta^2_p = .171$). No significant effects or interactions with age group were observed, indicating that overall amplitude and the pattern of site and valence effects did not differ significantly between adolescents and adults.

In line with the above findings, the parietal P3 on the ‘burst minus successful pump’ difference waveform was parietally distributed and did not differ between adolescents and adults (Fz vs. FCz, $t(49) = -5.99, p < .001, d = 0.84$; FCz vs. Cz, $t(49) = -5.49, p < .001, d = 0.69$; Cz vs. Pz, $t(49) = -3.92, p < .001, d = 0.55$).

**Discussion**

The current study examined the development of feedback processing and risky decision-making during the adolescent-adult transition using a dynamic risky decision-making task. In line with previous BART studies, we observed a significant enhancement in FRN and P3 amplitude to burst feedback compared to successful pump feedback. The results showed a heightened responsiveness to external feedback in adolescents compared to adults, reflected by overall enhancements in P2 and FRN amplitudes. However, similarities in the FRN/P3 valence effects suggest that the ability to discriminate and evaluate different outcomes are relatively mature by mid-to-late adolescence. Given the maturity of discrimination and evaluative processes, one interpretation of the current findings is that adolescents and adults differ in the degree of attention allocated to process feedback, potentially reflecting differences in task approach. Additionally, a frontocentral feedback P3, distinct from parietal feedback P3 was also identified in this study. Previous studies have shown sensitivity of the frontocentral subcomponent to outcome likelihood and task relevance, however the functional significance of this subcomponent is relatively unclear.
Maturity of feedback-related processing during the adolescent-adult transition

Differences in overall FRN amplitude are consistent with developmental reductions observed in previous adolescent-adult comparisons and other developmental studies using unpredictable gambling and probabilistic learning tasks (Crowley et al., 2013; Eppinger et al., 2009; Grose-Fifer et al., 2014; Hämmerer et al., 2011; Martínez-Velázquez et al., 2015; Zottoli & Grose-Fifer, 2012). Greater amplitudes in adolescents were interpreted as reflecting a greater reliance on external feedback and age-related reduction were interpreted as reflecting a shift from external to internal monitoring processes. However, there were no significant differences in adjusted average pumps or bursts to indicate changes in risky decision-making, nor were there differences in the FRN/Parietal P3 valence effects to suggest that differences in the ability to discriminate and evaluate feedback.

The results largely revealed small effect sizes for age by valence interactions for P2, FRN and P3 valence effects suggesting that the results were unlikely due to insufficient power. However, a moderate effect size was observed for average adjusted pumps in the direction that adolescents pumped the balloon less than adults suggesting that adolescents may be more risk-averse, highlighting insufficient power as a possibility in this contrast. While this finding is contrary to theories of adolescent risk-taking, it is consistent with the observation of enhanced ERP amplitudes in adolescents. With regards to the possibility that overall differences in ERP amplitude were due to non-neural factors such as scalp thickness, sweat and skin dryness were also considered unlikely as these factors should have affected all ERP components including the P3 which did not differ between groups. The possibility of stimulus-processing jitter was also considered unlikely as differences or jitter in stimulus processing should result in greater variability in early ERP components (P2) and varied overlap with subsequent components (FRN), resulting in an overall change in the morphology of the P2/FRN complex. However, the distribution and magnitude of the FRN did not differ between groups.

Group differences in P2 and FRN show that developmental differences during the adolescent-adult transition manifest as a constant additive gain to the feedback response rather than a different ratio in the feedback response to positive and negative feedback. The
idea of an additive compared to a multiplicative (ratio-based) models in feedback processing might be useful for investigating whether developmental differences scale with outcome magnitude and probability, a potential avenue for future research. As the differences were observed in the P2, they could be attentional in nature as the P2 amplitude has been shown to be modulated by attention. The role of the anterior P2 in attentional processing has been demonstrated in studies using the oddball paradigm reporting the sensitivity of P2 amplitude to stimulus features specified by task instructions as being significant (Luck & Hillyard, 1994; Potts et al., 1996). Previous studies using the BART have also examined the P2 elicited to external feedback, highlighting its sensitivity to outcome uncertainty, with reported increases in P2 amplitude as a function of risk (Kiat et al., 2016; Polezzi et al., 2008). Larger P2 amplitudes in adolescents could reflect the increased allocation of attentional resources to facilitate the processing of feedback, indicating that they identified external feedback as being more important and/or perceived that trials had greater uncertainty.

While the discrimination between positive and negative outcomes appears to be mature, group differences in P2 and FRN indicates a constant additive gain to the feedback response rather than a different ratio in the feedback response to positive and negative feedback. These group differences could be attentional in nature as the P2 amplitude is modulated by attention.

**Samples differences and developmental trajectory of FRN during adolescence**

Although the task produced significant valence effects and had sufficient power to detect overall differences in P2 and FRN amplitudes, the absence of adolescent-adult differences in risky decision-making and the FRN valence effect are inconsistent with the findings of previous studies (Figner et al., 2009; Grose-Fifer et al., 2014; Hämmerer et al., 2011; Zottoli & Grose-Fifer, 2012). There are some differences in sample and task characteristics that may explain the discrepant findings. We also note that previously differences in the FRN valence effect is very sensitive to the way it is measured. This sensitivity suggests that developmental differences in the valence effect is much weaker than the overall effect which could contribute to the discrepant findings.
With respect to the development of the FRN during adolescence and the adolescent-adult transition, studies mostly focus on the overall developmental reductions in the FRN and increases in the FRN valence effect. While these reductions are robust, reported differences in the FRN valence effect appear to be smaller and sensitive to the measurement of the valence effect. For example, Hammerer et al. (2011) reported significant FRN valence effects in all children, adolescent, young and older adult groups. When the FRN valence effect was measured on the difference waveform, an unusual developmental pattern was observed. FRN valence effects in adults differed from older adults and adolescents but showed comparable effect sizes in children. However, when measured as a ratio score, it was shown that young adults showed a greater difference compared to children, adolescents and older adults.

Similarly, Zottoli and Grose-Fifer (2012) also only reported a small but significant increase in FRN valence effect when measured as a ratio score but not on separate waveforms (gain vs. loss). Grose-Fifer et al., (2014) reported significant difference in FRN valence effect both on separate waveforms and ratio scores, with modest and large effect sizes respectively. The variability indicates that developmental differences in the valence effect is smaller than overall amplitude effects.

As this effect is relatively small, the absence of significant FRN valence effects could be due to sample differences, particularly the age gap between adolescents and adults. The current study focused on the adolescent-adult transition examining adolescents aged 14-17 years and adults aged 18 years and above, whilst previous studies used samples with large age gaps separating the adolescent and adult groups. In Hämmerer et al. (2011), relatively younger adolescents ($M_{\text{age}} = 14.03$ years) and older adults were recruited ($M_{\text{age}} = 24.12$ years). Discrepancies may be attributed to greater immaturity of feedback discrimination processes in younger adolescents and/or further development during young adulthood. Zottoli and Grose-Fifer (2012) and Grose-Fifer et al. (2014) also reported increases in the FRN valence effect in similarly aged adolescents ($M_{\text{age}} = 15.0$ years and $M_{\text{age}} = 15.1$ years) but relatively older adults ($M_{\text{age}} = 24.1$ years and $M_{\text{age}} = 28.2$ years). Collectively, these findings indicate that there are developmental changes in the FRN valence effect between adolescents and adults but this effect is relatively small and may only observable over longer
periods. Future studies examining the valence effect during this period should consider recruiting larger samples.

Task-differences between dynamic risky decision-making tasks

With respect to task differences, the absence of a significant FRN valence effect could be due to increased variability of trial characteristics in the BART (i.e. changes in the probability and the magnitude of outcomes across trials). As the FRN is sensitive to these characteristics, the trial-to-trial variability in the FRN could dilute the FRN valence effect observed on the individual averages, obscuring group effects. Despite higher variability in the FRN at the trial level, the morphology of the ERP waveform was similar across adolescent and adult groups and comparable with other ERP studies examining the feedback response, indicating a relative consistent feedback response within and across studies (Zottoli and Grose-Fifer, 2012; Grose-Fifer et al., 2014, Hammerer et al., 2011). Furthermore, significant group differences in the overall FRN and P2 amplitudes despite variability in trial characteristics indicate that these overall earlier effects are robust and insensitive to the variation in outcome probability, magnitude and valence.

With respect to risky decision-making, the behavioural results are inconsistent with findings showing heightened risk-taking in adolescents compared to adults. Of particular relevance, the results do not support the results obtained on an alternative dynamic risk-taking task, the Cambridge Card Task (Figner et al., 2009). Although both tasks can be described as dynamic risk-taking tasks due to the sequential change in outcome properties, there are some notable differences between the BART and the Cambridge Card Task which may explain the discrepancy in behavioural findings.

In the Cambridge Card Task participants are presented with an array of 32 cards and are informed that one card is associated with a negative outcome, whilst negative outcomes (bursts) in the BART are a single element within an unknown sized array. Although the Cambridge Card Task appears to be more descriptive, the outcomes are unpredictable as the task is programmed such that the loss card is not revealed until the end, a feature implemented to enable the detection of voluntarily stopping during the task. To give participants the impression they were performing a chance-based task, 9 trials of loss
outcomes were randomly interspersed among 54 trials. Therefore, while the task is sequential in nature, the risks do not accumulate across trials and the overall design of the task means that occurrence of negative outcomes is very low and is not dependent on behaviour. A recent study by Markiewicz, Kubińska and Tyszka, (2015) argued that the measure of risk-taking in the Cambridge Card Task is contaminated. While it may reflect risk preference, it is also sensitive to the decision-maker’s belief in trend continuation (i.e. their tendency to adopt a win-stay and lose-switch strategy). It was suggested that developmental differences between adolescents and adults might be due to developmental differences in belief in trend continuation rather than risk-taking propensity.

Given that enhanced P2/FRN showing a heightened responsiveness to overall external feedback, reduced negative feedback on the Cambridge Card Task would result in stronger reinforcement of risky decisions (card turns) supporting the trend continuation account. Therefore, the absence of developmental differences using the BART could be due to the presence of relatively frequent and consistent, negative feedback regulating responses. Collectively, this discussion highlights that different implementations of voluntary stopping may have a big impact on the pattern of results as they have an impact on learning from feedback.

*Frontocentral P3 subcomponent in response to feedback.*

With respect to feedback processing, a frontocentral P3 subcomponent distinct from the parietal P3 was observed in response to negative (burst) feedback. The distribution and relative time-course of this subcomponent bears similarities with the frontocentral P3 elicited following an infrequent stimulus in the oddball paradigm (Pires, Leitão, Guerrini, & Simões, 2014; Polich, 2007) and the frontocentral subcomponent of the error positivity (Pe) which is elicited following an erroneous response (Arbel & Donchin, 2009; Ruchsow, Spitzer, Gron, Grothe, & Kiefer, 2005; Van Veen & Carter, 2002). These components are thought to reflect deeper cognitive processing and the conscious evaluation of errors. However, there is a currently limited knowledge regarding the functional significance of the frontocentral P3 following feedback presentation. Therefore, we draw on these similar components to comment on the role of the feedback frontocentral P3 subcomponent.
In the oddball paradigm, the frontocentral P3 is thought to reflect focal attention processing of the current event. It has been shown that the amplitude of frontocentral P3 is sensitive to the stimulus probability, where larger amplitudes are observed on infrequent compared to frequently occurring stimuli (Pires et al. 2014; Polich, 2007). In error-monitoring however, the functional significance of the frontocentral Pe subcomponent is unclear. Based on observed similarities in the time-frequency characteristics, distribution and source of the signal, it has been suggested that the frontocentral Pe may be resolution or continuation of the error-related negativity (ERN) (Debener et al., 2005; Luu, Tucker, & Makeig, 2004; Van Veen & Carter, 2002), although there have been reports functionally differentiating the ERN and the frontocentral Pe (Arbel & Donchin, 2009; Endrass, Klawohn, Gruetzmann, Ischebeck, & Kathmann, 2012; Endrass, Klawohn, Preuss, & Kathmann, 2012; O’Connell et al., 2007). The ERN and frontocentral Pe have been examined under different experimental manipulations such as task difficulty, task instructions emphasising accuracy, and perception or awareness of trial outcomes, and these studies largely show that the ERN was sensitive but the frontocentral Pe was insensitive to these manipulations.

In feedback monitoring, recently a few studies have examined the frontocentral P3 subcomponent and found that it was sensitive to outcome likelihood, similar to the frontocentral P3 in oddball tasks. A recent meta-analysis by Sambrook and Goslin (2015) used cluster-based analyses and identified a frontocentral cluster following the FRN, spanning 209-500 ms, showing it was significantly modulated by outcome likelihood and control over the task. In terms of outcome likelihood, larger amplitudes were observed for unlikely outcomes. With respect to task control, larger effects were observed on probabilistic-learning tasks with informative feedback compared to passive or guess designs where feedback was uninformative. In a recent study by Walentowska, Moors, Paul, and Pourtois (2016), manipulated outcome probability/task relevance and found that the frontocentral subcomponent was sensitive to outcome likelihood, consistent with the function of the frontocentral P3. In the relevant feedback condition where feedback was informative (and positive feedback was infrequent), frontocentral P3 amplitudes to positive compared to negative feedback. In the irrelevant condition where feedback outcomes were uninformative (and outcomes were equiprobable), no significant frontocentral P3 effects were observed.
The sensitivity of the frontocentral P3 to outcome probability could suggest a potential role in the signalling a violation of expectancy or internal models of the task. Supporting this view, Mathewson, Dywan, Snyder, Tays, and Segalowitz, (2008) observed modulations in the P3 effect in a non-probabilistic learning task where the P3 was enhancement to negative feedback greater on the test compared to the learning phase of the task.

Collectively, the frontocentral P3 to feedback is sensitive to the probability of feedback outcomes, where amplitudes are enhanced for infrequently presented outcomes, similar to the frontocentral P3 in the oddball task. It is likely that the enhanced frontocentral P3 subcomponent on burst trials reflects the infrequent occurrence of bursts compared to successful pumps. Differences between stimulus (frontocentral P3 and frontocentral feedback P3) and response-related (frontocentral Pe) components could reflect differences between internal and external monitoring processes. Sensitivity to outcome probability in stimulus, but not response-related components, could be related to additional processing of an external stimulus and the integration of the extracted information with internal representations of the task.

Conclusion

This study showed evidence of ongoing changes while highlighting the relative maturity of feedback processes during the adolescent-adult transition. Enhanced P2 and FRN amplitudes were interpreted as reflecting a greater allocation of attentional processes, as similarities in risky decision-making, the FRN valence effect and P3 indicated that adolescents were proficient at processing feedback information. While these similarities highlight the relative maturity of feedback-related processes, they were inconsistent with previous findings. The discrepancies were attributed to a combination of sample and task differences. Comparison with previous findings suggest slow but ongoing development of the FRN valence effect throughout young adulthood. Dynamic risk-taking tasks such as the BART provides a novel way of examining risky decision-making and feedback processing, however future research should consider the impact of trial variability and risk-parameters on behavioural and ERP indices.
Supplementary Figures

Figure 4.4

*Feedback*-locked, grand-averaged waveforms and waveforms of microvolt-scaled factor loadings on successful pumps and bursts, as well as the scalp topography, percentage of total variance explained, and the peak latency of loadings for each factor.*
References


Chapter 5: Response Inhibition, Feedback Processing and Excessive Alcohol Use in Young Adults

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Abstract

Abnormalities in both response inhibition and feedback processing have been implicated in risk-taking behaviour, however, these studies are typically examined separately with few investigating their combined contribution. In the context of alcohol consumption, we compared behavioural and ERP measures of response inhibition, feedback processing and risky decision-making between low and high drinking young adults (low drinking group: $N = 37$, $M(SD)_{age} = 21.51(2.85)$ years; high drinking group: $N = 32$, $M(SD)_{age} = 20.07(2.62)$ years). Risky decision-making and electrophysiological indices of response inhibition and feedback processing were examined using a modified go/nogo task and the Balloon Analogue Risk Task (BART). A significantly lower number of adjusted average pumps was observed in the high drinking group compared to the low drinking group, suggesting a greater preference for immediate reward. While the lower adjusted number of average pumps in the high drinking group is unusual, they have been previously observed and could suggest an alternate pathway towards risk-taking. Despite behavioural differences, there were no differences in ERP indices of response inhibition or feedback processing. There were also no significant differences in personal sensitivity to external feedback, as measured by the Behavioural Inhibition and Activation System (BIS/BAS) scale. The absence of ERP and personal differences suggest that high drinking is not necessarily associated with abnormalities in inhibitory or feedback processing and may be driven by transient situational and environmental factors. The contribution of underlying inhibition and feedback-related processes in risk taking may be more apparent in early developing and dependent samples and impairments are greater and more generalised, and alcohol use is more persistent.
Introduction

Risk-taking and heavy alcohol consumption

Risk-taking behaviour has been defined in numerous ways and has been examined from different perspectives including decision-making and public health. In decision-making research, risk-taking has been operationalised as the preference for immediately appealing but long-term sub-optimal outcomes and as a preference for options with greater outcome uncertainty (Paulsen, Platt, Huettel, & Brannon, 2011). However, risk-taking has also been used to describe behaviours which are anti-social or self-destructive, often referring to various substance-use and delinquent behaviours examples of risk-taking (e.g. excessive alcohol consumption, smoking, illicit substance use, problem gambling, violence, theft, reckless driving, and unprotected sex) (Arnett, 1992; Reyna & Farley, 2006). Motivated by the potential negative impacts of these behaviours, researchers have been focused on characterising the nature of risk-taking.

The current study focuses on alcohol consumption which has been widely investigated in risk-taking literature due to its availability, association other risky behaviours and negative health and social outcomes. Globally in 2012, excessive alcohol consumption contributed to approximately 3.3 million deaths (or 5.9% of all deaths) and 5.1% of the global burden of disease (World Health Organization, 2014). In Australia, the National Health and Medical Research Council recommends consuming no more than 4 standard drinks (equivalent to 40g of alcohol) on a single occasion to reduce risk of alcohol-related harm in the short-term (National Health and Medical Research Council, 2009). Based on these guidelines, national surveys in 2014-2015 revealed that a substantial proportion of individuals consume alcohol above this level, increasing the risk for short-term harm. In young adults, 69.4% and 60.6% of males and females reported exceeding these guidelines at least once per year. Road traffic accidents are a notable consequence of excessive consumption or misuse of alcohol, where alcohol is reported to contribute to 8.38% to 14.95% of traffic accidents in Australia (Ferris, Devaney, Sparkeys-Carroll, & Davis, 2015).
Impaired response inhibition in heavy alcohol users

Response inhibition refers to the ability to suppress compelling but inappropriate responses. In the context of alcohol use, impaired inhibitory control is theorised to manifest as a reduced ability to decrease or regulate use, facilitating the development and maintenance of alcohol dependence. In the laboratory, response inhibition is commonly examined using the go/nogo and the stop-signal task where participants rapidly respond to frequently presented target stimuli whilst withholding responses to infrequently presented nogo stimuli/stop signals. Numerous studies have reported significant deficits in response inhibition (reflected by reductions in accuracy) in heavy and dependent alcohol using groups relative to controls (Bjork, Hommer, Grant, & Danube, 2004; Goudriaan, Oosterlaan, De Beurs, & Van Den Brink, 2005; Noël et al., 2007; Pandey et al., 2012; Rubio et al., 2007), with a recent meta-analysis by Smith et al. (2014) reporting a statistically significant combined effect across these investigations.

In addition to behavioural measures, numerous studies have also reported differences in underlying inhibitory processes reflected by event-related potentials (ERP) in both the go/nogo and stop-signal tasks. While there are similarities in ERPs elicited by the go/nogo and stop-signal tasks (Van Boxtel, Van der Molen, Jennings, & Brunia, 2001), fMRI studies have reported differences in the involvement of common brain regions between the two tasks (Chikazoe et al., 2009; Chikazoe, Konishi, Asari, Jimura, & Miyashita, 2007). A meta-analysis comparing fMRI activation between tasks found that although there were common clusters, the two tasks engaged distinct neural circuits (Swick, Ashley, & Turken, 2011). Therefore, the current study will focus on inhibitory processes reflected by the go/nogo task.

Research using the go/nogo task predominantly focuses on the frontocentral N2 and central P3 peaks elicited following the presentation of go and nogo stimuli. The amplitudes of both peaks are typically enhanced on nogo trials compared to go trials and these enhancements are thought to reflect inhibition-related processing. Although the N2 enhancement was initially implicated in response inhibition, subsequent studies found that this effect was not exclusive to trials that required inhibition (Donkers & Van Boxtel, 2004; Fox, Michie, Wynne, & Maybery, 2000; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003). It was later proposed that the N2 reflects conflict monitoring, a process
that detects when multiple incompatible representations are simultaneously activated (Botvinick, Braver, Barch, Carter, & Cohen, 2001). On the other hand, central P3 enhancements have been shown to be more specific to trials that required response inhibition. With researchers finding that P3 enhancements were only observed when participants were required to change responses or suppress a planned response (Randall & Smith, 2011; Smith, 2011; Smith, Johnstone, & Barry, 2007; 2008). As such, the enhancement of P3 amplitude in inhibition trials was thought to reflect inhibitory processing.

Several studies using the go/nogo task have reported differences in inhibitory processes between alcohol dependent and at-risk groups compared to healthy control (Cohen, Porjesz, Begleiter, & Wang, 1997; Colrain et al., 2011; Fallgatter, Wiesbeck, Weijers, Boening, & Strik, 1998; Kamarajan et al., 2005; Karch et al., 2008; Luijten et al., 2014; Pandey et al., 2012; Pfefferbaum, Rosenbloom, & Ford, 1987). With respect to P3, studies have reported reductions in go and/or nogo P3 amplitude, reflecting deficits in response production and inhibitory control respectively. Diminished overall P3 amplitudes and nogo P3 effects were reported in individuals with a family history of alcohol dependence compared to those without, reflecting deficits in response production and inhibitory control (Cohen et al., 1997; Colrain et al., 2011; Kamarajan et al., 2005; Pfefferbaum et al., 1987). Studies have also reported significant reductions in the N2 amplitude, suggesting abnormalities in conflict monitoring (Cristini, 2003; Pandey et al., 2012). Deficits in conflict monitoring and inhibitory processes, reflected by reductions in the N2 and P3 amplitude, have also been reported in cocaine and internet addiction (Dong, Lu, Zhou, & Zhao, 2010; Sokhadze, Stewart, Hollifield, & Tasman, 2008).

Although the aforementioned studies reported reductions in performance and ERP amplitude in alcohol dependent and other addictive behavioural groups, not all studies report significant reductions in P3 amplitude (Fallgatter et al., 1998; Karch et al., 2008) and some ERP studies examining opiate and behavioural addictions (excessive internet use and gaming) have reported enhanced Go N2, Nogo N2 and Nogo P3 amplitudes interpreted as compensatory activation (Dong et al., 2010; Littel et al., 2012; Yang et al., 2009). Furthermore, brain-behaviour associations are infrequently observed, contributing to
difficulties interpreting the functional significance of reported ERPs (Colrain et al., 2011; Fallgatter et al., 1998; Karch et al., 2008; Pfefferbaum et al., 1987).

**Abnormal feedback processing in heavy alcohol users**

In addition to inhibitory deficits, heavy alcohol use and alcohol dependence has also been associated with abnormalities in feedback-related processing. The ability to process and learn from external feedback and to adjust behaviours accordingly is important for adapting to novel situations. It has been hypothesised that abnormalities in these processes could result in abnormal reinforcement of substance use behaviours or insensitivity to negative outcomes associated with substance-use and interfere with the ability to regulate substance use (Everitt & Robbins, 2005). Feedback processing is typically studied by examining the neural response to feedback on risky decision-making tasks where participants make decisions under conditions of uncertainty and are required to learn response-outcome associations.

In ERP studies, feedback-related processes are reflected by the feedback-related negativity (FRN) and parietal P3 elicited to feedback presentation. The FRN refers to a frontocentrally distributed negative peak that occurs approximately 200-300 ms following feedback presentation. According to the reinforcement-learning theory, the FRN reflects the encoding of a reward prediction error signal by the anterior cingulate cortex (Holroyd & Coles, 2002). The FRN amplitude is sensitive to valence, with enhanced amplitudes following negative feedback than positive feedback. Its amplitude is also sensitive to other characteristics such as outcome likelihood and magnitude (Eppinger, Mock, & Kray, 2009; Sambrook & Goslin, 2015; Yi et al., 2012). As such, the FRN is thought to reflect processing and discrimination of outcomes. The parietal feedback P3 is another measure used to examine reward-related processing and refers to a parietally distributed positive peak that occurs approximately 300-600 ms following feedback. This component is sensitive to outcome likelihood and the magnitude of outcomes, with a few studies also reporting sensitivity to valence (Hajcak, Moser, Holroyd, & Simons, 2007; Sato et al., 2005; Wu & Zhou, 2009; Yeung & Sanfey, 2004). Modulation of this component to magnitude is hypothesised to reflect deeper evaluation, indexing trial utility and/or motivation salience.
Several ERP studies that have reported significant differences in the FRN in alcohol and substance dependent groups, in at-risk and other risk-taking groups (Fein & Chang, 2008; Kamarajan et al., 2010; 2015; Parvaz et al., 2015; Yau, Potenza, Mayes, & Crowley, 2015). In alcohol dependent males, Kamarajan et al. (2010) reported significant reductions in FRN and feedback P3 amplitudes suggesting abnormalities in the processing and evaluation of feedback. Reductions in FRN amplitude have also been observed in individuals with cocaine use disorder and in problematic internet users (Parvaz et al., 2015; Yau et al., 2015). Similar modulations in FRN and feedback P3 have also been observed in high-risk populations, highlighting the role of genetics and predisposition. Fein and Chang (2008) reported that greater family history density of alcoholism was associated with a reduction in FRN amplitude, and Kamarajan et al. (2015) observed reduced feedback P3 amplitudes in offspring of alcoholics. Although studies in risk-taking groups have reported reductions in FRN and feedback P3 in risk-taking groups indicating reduced ability to evaluate, studies examining individual differences in typical adult samples have reported that increases in behavioural approach and unplanned impulsivity, which are typically elevated in risky groups, is associated with enhancements in FRN amplitude (Lange, Leue, & Beauducel, 2012; Onoda, Abe, & Yamaguchi, 2010).

Collectively, ERP studies examining response inhibition and feedback processing in alcohol dependent and other addictive groups reported reduced amplitudes on both inhibition and feedback monitoring measures. However, there are noted inconsistencies in the pattern of results and infrequent observations of brain-behaviour associations. In the go/nogo task, the absence of these effects could be attributed to low task difficulty resulting in a lack of variability in accuracy, reducing sensitivity to differences in performance. Variability in accuracy is affected by the ratio of go to nogo trials, with low go-to- nogo ratios having reduced task difficulty due to a weaker tendency to respond. In feedback processing, the absence of brain-behaviour associations can be attributed to existing evidence being largely derived from unpredictable gambling tasks where the probabilities of all outcomes are equal. While this allows for examination of the general responsiveness and sensitivity to different outcomes, the feedback during these tasks are largely uninformative and may not reflect processes associated with learning from feedback which is important in risk-taking behaviour. In addition to task factors, inconsistencies between studies could also be due to a
combination of inhibition and feedback processing deficits that are not represented when independently examining these systems. A model of the interaction of these systems has been proposed in the developmental literature.

Neurodevelopment of inhibition and feedback processing, and risk-taking

The ability to regulate behaviours and process external feedback has also been implicated in developmental change in risk-taking, notably the observation of heightened risk-taking during adolescence. It is proposed that the normal but divergent development of these systems produces a bias towards risk-taking, where the coupling of an early maturing, hyperactive reward processing system with an immature cognitive control system results in increased but under-regulated tendency to pursue rewarding stimuli (Shulman et al., 2016). This model highlights the interaction between response inhibition and feedback processing. While alcohol dependence studies have reported impairments in underlying inhibitory and feedback processes, not all studies have reported this. Examining both processes could provide insight into the relationship between heavy alcohol use, risk-taking and underlying inhibitory and feedback processing.

Current study

The current study aimed to further understand the role of underlying inhibitory and feedback processing in heavy alcohol consumption. Building upon some limitations of previous research, the current study uses a modified go/nogo task that is sensitive to partial responses and has a high go-to-nogo ratio to increase sensitivity to performance differences. Highlighting its utility, Chapters 2 and 3 showed partial inhibitions accounted for a substantial proportion of errors on nogo trials and measuring partial inhibitions allowed for the discrimination between mid-to-late adolescents and young adults. In terms of feedback processing, the current study used a dynamic risky decision-making task called the Balloon Analogue Risk Task (BART). The dynamic nature of this task simulates the novelty of real-world risk-taking, contrasting with previous investigations which predominantly use unpredictable gambling tasks to examine feedback processing. Supporting the validity of the BART as a measure of risk-taking numerous studies have reported behavioural differences in the number of adjusted average pumps in various risky groups (Bornovalova, Daughters,
Hernandez, Richards, & Lejuez, 2005; Coffey, Schumacher, Baschnagel, Hawk, & Holloman, 2011; Hopko et al., 2006; Ledgerwood, Alessi, Phoenix, & Petry, 2009; Lejuez et al., 2002, 2003; Lejuez, Simmons, Aklin, Daughters, & Dvir, 2004). Unlike unpredictable gambling tasks, the task provides informative feedback which requires use external feedback on previous trials to inform future decisions, allowing the examination of behavioural differences in risky decision-making.

To examine whether heavy alcohol use may be linked to variations in underlying inhibitory and feedback processing, the current study examines both behavioural and ERP indices of inhibition (nogo N2 and P3) and reward processing (FRN and feedback P3). In line with alcohol dependence literature, the heavy drinking group is expected to show poorer response inhibition and feedback processing, reflected by reduced accuracy and reduced N2 and P3 amplitude in the go/nogo task, some increased adjusted average numbers of pumps, diminished FRN and parietal feedback P3 amplitudes on the BART respectively.

Alternatively, and in line with the neurodevelopmental model, high alcohol use could be due to the coupling of poor inhibition with a heightened sensitivity to external feedback, reflected by reduced N2 and P3 amplitude in the go/nogo task and enhanced overall FRN amplitude in the BART.

In addition to inhibition and feedback processes, the current study also examined the anterior P2 on both tasks which has been associated with orientation of attention to the stimulus. It was demonstrated in Chapters 3 and 4 that differences in early attention reflected by modulations in P2 revealed impulsive responding in adolescents during the go/nogo task and suggesting a heightened sensitivity to overall feedback in adolescents during the BART. The measurement of P2 could provide useful insight into task approach during response inhibition and feedback processing in low and high alcohol use. If high drinking is driven by deficits in early attentional processing during inhibitory processing, we would expect delayed P2 latency in the go/nogo task. If high drinking is driven by greater attention allocation towards external feedback, we expect enhanced P2 amplitudes to feedback presentation in the BART.
Methods

Participants

The sample consisted of thirty-seven low drinkers ($M(SD)_{age} = 21.51(2.85)$ years, 18 female) and thirty-two high drinkers ($M(SD)_{age} = 20.07(2.62)$ years, 17 females). Participants consisted of psychology undergraduates from the University of Western Australia and volunteers from the local community. Participants were classified into low and high drinking groups, after recruitment. All participants provided informed consent prior to participation and the protocol was approved by the human research ethics committee of the University of Western Australia. All participants had normal or corrected vision and no participants were colour blind. Four participants were excluded due to missing data.

Low and high drinkers were defined according to the Australian National Health and Medical Research Council (National Health and Medical Research Council, 2009) where it is recommended that both men and women that drinking no more than 4 standard drinks on a single occasion will reduce the risk of alcohol-related injury arising from a single occasion, and drinking no more than 2 standard drinks on a single day reduces the lifetime risk of alcohol-related harm. Low and high drinkers were identified based on the self-reported frequency and quantity of alcohol consumption on the first three items of the Alcohol Use Identification Disorders Test (AUDIT). NHMRC guidelines have been previously used to define low and high drinkers, showing differences in response inhibition in young female drinkers (Smith & Mattick, 2013). These items were used to focus on consumption behaviour, rather than perceptions and consequences of alcohol use. High drinkers were identified as individuals who reported drinking 5-6 drinks between 2-4 times a month, and/or indicating consuming more than 6 drinks per occasion. While the high-drinking group reflects a risky level of consumption by NHMRC standards, it should be noted that participants were not clinically-diagnosed with alcohol dependence. Individuals who reported consuming than 1-4 drinks per occasion were identified as low drinkers. Individuals who reported consuming a greater quantity per occasion but indicated that the frequency was less than monthly were identified as low drinkers.
The gender composition was similar between low and high drinking groups (49% and 53% females in the low and high drinking group respectively) and initial analysis showed that there were no significant differences in cognitive ability between the low and high drinking groups, as estimated by the Wechsler’s Abbreviated Test of Intelligence – Second Edition ($t(67) = -1.27, p = .208, d = 0.31$).

**Materials**

*Wechsler Abbreviated Scale of Intelligence – Second Edition (WASI-II, Wechsler, 1999)*

The full-scale IQ-2 score from the WASI-II was used to provide an estimate of general cognitive ability. This full-scale IQ–2 score is a normed composite of vocabulary and matrix reasoning subtest scores. In the vocabulary subtest, participants named pictures and/or provided definitions of words that were presented visually and orally, and more points were awarded for correct and clear definitions. This subtest is designed to provide a measure of word knowledge and verbal concept formation. In the matrix reasoning subtest, incomplete arrays were presented visually and participants were required to select the response option that completes the array. This subtest requires participants to identify perceptual patterns and taps various aspects of intelligence including fluid intelligence, visuospatial ability, and perceptual organisation.

*Alcohol-Use Disorder Identification Test (AUDIT, Saunders, Aasland, Babor, de la Fuente, & Grant, 1993)*

The AUDIT is a self-report questionnaire used to screen for disorders related to alcohol-use. The full questionnaire consists of 10 items inquiring about the quantity and frequency of alcohol consumed, dependence on alcohol and consequences of drinking. In the current study, only scores from the first three items were used which inquired about the quantity and frequency of alcohol consumption. Participants indicated their responses to the first 3 items on a 5-point ordinal scale.

*Behavioural Inhibition and Activation System Scale (BIS/BAS scale, Carver & White, 1994)*
The BIS/BAS scale is a self-report questionnaire which provides a measure of personal sensitivity to external feedback. This measure was incorporated to examine whether there were differences in the responsiveness to rewarding stimuli or differences in sensation seeking which have been previously implicated in risky decision-making (Lauriola, Panno, Levin, & Lejuez, 2014). This scale was initially developed to examine the behavioural approach and inhibition systems, which are general motivational systems proposed by Gray’s biopsychological theory of personality and the reinforcement sensitivity theory (Gray & McNaughton, 2003). These motivational systems were thought to underpin goal-directed behaviour and the affective response to reward and punishment. The scale consists of 24 items measured on a four-point scale with no neutral response (1 = very true for me, 4 = very false for me). Items measuring the responsiveness of the behavioural activation system (BAS subscale) can be split into three subscales focusing on sensitivity to rewards, motivation to pursue reward and positive affect (reward responsiveness, drive and fun seeking subscales). The responsiveness of behavioural inhibition system is measured on a single scale with items focusing on sensitivity to punishment and avoidant tendencies (BIS subscale). Scores on the BAS subscale can range from 4 to 52 and scores on the BIS subscale can range from 4 to 28, with higher scores indicating a more responsive behavioural approach/inhibition system.

*Modified go/nogo task (Cragg & Nation, 2008)*

This task was originally designed by Cragg and Nation (2008) and is sensitive to partial responses. A modification version by Nguyen, Moyle and Fox (2016) was used in the current study to counterbalance the stimuli associated with go and nogo responses. On each trial, either soccer ball or a rugby ball was presented. Across participants, pairings between the soccer/rugby balls with the go/nogo stimuli were mixed. Participants were instructed to respond to go stimuli and withhold their responses to nogo stimuli. They responded to go stimuli by releasing the left-mouse button then pressing the right-mouse button. On nogo trials, participants withheld their responses by keeping the left-mouse button held down. Partial responses were identified as nogo trials where the left-mouse button was released but the right-mouse button was not pressed. Participants were instructed to use their right index finger to control both buttons, and respond as quickly and accurately as possible.
Participants completed two practice blocks of 30 go trials to encourage the development of a pre-potent response. Following the completion of the practice blocks, participants completed an additional two blocks, containing 75 go and 25 nogo trials in each block. Each stimulus was presented for 200 ms. For the first two practice blocks, variable inter-stimulus intervals were used to prevent the prediction of stimulus onset and to discourage anticipatory responding. The inter-stimulus intervals were selected randomly between 1600, 1800, 2200 and 2400 ms. For the two proper blocks, a fixed inter-stimulus interval of 2000 ms was used to encourage a regular pattern of responding.

![Example of background stimulus (left), soccer ball (middle) and rugby ball (right).](image)

Figure 5.1 Example of background stimulus (left), soccer ball (middle) and rugby ball (right).

**Balloon Analogue Risk Task (BART, Lejuez et al., 2002)**

The BART is a dynamic risky decision-making task that was designed by Lejuez et al. (2002) to simulate the novelty of risk-taking in the real world. The current task was based on a modified version used in Fein and Chang (2008) to allow for ERP analysis. Participants were presented with balloons on screen and they repeatedly indicated whether they want to inflate the balloon. The initial value of each balloon was $0.00 and this value increased with each successful pump. Although successful pumps increase the value of the balloon, this value is only transferred to their earnings when the participant chooses to cash-out. Successful inflations are also accompanied with an increase in the probability that the balloon will burst. If the pump is unsuccessful and the balloon bursts, the accumulated value is lost and the next balloon is presented. If the participant opted to cash-out, value of the balloon is transferred to their earnings and the next balloon is presented. Participants completed 50 balloons in a single block. They were informed that the burst probability
increased with each successive pump but were not informed about the actual probabilities or total number of balloons.

The maximum pumps possible for each balloon was shortened to 20 pumps to allow for implementation of ERP analyses. Successful pumps resulted in 5 cents increase in value. The probability of the balloon bursting after the first pump is $1/20$, $1/19$ for the second pump and so forth until the 20th pump when a burst outcome is certain. The stimulus remained on the screen until a response was made. Following each pump, visual feedback presented in white text over a black background for 500ms indicating updated value of the balloon or if it had burst. Following the end of a balloon (cash-out burst), additional feedback was presented for 500ms indicating the total money earned or that the accumulated value was lost.
Figure 5.2 Diagram depicting trial progression for possible responses (pump, cash-out) and feedback outcomes (successful pump, burst and cash-out) in the Balloon Analogue Risk Task
Procedure

Participants completed two subtests of the Wechsler Abbreviated Scale of Intelligence – Second Edition (WASI-II). Following this, participants were prepared for the EEG recording and completed the Alcohol-Use Disorder Identification Test (AUDIT) questionnaire during the cap application process. While the EEG was continuously recording, participants completed EEG-adapted version of the BART followed by a modified go/nogo task. The duration of the entire experiment was approximately 90 minutes and participants were provided with an opportunity to rest between and within each task.

Data acquisition

EEG data were acquired using SCAN 4.3.3 ™ and processed offline using EEGLAB 13.3.2 (Delorme & Makeig, 2004) and ERPLAB 5.0.0 (Lopez-Calderon & Luck, 2014). The data were recorded using Ag/AgCl electrodes at 33 scalp locations (FP1, FP2, F3, F4, F7, F8, Fz, FC1, FC2, FC5, FC6, FCz, FT9, FT10, C3, C4, Cz, T7, T8, CP1, CP2, CP5, CP6, P3, P4, P7, P8, Pz, PO9, PO10, O1, O2, Iz). Additional electrodes were placed at the left and right mastoid regions, where the right mastoid electrode was set as the online reference. Electrodes were also placed 2 cm above and below the left eye to record ocular movement, and an electrode placed at AFz was set as the ground. The online EEG was amplified using a NuAmps 40-channel amplifier, digitised at a sampling rate of 250 Hz and filtered online using a 0.05 – 30 Hz bandpass filter.

Offline processing of the go/nogo and the BART were conducted independently. The EEG data were filtered offline using a 0.5 Hz high-pass Butterworth filter (12 dB roll-off). Ocular artifacts were corrected using independent components analysis and guided by SASICA to identify and remove components associated with ocular muscle activity (Chaumon, Bishop, & Busch, 2015). Channels with excessive noise were spherically interpolated. On the go/nogo task, one channel was interpolated for ten participants, two channels for five participants, and three and six channels for one participant. On the BART, one channel was interpolated for seventeen participants and two channels for eight participants. EEG data were re-referenced offline to a common averaged reference.
On correctly identified go trials from the go/nogo task, the average number of epochs included in the individual average ERP waveforms were 144 (SD = 9) and 146 (SD = 6) for low and high drinking groups respectively. On successfully inhibited nogo trials, an average of 39 (SD = 8) and 39 (SD = 6) epochs were included. The number of epochs in each condition did not differ significantly between drinking groups (correct go, \( t(67) = -0.90, p = .372, d = 0.22 \); successful inhibition, \( t(67) = -0.13, p = .896, d = 0.03 \)). On trials with feedback indicating successful pumps from the BART, the average number of epochs included were 310 (SD = 78) and 284 (SD = 61) for low and high drinking groups. On burst feedback, the average number of epochs included were 21 (SD = 6) and 19 (SD = 4). The number of epochs did not differ between drinking groups on feedback indicating successful pumps, but low drinking group statistically significant greater number of epochs for burst feedback (successful pump, \( t(67) = 1.55, p = .125, d = 0.38 \); bursts, \( t(67) = 2.07, p = .042, d = 0.50 \))

Measurement of Event-Related Potentials

Mean amplitudes were calculated over a 40 ms window centred on peaks of the grand-averaged waveform. This short interval was used to minimise the interference of adjacent components. To account for potential latency variability on mean amplitude across conditions and drinking groups, we computed separate latency windows for each condition and drinking group. Within each condition and drinking group, ERP amplitudes at each site were measured using the same interval. This was achieved by averaging the identified windows across the extracted midline sites. The latency windows used to measure mean amplitudes in the go/nogo and the BART are specified in Table 1.

Modified go/nogo task: The P2, N2 and P3 were measured at Fz, FCz, and Cz, and the P3 was also measured at Pz. The P2 peak was identified as the most positive point between 130-230 ms post-stimulus onset. The N2 peak was identified as the most negative

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4 To examine whether differences in epoch number resulted in greater noise in the high drinking group, we compared noise in the EEG between drinking groups, as measured by the standard deviation of EEG amplitude in the baseline interval. A mixed ANOVA was conducted with intended sites of interest (Fz, FCz, Cz, Pz) and age group (low drinking, high drinking) as within- and between-subject factors. No significant main effect of drinking group and no drinking group × site interaction was observed indicating there were no statistically differences in EEG noise between drinking groups.
point between 200-350 ms and the P3 peak was identified as the most positive point between 250-450 ms. P2, N2 and P3 peak latencies for each individual were measured using the latency windows above. However, P3 peak latency on correctly identified go trials were not analysed due to the lack of clear peaks on all participants.

**BART:** On the grand-averaged waveform, feedback P2 peak was identified as the most positive points between 150-250 ms post-feedback presentation. To maintain consistency in the measurement of the FRN with previous research, FRN was measured in 3 ways: (1) as the negative peak following separate burst and successful pump feedback, (2) as a ratio of change between burst and successful pump feedback [(FRN_{burst} − FRN_{successful pump})/FRN_{successful pump}] and (3) as the relative negativity between feedback outcomes, reflected on the ‘burst-minus-successful pump’ difference waveform. The FRN peak on separate and difference waveforms were identified as the most negative point between 200-350 ms post-feedback presentation. In chapter 4, a principal components analysis of feedback ERPs showed distinct frontocentral and parietal P3 subcomponents, reflected by two positive peaks following the FRN. It was suggested that the earlier frontocentral subcomponent reflected outcome likelihood (Sambrook & Goslin, 2015; Walentowska, Moors, Paul, & Pourtois, 2016), whereas previous research suggests that the parietal subcomponent reflects deeper evaluation and motivational salience (Kardos et al., 2016; Yau et al., 2015). To examine motivational salience, the current study focused on the parietal P3 subcomponent. The parietal feedback P3 was identified as the most positive time-point between 600-800 ms following burst feedback and ‘burst minus successful pump’ difference waveform. The parietal P3 was not measured on successful pumps due to lack of a distinct positive peak.
Table 5.1

Mean amplitude latency windows for ERPs in the go/nogo task (left) and the BART (right).

Latencies are with respect to the onset of the stimulus.

<table>
<thead>
<tr>
<th>ERPs</th>
<th>Condition</th>
<th>Drinking group</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Modified go/nogo task</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>P2</td>
<td>Correct go</td>
<td>128-168 ms</td>
</tr>
<tr>
<td></td>
<td>Successful inhibition</td>
<td>152-192 ms</td>
</tr>
<tr>
<td>N2</td>
<td>Correct go</td>
<td>216-256 ms</td>
</tr>
<tr>
<td></td>
<td>Successful inhibition</td>
<td>224-264 ms</td>
</tr>
<tr>
<td>P3</td>
<td>Correct go</td>
<td>340-380 ms</td>
</tr>
<tr>
<td></td>
<td>Successful inhibition</td>
<td>352-392 ms</td>
</tr>
<tr>
<td><strong>Balloon analogue risk task</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feedback P2</td>
<td>Successful inhibition</td>
<td>180-220 ms</td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>184-224 ms</td>
</tr>
<tr>
<td>FRN</td>
<td>Successful inhibition</td>
<td>284-324 ms</td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>272-312 ms</td>
</tr>
<tr>
<td></td>
<td>Burst – Successful inhibition</td>
<td>264-304 ms</td>
</tr>
<tr>
<td>Parietal Feedback P3</td>
<td>Burst</td>
<td>668-708 ms</td>
</tr>
<tr>
<td></td>
<td>Burst – Successful inhibition</td>
<td>668-708 ms</td>
</tr>
</tbody>
</table>

Data analysis

Preliminary analyses were conducted to ensure the data were appropriate for further analyses. Across all variables and participants, 47 outliers were identified and winsorized to 2.2 times the interquartile range (Hoaglin & Iglewicz, 1987). All variables were normality distributed, as assessed by examining skew and kurtosis coefficients against critical thresholds of |2| and |7| (Curran, West, & Finch, 1996). Behavioural and ERP data were analysed using mixed ANOVAs. Greenhouse-Geisser corrected p-values were reported for effects where the assumption of sphericity was violated.

**BIS/BAS scale:** BIS and BAS total scores, and the reward responsiveness, fun seeking and drive subscales of the BAS were analysed using independent samples t-tests, comparing low and high drinking groups.

**Modified go/nogo task:** Task accuracy and the speed of responses were analysed using 2×2 mixed ANOVAs with condition (correct go, partial inhibitions) as the within-subjects factor and drinking group (low drinking, high drinking) as the between-subjects
factor. The mean amplitude and peak latency of the P2 and N2 were analysed using a 3×2×2 mixed ANOVA with site (Fz, FCz, Cz) and condition as the within-subjects factor, and drinking group as the between-subjects factor. P3 mean amplitude was analysed with an additional site (Pz) using a 4×2×2 mixed ANOVA. P3 peak latency was analysed using a 4×2 mixed ANOVA with site and drinking group as within- and between-subject factors. P3 latency was not analysed on correctly identified go trials due the lack of clear peaks on each individual. Significant main effects of site were followed by paired sample t-tests comparing adjacent sites and the sequential Holm-Bonferroni method was used to adjust for the accumulation of type-I error for multiple comparisons (Holm, 1979). Significant site × condition and site × drinking group interactions which suggest differences in the scalp distribution were followed with one-way ANOVAs, conducted on each condition and drinking group separately. The pattern of within-subject contrasts was used to interpret the data. Significant three-way interactions were followed with two-way ANOVAs conducted on each drinking group separately. To investigate whether individual differences in conflict monitoring and response inhibition were significant predictors of risky drinking, a multiple regression analysis was conducted with N2 (FCz) and P3 (Cz) mean amplitude as predictors and risky drinking as the dependent variable.

**BART:** The adjusted average pumps and the average number of bursts between the low and high drinking groups were compared using independent samples t-tests. FRN and P2 mean amplitude were analysed using 3×2×2 mixed ANOVA with site (Fz, FCz, Cz) and feedback valence (successful pump, burst) as within-subjects factors and drinking group (low drinking, high drinking) as the between-subjects factor. Significant main effects of site and site × feedback valence and site × drinking group interactions were followed using the same method as in the modified go/nogo task. For consistency with previous research, analyses of FRN amplitude was also measured on the ‘burst minus successful pump’ difference waveform and FRN was also represented as a ratio of change. These FRN measures were analysed using two 3×2 mixed ANOVAs with site and drinking group as within- and between-subject factors respectively. The parietal feedback P3 measured on the burst and the ‘burst minus successful pump’ difference waveform were analysed using two 4×2 mixed ANOVAs with site (Fz, FCz, Cz, Pz) as within-subjects factor and drinking group as the between-subjects variable. To investigate whether individual differences in early stimulus
processing and feedback monitoring was significant predictors of risky drinking, a regression analysis was conducted with FRN difference amplitude (Cz) as the predictor variable and risky drinking as the dependent variable.

Results

Behavioural Results

No significant group differences were observed on BIS/BAS scale, including BIS and BAS total score, and reward responsiveness, fun seeking and drive subscales of the BAS. In the go/nogo task, overall accuracy and speed of responses did not differ significantly between drinking groups. Accuracy was higher on go compared to nogo trials, and partial responses were faster than correct responses to go trials. There were also no significant group differences in pattern of results across task conditions. In the BART, the high drinking group showed reduced adjusted average pumps and bursts compared to the low drinking group (See Table 5.2).
Table 5.2

Independent sample t-tests and ANOVA effects for the BIS/BAS scale, behavioural performance indices for the modified go/nogo task and the Balloon Analogue Risk Task.

<table>
<thead>
<tr>
<th>Measure</th>
<th>Condition</th>
<th>Drinking group</th>
<th>ANOVA contrast</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Low drinking</td>
<td>High drinking</td>
<td></td>
</tr>
<tr>
<td><strong>BIS/BAS scale</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BIS Total Score</td>
<td></td>
<td>21(5)</td>
<td>20(5)</td>
<td>(t(67) = 0.90, p = .369, d = 0.21)</td>
</tr>
<tr>
<td>BAS Total Score</td>
<td></td>
<td>38(7)</td>
<td>38(7)</td>
<td>(t(67) = 0.55, p = .586, d = 0.13)</td>
</tr>
<tr>
<td>Reward responsiveness</td>
<td></td>
<td>16(3)</td>
<td>16(4)</td>
<td>(t(67) = 0.37, p = .714, d = 0.09)</td>
</tr>
<tr>
<td>Fun Seeking</td>
<td></td>
<td>11(3)</td>
<td>11(3)</td>
<td>(t(67) = -0.24, p = .811, d = 0.06)</td>
</tr>
<tr>
<td>Drive</td>
<td></td>
<td>11(2)</td>
<td>10(2)</td>
<td>(t(67) = 1.47, p = .146, d = 0.36)</td>
</tr>
<tr>
<td><strong>Modified go/nogo task</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proportion of error</td>
<td>Go</td>
<td>0.02(0.02)</td>
<td>0.02(0.02)</td>
<td>(F(1,67) = 0.08, p = .776, \eta^2_p = .001)</td>
</tr>
<tr>
<td></td>
<td>Nogo</td>
<td>0.20(0.15)</td>
<td>0.21(0.10)</td>
<td>(F(1,67) = 158.97, p &lt; .001, \eta^2_p = .704^*)</td>
</tr>
<tr>
<td>Release time</td>
<td>Correct go</td>
<td>271(36)</td>
<td>272(27)</td>
<td>(F(1,67) = 0.24, p = .629, \eta^2_p = .004)</td>
</tr>
<tr>
<td></td>
<td>Partial inhibition</td>
<td>257(59)</td>
<td>245(35)</td>
<td>(F(1,67) = 32.59, p &lt; .001, \eta^2_p = .327^*)</td>
</tr>
<tr>
<td><strong>Balloon Analogue Risk Task</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted average pumps</td>
<td></td>
<td>7.84(2.16)</td>
<td>6.72(1.60)</td>
<td>(t(67) = 2.43, p = .018, d = 0.63^*)</td>
</tr>
<tr>
<td>Number of bursts</td>
<td></td>
<td>21.62(5.40)</td>
<td>19.19(3.86)</td>
<td>(t(67) = 2.12, p = .037, d = 0.51^*)</td>
</tr>
</tbody>
</table>
Electrophysiological Results: Go/nogo task

Figure 5.3 LEFT: Stimulus-locked, grand-averaged waveforms at Fz, FCz, Cz and Pz depicting correctly identified go trials (solid green line) and successfully inhibited nogo trials (dashed red line) in low drinking (left) and high drinking groups (right). Voltage (μV) is represented on the vertical axis and time (ms) is represented on the horizontal axis. RIGHT: Scalp distributions of the P2, N2 and P3 in the low and high drinking group (Latency windows are specified in Table 5.1).
Table 5.3
Mean and standard deviation of mean amplitude (μV) and peak latency (ms) of the P2, N2 and P3.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Site</th>
<th>P2 mean amplitude</th>
<th>P2 peak latency</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Low drinking</td>
<td>High drinking</td>
</tr>
<tr>
<td>Correct Go</td>
<td>Fz</td>
<td>1.75 (2.41)</td>
<td>0.74 (1.58)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>2.11 (2.34)</td>
<td>1.11 (1.48)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>2.19 (2.24)</td>
<td>1.31 (1.48)</td>
</tr>
<tr>
<td>Successful Inhibition</td>
<td>Fz</td>
<td>2.01 (3.04)</td>
<td>0.84 (2.28)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>1.87 (3.08)</td>
<td>1.20 (2.31)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>1.50 (3.04)</td>
<td>1.28 (1.93)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correct Go</td>
<td>Fz</td>
<td>-3.12 (2.30)</td>
<td>-3.37 (2.48)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>-2.90 (2.86)</td>
<td>-3.16 (2.65)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>-1.14 (3.02)</td>
<td>-1.13 (2.60)</td>
</tr>
<tr>
<td>Successful Inhibition</td>
<td>Fz</td>
<td>-3.81 (3.08)</td>
<td>-4.92 (3.83)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>-3.79 (3.20)</td>
<td>-5.18 (3.93)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>-2.03 (3.35)</td>
<td>-3.10 (3.82)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Correct Go</td>
<td>Fz</td>
<td>-0.55 (2.07)</td>
<td>-0.78 (3.10)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>0.12 (2.07)</td>
<td>0.15 (2.64)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>1.14 (2.39)</td>
<td>1.31 (2.11)</td>
</tr>
<tr>
<td></td>
<td>Pz</td>
<td>3.17 (1.93)</td>
<td>3.56 (2.74)</td>
</tr>
<tr>
<td>Successful Inhibition</td>
<td>Fz</td>
<td>3.56 (2.74)</td>
<td>2.13 (3.70)</td>
</tr>
<tr>
<td></td>
<td>FCz</td>
<td>6.91 (4.68)</td>
<td>6.98 (3.65)</td>
</tr>
<tr>
<td></td>
<td>Cz</td>
<td>7.33 (3.57)</td>
<td>7.40 (3.22)</td>
</tr>
<tr>
<td></td>
<td>Pz</td>
<td>5.12 (2.53)</td>
<td>5.60 (3.12)</td>
</tr>
</tbody>
</table>
Table 5.4

ANOVA effects of drinking group (low drinking, high drinking), site (Fz, FCz, Cz, Pz) and condition (Correct go, Successful inhibition) for P2, N2 and P3 ERP components

<table>
<thead>
<tr>
<th>ANOVA effects</th>
<th>ERP</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amplitude</strong></td>
<td>P2</td>
<td>N2</td>
<td>P3</td>
</tr>
<tr>
<td><strong>Main effects</strong></td>
<td>F(1,67) = 2.86, p = .096, η² = .041</td>
<td>F(1,67) = 1.20, p = .277, η² = .018</td>
<td>F(1,67) &lt; 0.01, p = .957, η² &lt; .001</td>
</tr>
<tr>
<td>Drinking group</td>
<td>F(1,67) = 2.86, p = .096, η² = .041</td>
<td>F(1,67) = 1.20, p = .277, η² = .018</td>
<td>F(1,67) &lt; 0.01, p = .957, η² &lt; .001</td>
</tr>
<tr>
<td>Site</td>
<td>F(2,134) = 1.93, p = .167, η² = .028</td>
<td>F(2,134) = 35.69, p &lt; .001, η² = .348*</td>
<td>F(3,201) = 40.31, p &lt; .001, η² = .376*</td>
</tr>
<tr>
<td>Condition</td>
<td>F(1,67) = 0.15, p = .699, η² = .002</td>
<td>F(1,67) = 22.95, p &lt; .001, η² = .255*</td>
<td>F(1,67) = 220.71, p &lt; .001, η² = .767*</td>
</tr>
<tr>
<td><strong>2-way interactions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site × Condition</td>
<td>F(2,134) = 6.53, p = .002, η² = .089*</td>
<td>F(2,134) = 1.19, p = .288, η² = .017</td>
<td>F(3,201) = 56.65, p &lt; .001, η² = .458*</td>
</tr>
<tr>
<td>Site × Drinking group</td>
<td>F(2,134) = 1.92, p = .150, η² = .028</td>
<td>F(2,134) = 0.16, p = .855, η² = .002</td>
<td>F(3,201) = 0.99, p = .397, η² = .015</td>
</tr>
<tr>
<td>Condition × Drinking group</td>
<td>F(1,67) = 0.34, p = .563, η² = .005</td>
<td>F(1,67) = 3.42, p = .069, η² = .049</td>
<td>F(1,67) = 0.14, p = .705, η² = .002</td>
</tr>
<tr>
<td><strong>3-way interaction</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site × Condition × Drinking group</td>
<td>F(2,134) = 4.12, p = .018, η² = .058*</td>
<td>F(2,134) = 0.18, p = .840, η² = .003</td>
<td>F(3,201) = 0.68, p = .567, η² = .010</td>
</tr>
<tr>
<td><strong>Latency</strong></td>
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<td></td>
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</tr>
<tr>
<td><strong>Main effects</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Drinking group</td>
<td>F(1,67) = 1.08, p = .302, η² = .016</td>
<td>F(1,67) = 0.03, p = .855, η² = .001</td>
<td>-</td>
</tr>
<tr>
<td>Site</td>
<td>F(1,134) = 8.49, p = .002, η² = .112*</td>
<td>F(1,134) = 16.91, p &lt; .001, η² = .201*</td>
<td>-</td>
</tr>
<tr>
<td>Condition</td>
<td>F(1,67) = 23.92, p &lt; .001, η² = .263*</td>
<td>F(1,67) = 4.19, p = .044, η² = .059*</td>
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<tr>
<td><strong>2-way interactions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site × Condition</td>
<td>F(2,134) = 3.05, p = .059, η² = .044</td>
<td>F(2,134) = 1.78, p = .181, η² = .026</td>
<td>-</td>
</tr>
<tr>
<td>Site × Drinking group</td>
<td>F(1,134) = 0.87, p = .422, η² = .013</td>
<td>F(1,134) = 0.29, p = .752, η² = .004</td>
<td>-</td>
</tr>
<tr>
<td>Condition × Drinking group</td>
<td>F(1,67) = 3.27, p = .075, η² = .047</td>
<td>F(1,67) = 0.41, p = .526, η² = .006</td>
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</tr>
<tr>
<td><strong>3-way interaction</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site × Condition × Drinking group</td>
<td>F(2,134) = 0.37, p = .695, η² = .005</td>
<td>F(2,134) = 0.52, p = .598, η² = .008</td>
<td>-</td>
</tr>
</tbody>
</table>
**P2 mean amplitude**

No significant effect of drinking group or two-way interactions with drinking were observed, indicating that there were no differences in overall P2 amplitude, distribution and condition effects between risk groups. However, significant interactions were observed for site × condition and site × condition × drinking group. The two-way interaction indicated that the P2 distribution differed between correct go and successful inhibitions, but this effect was only apparent in the low drinking group. Follow-up analyses in the low drinking group showed a frontocentral-to-central P2 on correct go while the P2 on successful inhibitions was broadly distributed. The prior was indicated by a significant quadratic trend showing larger amplitudes at FCz and Cz (linear, \( F(1,36) = 2.28, p = .138, \eta_p^2 = .060 \); quadratic, \( F(1,36) = 6.67, p = .014, \eta_p^2 = .156 \)), and the latter was indicated by non-significant linear and quadratic trends (linear, \( F(1,36) = 2.08, p = .158, \eta_p^2 = .055 \); quadratic, \( F(1,36) = 1.89, p = .178, \eta_p^2 = .050 \)). In contrast, follow-up analysis on the high drinking group showed a non-significant site × condition interaction which indicated that distribution of P2 did not differ between conditions in this group (\( F(2,62) = 0.29, p = .749, \eta_p^2 = .009 \))

**P2 peak latency**

Significant effects of condition and site were observed showing delayed P2 latency on successful inhibitions compared to correctly go trials and that latencies were shortest at Cz (Fz vs. FCz, \( t(68) = 2.15, p = .035, d = 0.26 \); FCz vs. Cz, \( t(68) = 2.89, p = .005, d = 0.35 \)). A marginally significant condition × drinking group interaction was observed. Although this effect was not statistically significant, differences in P2 latency were hypothesised and follow-up analyses were conducted to explore the direction of this trending effect. Follow-up analyses did not show a statistically significant effect but the pattern of results could indicated reduced latencies in the low drinking compared to the high drinking group on correct go trials (\( t(67) = -1.70, p = .093, d = 0.41 \)) but not inhibition trials (\( t(67) = 0.02, p = .986, d < 0.01 \)). No other effects were statistically significant.
**N2 mean amplitude**

A nogo N2 effect was evident, revealed by a significant condition effect showing enhanced amplitude on successful inhibitions compared to correct go trials. A significant site effect was also observed, indicating an overall frontocentral distribution (Fz vs. FCz, \( t(68) = -0.26, p = .798, d = .03 \); FCz vs. Cz, \( t(68) = -10.10, p < .001, d = 1.22 \)). A marginally significant condition \( \times \) drinking group interaction was observed and follow-up analyses were conducted to explore the direction of this trending effect as N2 modulations between groups was hypothesised. Follow-up comparisons showed significant N2 effects for low drinking (\( t(36) = 2.91, p < .006, d = 0.48 \)) and the high drinking group (\( t(31) = 3.67, p = .001, d = 0.65 \)) and the examination of effect sizes suggests that the N2 effect may be greater in high drinking group indicating greater conflict experienced.

**N2 peak latency**

Significant effects of condition and site were observed showing delayed N2 latency on successful inhibitions compared to correctly go trials and that latencies were shortest at Cz (Fz vs. FCz, \( t(68) = 4.67, p < .001, d = .56 \); Fz vs. Cz, \( t(68) = 4.68, p < .001, d = .56 \); FCz vs. Cz, \( t(68) = 2.81, p = .004, d = .34 \)). No interaction between site and condition were observed. In addition, no significant effects or interactions with drinking were observed, indicating that overall N2 latency and the pattern of condition and site effects did not differ between low and high drinking groups.

**P3 mean amplitude**

A nogo P3 effect was evident, revealed by a significant condition effect showing enhanced amplitudes on successful inhibitions compared to correct go trials. A significant site \( \times \) condition interaction was observed, showing a frontocentral-to-central P3 on successful inhibitions and a parietal P3 on correct go trials. The frontocentral-to-central distribution was indicated by significant linear, quadratic and cubic, but predominantly quadratic trend showing a FCz-Cz maximum (linear, \( F(1,68) = 12.68, p = .001, \eta^2_p = .157 \); quadratic, \( F(1,68) = 136.75, p < .001, \eta^2_p = .150 \); cubic, \( F(1,68) = 9.50, p = .004, \eta^2_p = .123 \)). The parietal distribution was indicated by significant linear, quadratic and cubic, but predominantly linear trend showing a curvilinear increase from Fz to Pz (linear, \( F(1,68) = \))
67.25, $p < .001$, $\eta^2_p = .497$; quadratic, $F(1,68) = 16.94$, $p < .001$, $\eta^2_p = .199$; cubic, $F(1,68) = 8.75$, $p = .004$, $\eta^2_p = .114$). No significant effect or interactions with risk were observed, indicating that overall P3 amplitude and distribution, and the nogo P3 effect did not differ between low and high drinking groups. No other effects were statistically significant. The multiple regression analysis predicting risky drinking based on indices of conflict monitoring and inhibition revealed a non-significant regression equation ($F(2,66) = 1.29$, $p = .283$) with $R^2$ of .04. Neither the N2 or P3 mean amplitude on successful inhibitions were significant predictors of risky drinking ($p < .05$)
Electrophysiological Results: BART

Figure 5.4: **LEFT:** Stimulus-locked, grand-averaged waveforms at Fz, FCz, Cz and Pz depicting successful pump feedback (solid green line), burst feedback (dashed red line), and the ‘burst minus successful pump’ difference (dotted black line) for low and high drinking groups. Voltage (μV) is represented on the vertical axis and time (ms) is represented on the horizontal axis. **RIGHT:** Scalp distributions of the feedback P2, FRN and parietal feedback P3 for low and high drinking groups (Latency windows are depicted in Table 5.1).
Table 5.5
Mean and standard deviation of mean amplitude (μV) and peak latency (ms) of the feedback
P2, FRN and parietal feedback P3.

<table>
<thead>
<tr>
<th>ERP</th>
<th>Condition</th>
<th>Site</th>
<th>Low drinking</th>
<th>High drinking</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>P2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Successful pump</td>
<td>Fz</td>
<td>2.47(1.74)</td>
<td>2.84(1.86)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FCz</td>
<td>2.82(1.74)</td>
<td>3.21(1.75)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>2.25(1.50)</td>
<td>2.55(1.51)</td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>Fz</td>
<td>3.27(2.54)</td>
<td>3.47(2.81)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FCz</td>
<td>3.54(2.70)</td>
<td>3.41(2.77)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>2.17(2.19)</td>
<td>2.12(2.43)</td>
</tr>
<tr>
<td></td>
<td>FRN</td>
<td>Fz</td>
<td>0.00(1.41)</td>
<td>0.08(1.55)</td>
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<tr>
<td></td>
<td></td>
<td>FCz</td>
<td>0.58(1.47)</td>
<td>0.40(1.51)</td>
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<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>1.34(1.38)</td>
<td>1.11(1.36)</td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>Fz</td>
<td>0.32(2.26)</td>
<td>0.09(2.32)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FCz</td>
<td>0.39(2.31)</td>
<td>-0.34(2.88)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>0.47(2.32)</td>
<td>-0.49(3.13)</td>
</tr>
<tr>
<td></td>
<td>Difference</td>
<td>Fz</td>
<td>0.19(2.23)</td>
<td>0.00(1.96)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FCz</td>
<td>-0.37(2.16)</td>
<td>-0.75(2.30)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>-0.96(2.15)</td>
<td>-1.61(2.49)</td>
</tr>
<tr>
<td></td>
<td>Ratio score</td>
<td>Fz</td>
<td>-0.11(4.13)</td>
<td>0.94(4.66)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FCz</td>
<td>-0.33(3.01)</td>
<td>0.16(3.44)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>-0.31(2.91)</td>
<td>-0.62(2.72)</td>
</tr>
<tr>
<td></td>
<td>Parietal P3</td>
<td>Fz</td>
<td>1.37 (1.97)</td>
<td>1.24 (3.48)</td>
</tr>
<tr>
<td></td>
<td>Burst</td>
<td>FCz</td>
<td>3.08 (2.48)</td>
<td>3.31 (3.09)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>3.98 (1.77)</td>
<td>3.94 (2.33)</td>
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<td>Pz</td>
<td>4.46 (2.11)</td>
<td>3.77 (2.65)</td>
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<tr>
<td></td>
<td>Difference</td>
<td>Fz</td>
<td>1.41 (2.32)</td>
<td>1.51 (3.57)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>FCz</td>
<td>3.07 (2.73)</td>
<td>3.33 (3.22)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cz</td>
<td>3.89(2.01)</td>
<td>3.95 (2.60)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pz</td>
<td>4.45(2.25)</td>
<td>3.50 (2.49)</td>
</tr>
</tbody>
</table>
### Table 5.6

**ANOVA effects of drinking group (low drinking, high drinking), site (Fz, FCz, Cz, Pz) and valence (Successful pump, Burst) for P2, FRN (separate, difference and ratio scores) and P3 (burst and difference) ERP components**

<table>
<thead>
<tr>
<th>ANOVA effects</th>
<th>P2</th>
<th>ERP</th>
<th>P3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main effects</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drinking group</td>
<td>$F(1,67) = 0.16, p = .692, \eta_p^2 = .002$</td>
<td>$F(1,67) = 0.94, p = .335, \eta_p^2 = .014$</td>
<td>$F(1,67) = 0.12, p = .729, \eta_p^2 = .002$</td>
</tr>
<tr>
<td>Site</td>
<td>$F(2,134) = 23.49, p &lt; .001, \eta_p^2 = .260^*$</td>
<td>$F(2,134) = 5.40, p = .014, \eta_p^2 = .075^*$</td>
<td>$F(3,201) = 31.82, p &lt; .001, \eta_p^2 = .332^*$</td>
</tr>
<tr>
<td>Valence</td>
<td>$F(1,67) = 2.72, p = .104, \eta_p^2 = .039$</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>2-way interactions</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site × Valence</td>
<td>$F(2, 134) = 20.62, p &lt; .001, \eta_p^2 = .235^*$</td>
<td>$F(2,134) = 13.02, p &lt; .001, \eta_p^2 = .163^*$</td>
<td>-</td>
</tr>
<tr>
<td>Site × Drinking group</td>
<td>$F(2,134) = 0.19, p = .827, \eta_p^2 = .003$</td>
<td>$F(2,134) = 1.82, p = .166, \eta_p^2 = .026$</td>
<td>$F(3,201) = 0.72, p = .544, \eta_p^2 = .011$</td>
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<tr>
<td>Valence × Drinking group</td>
<td>$F(1,67) = 0.86, p = .357, \eta_p^2 = .013$</td>
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</tr>
<tr>
<td><strong>3-way interaction</strong></td>
<td></td>
<td></td>
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<tr>
<td>Site×Valence×Drinking group</td>
<td>$F(2,134) = 0.64, p = .531, \eta_p^2 = .009$</td>
<td>$F(2,134) = 0.12, p = .890, \eta_p^2 = .002$</td>
<td>-</td>
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</table>

<table>
<thead>
<tr>
<th>ERP (difference wave)</th>
<th>FRN (ratio score)</th>
<th>P3 (difference wave)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Main effects</strong></td>
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<td></td>
</tr>
<tr>
<td>Drinking group</td>
<td>$F(1,67) = 0.73, p = .395, \eta_p^2 = .011$</td>
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<tr>
<td>Site</td>
<td>$F(2,134) = 22.29, p &lt; .001, \eta_p^2 = .250^*$</td>
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<tr>
<td><strong>2-way interactions</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Site × Drinking group</td>
<td>$F(2,134) = 0.61, p = .546, \eta_p^2 = .009$</td>
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</tr>
<tr>
<td>FRN (difference wave)</td>
<td>FRN (ratio score)</td>
<td>P3 (difference wave)</td>
</tr>
</tbody>
</table>
Feedback P2

No significant effect or interactions with risk were observed, indicating that the overall P2 amplitude and site effects did not differ between low and high drinking groups. A significant effect of site was observed, and follow-up analyses showed that the P2 was frontally distributed (Fz vs. FCz, \( t(68) = -2.25, p = .028, d = 0.27 \); Fz vs. Cz, \( t(68) = 3.94, p < .001, d = 0.47 \); FCz vs. Cz, \( t(68) = 7.09, p < .001, d = 0.85 \)). P2 amplitude did not differ with feedback valence, but a site \( \times \) condition interaction was observed showing a frontocentrially distributed P2 on successful pump trials and a broad frontal-to-frontocentral P2 on burst feedback trials. The frontocentral P3 was indicated by significant a quadratic trend showing a FCz maximum (linear, \( F(1,68) = 2.38, p = .128, \eta^2_p = .034 \); quadratic, \( F(1,68) = 89.87, p < .001, \eta^2_p = .569 \)). The broad frontocentral distribution was indicated by significant linear and quadratic trends showing a curvilinear reduction from Fz to Cz (linear, \( F(1,68) = 26.28, p < .001, \eta^2_p = .279 \); quadratic, \( F(1,68) = 29.03, p < .001, \eta^2_p = .299 \)).

Feedback-related negativity

No significant effect or interactions with risk were observed, indicating that the overall FRN amplitude and distribution, and the FRN effect did not differ between low and high drinking groups. Significant effects of site and valence were observed, showing an overall frontocentral FRN which was enhanced (more negative) on burst compared to successful pump feedback trials. A site \( \times \) valence interaction and follow-up analyses showed a frontocentral FRN on successful pump feedback trials and a broadly distributed FRN was observed on burst feedback trials. The frontal distribution on successful pumps was indicated by significant linear and quadratic trends showing a curvilinear reduction in the FRN (more positive) from Fz to Cz (linear, \( F(1,68) = 58.65, p < .001, \eta^2_p = .463 \); quadratic, \( F(1,68) = 8.91, p = .004, \eta^2_p = .116 \)). The broad distribution of FRN on burst trials was indicated by a non-significant effect of site (\( F(2,136) = 0.42, p = .594, \eta^2_p = .006 \)).

No effects or interactions with risk were observed when the FRN valence effect was measured on the ‘burst minus successful pump’ difference waveform and represented as a ratio of change. Significant effects of site were observed on the difference wave but not the ratio score, indicating that the FRN valence effect was centrally distributed (Fz vs. FCz, \( t(68) \))
\[ = 3.52, p = .001, d = 0.42; Fz vs. Cz, t(68) = 5.32, p < .001, d = 0.64; FCz vs. Cz, t(68) = 4.33, p < .001, d = 0.52). The regression analysis predicting risky drinking with indices of feedback processing revealed a non-significant regression equation \((F(1,67) = 1.36, p = .248)\) with an \(R^2\) of .02.

**Parietal Feedback P3**

No significant effects or interactions with risk were observed on P3 measured on the burst feedback waveform or on the ‘burst minus successful pump’ difference waveform, indicating that the amplitude and distribution of the P3 to burst trials, and the P3 valence effect did not differ between low and high drinking groups. Analysis of the ‘burst minus successful pump’ waveform indicated a significant P3 valence effect, as the amplitude was significantly greater than 0 \((t(68) = 12.47, p < .001, d = 1.50)\). The parietal feedback P3 to burst feedback and the P3 valence effect were both centro-parietally distributed (Burst: Fz vs. FCz, \(t(68) = -11.85, p < .001, d = 1.42\); FCz vs. Cz, \(t(68) = 3.64, p = .001, d = .44\); Cz vs. Pz, \(t(68) = -0.70, p = .485, d = 0.08\); Difference: Fz vs. FCz, \(t(68) = -11.25, p < .001, d = 1.35\); FCz vs. Cz, \(t(68) = -3.41, p = .001, d = 0.41\); Cz vs. Pz, \(t(68) = -0.36, p = .723, d = 0.04\).
Discussion

Using behavioural tasks, ERPs and questionnaires, the current study examined risky decision-making, response inhibition, feedback processing and personal sensitivity to external feedback in low and high drinking university students. The results showed a significantly lower number of adjusted average pumps and bursts in the high drinking group. However, there were no differences in ERP indices of feedback processing and response inhibition (FRN, P3), or self-reported personal sensitivity to external feedback (BIS/BAS scores). There were also no differences in ERP indices of early attentional processing in both tasks (P2). Differences in risky decision-making measured by BART provides support for its sensitivity of the task to differences in real-world behaviour, suggesting a greater preference for immediate reward in the high drinking group. However, similarities in ERP indices of response inhibition, feedback monitoring and early attentional processes, and similarities in BIS/BAS scores indicate that behavioural differences were not due to impairments in these underlying processes or deviations in personal sensitivity to external feedback. Collectively, these results suggest that impairments may be reflecting a domain-specific risk-taking driven by situational and environmental factors.

*Risky decision-making and immediate gratification in high drinkers*

Although differences in risky decision-making support the validity of the BART as a behavioural measure of risk-taking, previous studies typically report a greater number of pumps in high-risk compared to low-risk comparison groups. Studies have reported a greater number of adjusted average pumps in typical groups of individuals that reported engaging in various risky behaviours (e.g. alcohol use, smoking, illicit substance use, gambling, unprotected sex) compared to non-risk controls (Bishara et al., 2009; Lejuez et al., 2002, 2003). Studies have also reported differences between non-risk controls and various risky groups such as inner-city drug users and problem gamblers with or without a history of substance use disorder (Bornovalova et al., 2005; Coffey et al., 2011; Hopko et al., 2006; Ledgerwood et al., 2009).

Despite the discrepancies, a few studies have reported similar results (Bogg, Fukunaga, Finn, & Brown, 2012; Courtney et al., 2012; Dean, Sugar, Hellemann, & London,
Courtney et al. (2012) examined the role of response inhibition, risky decision-making, impulsive delayed discounting and risk attitudes in alcohol use and alcohol-related problems. They reported a negative relationship between the number of adjusted average pumps and alcohol-related problems, where a lower number of pumps was predictive of more alcohol-related problems. They also reported a significant positive relationship between delayed discounting and alcohol use/alcohol-related problems, as well as a negative relationship between adjusted average pumps and delayed discounting. Collectively, their findings provide evidence for the contribution of immediate reward preference to alcohol use and alcohol-related problems, where this preference manifested as a reduced number of pumps in the BART as participants voluntarily ended trials earlier. As such, the lower number of adjusted average pumps in the high drinking group in our study could indicate a preference for immediate reward.

This reverse association was also reported in two studies examining smoking and self-reported disinhibition (Bogg et al., 2012; Dean et al., 2011). In a study examining risk-taking in smokers and non-smokers, Dean et al. (2011) reported that non-smokers increased pumps over time while smokers decreased pumps over time. However, average measures of performance on the BART did not differ between the groups. In line with Courtney et al. (2012) delayed discounting was greater in smokers but they failed to show a statistically significant negative association with average pumps. This finding was interpreted as reflecting a failure to take risks in situations where risk-taking is adaptive. In a study examining risk-taking and alcohol use, Bogg et al. (2012) reported that disinhibition as measured by the Zuckerman sensation seeking subscale was positively related to weekly alcohol use and negatively related to the number of average pumps and bursts on the BART. However, no significant relationship between BART performance and weekly drinking was reported. In line with Courtney et al. (2012) these findings were interpreted as reflecting a preference for immediate rewards. While studies have reported greater risky decision-making on the BART associated with greater risk-taking, these studies highlight the possibility of an alternative pathway towards risk-taking, driven by an immediate preference for reward.

Alternatively, as both groups showed risk-averse behaviour, the greater number of adjusted average pumps in the low drinking group could be described as less risk-averse.
indicating the use of a more effective strategy. Regardless of whether the differences in risky decision-making between low and high drinking groups are influenced by an immediate reward preference or strategy, observed similarities in underlying feedback processing indices indicate that the observed differences were not due to abnormalities in feedback processing. Furthermore, similarities in personal sensitivity to external feedback was also observed, indicating that the pattern of results cannot be simply explained by differences in sensitivity to reward. It is possible that differences in risky decision-making may be driven by situational and environmental factors which have more specific impacts on attitudes towards risk-taking.

**Heterogeneity in risk-taking: Engagement in risky behaviour is not necessarily associated with underlying processing deficits**

Discrepancies in the association between risky decision-making tasks and real-world risk-taking could reflect heterogeneity in risk-taking at various levels which may not be captured in an averaged sample. As most cross-sectional studies classify individuals based on their risk-taking phenotype, it is difficult to examine differences between individuals with high risk propensity. Discrepancies may also be related to differences between ‘risky’ behaviours in terms of the factors that contribute to those behaviours, where there may be differences between normative behaviours (non-antisocial risk behaviours) such as extreme sports and high caffeine intake compared to maladaptive risk behaviours (antisocial risky behaviours) such as alcohol misuse, smoking and illicit substance use.

The idea of heterogeneity in risk-taking between individuals is discussed in a recently proposed ‘relative state model’ by Mishra, Barclay and Sparks (2017). The model postulates that there are two non-independent motivational pathways to risk-taking (need-based and ability-based pathways) which are driven by a perceived competitive disadvantage or advantage. The perceived competitive (dis)advantage is referred to as the ‘relative state’, which is influenced by embodied (e.g. genetic factors, physical and cognitive ability) and environment/situational factors (e.g. socioeconomic status), and the perception their state. The need-based motivational pathway is engaged where disadvantaged individuals take risks to at least offer a chance to meet needs and more successfully compete with others; while the ability-based pathway refers to when advantaged individuals are motivated engage in risks.
due to higher success rate and/or lower cost of risk-taking in domains afforded by their abilities. These pathways are theorised to lead to different but overlapping forms of risk-taking (antisocial, non-antisocial and prosocial) which would account for differences between individuals with high risk propensity.

This model also discusses differences between risk-behaviours and offers predictions about domain-general and domain-specific manifestations of risk-taking. It argues that risk-taking is domain-specific when competitive (dis)advantage is in a distinct domain, and risk-taking is domain-general when the (dis)advantage occurs in multiple domains. In relation to our results, similarities in underlying inhibition, feedback processing and personal sensitivity to external feedback could be reflect similarities in embodied factors. As such, between-group differences in risky decision-making could reflect domain-specific risk-taking driven by situational and environmental factors. Situational and environmental factors affecting alcohol use in university students include living arrangements (Harford, Wechsler, & Muthén, 2002) and social affiliations (Harford, Wechsler, & Seibring, 2002).

*Alternative explanations for non-significant findings: Statistical power and non-clinical samples.*

While statistically significant differences in risky decision-making between low and high drinkers indicated there was significant power to detect behavioural differences. Observation of marginally significant interactions with drinking group on P2 latency and N2 amplitude suggests there may be insufficient power to detect underlying effects. As these indices were hypothesised to differ between drinking groups, follow-up analyses were conducted. The results suggested delays in P2 latency and enhancements in the nogo N2 effect in the high drinking group, potentially reflecting delayed processing and greater conflict experienced. As the pattern of effects are consistent with expected patterns, it is possible that these effects are of smaller magnitude and may be observed with greater statistical power.

Relating to the neurophysiological measurement of factors contributing to risk-taking, deviations in underlying inhibition, feedback processing and attentional processes may only be observable in more severe types of risk-taking (e.g. illicit substance dependence) where
risk-taking is domain-general. For example, genetically at-risk populations and clinical groups may be more likely to show domain-general risk-taking (Fein & Chang, 2008; Kamarajan et al., 2010, 2015; Parvaz et al., 2015). Differences in underlying processes may also be observed in developing samples and non-clinical samples who show broad deficits, such as studies showing differences in reward processing between adolescents and adults (Yau et al., 2015) and showing associations associated with general personality traits such as impulsivity (Lange et al., 2012; Onoda et al., 2010).

Our sample of high drinkers did not appear to have significant abnormalities in the ability to suppress responses or process external feedback, suggesting the role of environmental factors may have facilitated greater alcohol use. While these external factors can result in problematic levels of alcohol use, it has been argued that drinking patterns during young adulthood are transient, with reduction in alcohol use reported to be related to age and environmental factors such as new jobs, church attendance and marital status (Campbell & Demb, 2008; Dawson et al., 2005; Jackson, Sher, Gotham, & Wood, 2001; Schulenberg, Maggs, Steinman, & Zucker, 2001; Vik, Cellucci, & Ivers, 2003). Epidemiological evidence indicates that that 34% of individuals who meet criteria for alcohol dependence measures go into remission with 10 years following onset of dependence without professional help, and 50% of individuals remitting 16 years following onset (Heyman, 2013; Lopez-Quintero et al., 2011). It may be that inhibition and feedback monitoring are not impaired in individuals with immediately high levels of consumption, but rather in individuals with persistent heavy consumption. There is evidence that individuals who show persistent drinking at diagnostic levels for more than 5 years during young adulthood showed significant liabilities in late-life health measures but individuals with less persistent drinking (less than 5 years) did not show significant elevation in risk (Haber, Harris-Olenak, Burroughs, & Jacob, 2016).

Conclusion and future research

The current study showed differences in risky decision-making between low and high drinkers, suggestive of a greater preference for immediate reward in the high drinking group. Discrepancies between studies showing positive and negative associations between risky decision-making and alcohol use are suggested to indicate different pathways towards risk-
taking. The absence of underlying ERP differences in response inhibition, feedback processing and self-reported sensitivity to external feedback were thought to indicate domain-specific risk-taking driven by situational and environmental factors rather than deviations in underlying processes. As the current study was focused on examining the contribution of underlying processes to risk-taking, we acknowledge that the lack of measuring situational/environmental factors and other risky behaviours limit the conclusions that can be drawn about the generality or specificity of risk-taking. Additionally, the study did not constrain drug and alcohol-use prior to the experiment which should be controlled in future studies. Future research examining alcohol use in normative samples should consider measuring more situational and environmental factors reported to contribute to alcohol use in young adults (e.g. living arrangements and social affiliation) and different risk-taking behaviours to provide context for identifying domain-specific risk-taking. There may also be value in quantifying persistence of heavy drinking rather than current quantity and frequency of alcohol consumption.
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Chapter 6: General Discussion

Chapter Summary

The overall aim of this thesis was to explore role of neurodevelopment in risk-taking using the event-related potential (ERP) technique. The thesis focused on inhibition and reward processing components of the dual-systems model, a prominent neurodevelopmental model of risk-taking (Shulman et al., 2016). It aimed to study the development of both systems during the adolescent-adult transition, providing an integrated view of the model. It also aimed to investigate whether this dual-systems model could be used to explain individual differences in risk-taking behaviour. The findings provide partial support for the dual-systems model, showing developmental improvements in task performance during the response inhibition task during the adolescent-adult transition, as well as a hypersensitivity to feedback during adolescence. The thesis also makes several contributions to the literature. Firstly, the use of ERPs extends on our understanding of the development of inhibition during the adolescent-adult transition, allowing us to delineate between the contribution of early attentional and later cognitive processes. Secondly, the use of ERPs also provided insight regarding the development of feedback processing during this period, demonstrating adolescents were hyper-response responsive to overall feedback, not just positive feedback. The thesis also provides several insights regarding the functional significance of inhibition and feedback processing ERPs. This section will provide an overview of the studies conducted in the thesis which will be followed by further discussion of their contribution.

This thesis presented four investigations examining the behavioural and electrophysiological indices of response inhibition, feedback processing and risky decision-making in adolescents and young adults. The first two studies focused on examining response inhibition and its development. One challenge associated with examining the development of response inhibition during the adolescent-adult transition is detecting differences in performance. Differences between adolescents and adults are subtle and standard tasks may not be sensitive to these changes. To improve the sensitivity of the task to false alarm errors, used a modified go/nogo task that was designed to detect partial inhibitions. Study 1 provided an initial exploration of this task in young adults. It showed that partial inhibitions
accounted for a substantial amount of errors on nogo trials, highlighting the utility of the task. The ERP results also revealed that the processes underlying partial inhibitions differed from successfully inhibited nogo trials. The findings highlighted the importance of partial inhibitions and the need to distinguish partial from successful inhibitions. Modulations of the ERPs to partial inhibitions also provide support for functional significance of the nogo P3 as an index of response inhibition.

In Study 2, this modified task was used to examine the development of response inhibition during the adolescent-adult transition. The results further demonstrated the utility of this task by detecting statistically significant improvements in response inhibition during the adolescent-adult transition. Whilst significant differences in accuracy between adolescents and adults suggest improvements in the ability to effectively suppress responses, the ERP results indicated that the inhibitory processes were relatively mature by mid-to-late adolescents and the effect was better explained by combined differences in early attentional processing and impulsive responding.

Study 3 focused on examining the reward processing component of the dual-systems model. This was investigated by examining feedback responses elicited during a dynamic risky decision-making task called the Balloon Analogue Risk Task (BART). Study 3 provided evidence for the ongoing maturation of feedback processing during the adolescent-adult transition. Enhanced P2 and FRN amplitudes were observed in adolescents suggesting they placed a greater emphasis in processing external feedback regardless of its valence. However, the results also highlighted the maturity of processes involved in discriminating and evaluating feedback, demonstrated by group similarities in the FRN and parietal feedback P3 valence effects. There were no significant adolescent-adult differences in risky decision-making, as measured using adjusted average pumps, highlighting the maturity of decision-making in adolescence. We suggest that the observed adolescent-adult differences in feedback processing may be due to differences in task approach, with adolescents allocating more attentional resources to process external feedback as opposed to differences in feedback processing abilities.
Study 3 also provided two insights regarding the examination of ERPs using dynamic risky decision-making. Firstly, the observation of a small FRN valence effect highlighted the potential impact of variability in risk and reward from trial-to-trial. Previous ERP studies using the BART have also reported small FRN valence effects but researchers have yet to acknowledge the impact of this variation. Secondly, the results also revealed a frontocentral feedback P3 that was distinct from the parietal P3. This observation is noteworthy because frontocentral feedback P3s are infrequently observed. Existing but limited research shows the frontocentral feedback P3 is sensitive to outcome likelihood and it only appears to be elicited in tasks with informative feedback. Enhancements in frontocentral feedback P3 may reflect a response to violations of expectancy or internal models of the task however further research investigating its functional significance is required.

Applying the method for examining inhibition and feedback processing during the adolescent-adult transition, Study 4 explored whether these processes and/or attentional processes, reflected in the P2, differed between low and high drinking young adults. There were significant differences in risky decision-making between drinking groups where a smaller number of adjusted average pumps and bursts were observed in the high drinking group; a finding consistent with the idea that high drinkers had a greater preference for immediate reward (Courtney et al., 2012). No differences in underlying inhibition, feedback monitoring or attentional processes were observed indicating that there were no substantial abnormalities in these processes. Furthermore, self-reported personal sensitivity to external feedback examined using the BIS/BAS also did not differ. The contrasting differences in risky decision-making and the absence of underlying differences in inhibition, feedback and attentional processing, and personal sensitivity to external feedback were interpreted as suggesting an alternate pathway towards risk-taking, driven by situational and environmental factors.

In relation to the development of psychological processes implicated in risk-taking behaviour, this thesis presents three key findings. Firstly, it demonstrates ongoing development of early attentional processes whilst highlighting the relative maturity of inhibition and feedback-related processes during the adolescent-adult transition. Secondly, it demonstrates that the early attentional processes may facilitate or impede subsequent
inhibitory control processes but did not have clear impact on feedback discrimination or evaluation. Lastly, it demonstrated that differences in risky decision-making and heavy drinking in typically functioning adults are not necessarily underlying impairments in inhibition and feedback processing, highlighting the role of transient situational and environmental factors. The thesis also raises various questions about measurement of underlying inhibition and feedback processes, use of dynamic risk-taking tasks, and the conceptualisation of the role of psychophysiology in risk-taking.

The following sections will discuss (i) the importance of studying early attentional processes which support subsequent processes, (ii) the use of dynamic risk-taking tasks and factors to consider from both behavioural and electrophysiological perspectives, (iii) the functional significance of inhibition and feedback processing ERPs and (iii) limitations of the current approach taken to study risk-taking.

**Role of Attentional Processes**

The anterior P2 has been implicated in attention and is thought to reflect attentional selection and salience detection processes. Support for this idea was derived from oddball tasks reporting enhancements P2 to novel-irrelevant and task-relevant stimuli, relative to irrelevant standard stimuli (Luck & Hillyard, 1994; Potts, Liotti, Tucker, & Posner, 1996). Enhanced P2 to stimulus features designated as task-relevant were thought to reflect a top-down attention selection process facilitating processing of relevant features, and enhancements to novel but irrelevant stimuli were thought to reflect a bottom-up salience detection process.

Observed differences in the anterior P2 in Studies 2 and 3 may reflect developmental differences in efficiency of attention allocation processes and/or a different approach to the task. Delays in P2 latency in adolescents during inhibition could reflect slower orientation and processing of the stimulus compared to adults; and enhancements in P2 amplitude in adolescents during feedback processing could reflect greater allocation of attention due to a greater reliance on external feedback. In the absence of differences in inhibition and feedback-related processes reflected by the nogo N2, P3 and the FRN, parietal feedback P3,
these findings warrant the consideration of attentional processes and its impact on subsequent processes.

Although previous studies have consistently reported that P2 varies with attention between conditions, reports of between-group effects are limited. Changes in anterior P2 during response inhibition from childhood to adulthood, and differences in P2 between various risk-taking groups have not been reported. However, several studies have examined anterior P2 elicited to external feedback. For example, studies have examined P2 in groups differing in trait psychoticism, impulsivity, problematic internet gaming and alcohol use (Duven, Müller, Beutel, & Wölfling, 2015; Franken, Van Den Berg, & Van Strien, 2010; Potts, George, Martin, & Barratt, 2006; Salim, van der Veen, van Dongen, & Franken, 2015). However, the results are varied and are largely derived from passive tasks, making it difficult to draw conclusions about its impact on behaviour. More recently, a few studies have examined anterior P2 in active risk-taking tasks (Kardos et al., 2016; Kiat, Straley, & Cheadle, 2016; Polezzi, Lotto, Daum, Sartori, & Rumiati, 2008; Schuermann, Endrass, & Kathmann, 2012). Comparisons in the response to uncertain and certain choices show that enhanced P2 is observed to uncertain choices irrespective of outcome, consistent with the idea that there is a general top-down facilitation of uncertain outcomes (Polezzi et al., 2008). On the other hand, there is also some suggestion in the literature that the anterior P2 as an index of the reward prediction error signal, similar to the FRN, however this idea needs to be further explored (Kardos et al., 2016; Potts et al., 2006).

Current understanding of the anterior P2 and its role in behaviour is still quite limited and future research should focus on examining the anterior P2 at the between-subjects level. In context of its proposed role in attention, studies should examine whether there is a relationship between P2 modulation and individual differences in response inhibition, feedback processing and risky decision-making. Future research could also examine these modulations co-vary with other indices of attitudes and motivation towards relevant stimuli, whether P2 differences across tasks are related. Future studies should also explore its proposed role as an index of reward prediction, as opposed to an index of attention.
Dynamic Risk-Taking Tasks and the BART

Dynamic risk-taking tasks are argued to be more affectively engaging and are a better simulator of real-world risk-taking compared to static risk-taking tasks (Figner, Mackinlay, Wilkening, & Weber, 2009; Lejuez et al., 2003). To our knowledge, this thesis presented the first investigations directly comparing feedback ERPs in the BART between mid-to-late adolescents and adults, and between low and high drinking groups. Through using the BART, I comment on four key points; (i) the importance of providing informative feedback in dynamic tasks, (ii) the sensitivity of the BART to preference for immediate reward and (iv) the impact of trial variability in risk on the measurement of the FRN.

Task Differences: Importance of informative feedback

The absence of an adolescent-adult difference in risky decision-making in Chapter 5 is inconsistent with previous findings; especially with Figner et al. (2009) who reported heightened adolescent risk-taking using a dynamic risk-taking task (Cambridge Card Task). Although both the BART and the Cambridge Card Task can be described as dynamic due to their sequential properties, Study 3 discussed that differences in the accumulation of risk and the frequency of negative feedback between the two tasks may contribute to the discrepancy in behavioural findings.

By design, negative outcomes in the Cambridge Card Task were not revealed until the end of the sequence. This was done enable detection voluntary stopping during the task and 9 loss trials were randomly interspersed among the 54 trials to give participants the impression they were performing a chance-based task. This design choice meant that risks do not accumulate across trials and the occurrence of negative outcomes is very low and is not dependent on behaviour. It has been argued that this lack of negative feedback results a contaminated measure of risk-taking that is sensitive to the decision-maker’s belief in trend continuation (i.e. their tendency to adopt a win-stay and lose-switch strategy), rather than risk preference (Markiewicz, Kubińska, & Tyszka, 2015). As such, it was suggested that developmental differences between adolescents and adults might be due to developmental differences in belief in trend continuation rather than risk-taking propensity.
Although the current findings did not show any adolescent-adult differences in risky decision-making, observed enhancement in responsiveness to overall external feedback in adolescents (reflected by overall enhancement in P2/FRN amplitude) does provide support for the developmental differences in trend continuation idea. Reduced negative feedback on the Cambridge Card Task results in a disproportionate increase in positive feedback which could lead to stronger reinforcement of risky decisions (i.e. card turns). As such, it can also be argued that the absence of developmental differences using the BART is due to the presence of relatively frequent and consistent, negative feedback regulating responses.

Collectively, this discussion highlights that different implementations of voluntary stopping may have a big impact on the pattern of results as they have an impact on learning from feedback. While the ability to discriminate feedback appears to be mature and there are no apparent biases towards positive or negative feedback, a heightened responsiveness to overall feedback may manifest behaviourally in certain learning conditions where there are disproportionately large amounts of positive (and possibly negative) feedback.

*Sensitivity to preference for immediate reward*

In the BART, a greater number of pumps are typically interpreted as reflecting greater risk-taking as numerous studies have reported greater adjusted average pumps in various risky groups (Bishara et al., 2009; Bornovalova, Daughters, Hernandez, Richards, & Lejuez, 2005; Coffey, Schumacher, Baschnagel, Hawk, & Holloman, 2011; Hopko et al., 2006; Ledgerwood, Alessi, Phoenix, & Petry, 2009; Lejuez et al., 2002, 2003). Contrary to these findings, Chapter 5 reported a smaller number of adjusted average pumps in the high drinking group, suggesting that heavy drinking is associated with reduced risk-taking. While this finding appears to be counterintuitive, an alternative and more compatible interpretation is that the smaller number of adjusted average pumps in the high drinking group may a preference for immediate rewards.

The immediate reward preference interpretation was previously suggested by Courtney et al. (2012) who examined the role of response inhibition, risky decision-making, impulsive delayed discounting and risk attitudes in alcohol use and alcohol-related problems. They reported a negative relationship between the number of adjusted average pumps and
alcohol-related problems, where a lower number of pumps was predictive of more alcohol-related problems. They also reported a significant positive relationship between delayed discounting and alcohol use/alcohol-related problems, as well as a negative relationship between adjusted average pumps and delayed discounting.

Similar results have also been reported in two studies examining smoking and self-reported disinhibition (Bogg, Fukunaga, Finn, & Brown, 2012; Dean, Sugar, Hellemann, & London, 2011). In a study examining risk-taking in smokers and non-smokers, it was reported that the number of pumps in non-smokers increased over time while smokers decreased pumps over time (Dean et al., 2011). In line with Courtney et al. (2012), delayed discounting was greater in smokers but they failed to show a statistically significant negative association with average pumps. This finding was interpreted as reflecting a failure to take risks in situations where risk-taking is adaptive.

In a study examining risk-taking and alcohol use, Bogg et al. (2012) reported that trait disinhibition as measured by the Zuckerman sensation seeking subscale was positively related to weekly alcohol use and negatively related to the number of average pumps and bursts on the BART. However, no significant relationship between BART performance and weekly drinking was reported. In line with Courtney et al. (2012) these findings were interpreted as reflecting a preference for immediate rewards. Collectively, these studies highlight the possibility of an alternative pathway towards risk-taking, driven by an immediate preference for reward.

Variability in risk across trials and its impact on the FRN

The FRN valence effect elicited in the BART relatively small compared to reported effects using static tasks (e.g. unpredictable gambling or probabilistic learning tasks). The FRN valence effects elicited in Studies 2 and 3 ranged from 0.96 – 1.61μV. Similarly, Yau, Potenza, Mayes and Crowley (2015) reported FRN valence effects ranging from 0.22 – 1.81μV. On the other hand, valence effects reported in adolescent-adult comparisons using unpredictable gambling tasks and probabilistic learning tasks range from range from 1.1 – 4.7μV (Grose-Fifer, Migliaccio, & Zottoli, 2014; Hämmerer, Li, Müller, & Lindenberger, 2011; Zottoli & Grose-Fifer, 2012).
These reduced effects could be attributed to changes in risk and reward from trial-to-trial due to the dynamic nature of the task. In the BART, the magnitude and probability of positive and negative outcomes changes between each decision. As feedback-related ERPs are reported sensitive these features, increased variability in the feedback response may make it difficult to observe developmental or group differences in the grand-averaged FRN. Future research should examine the impact of dynamic changes on feedback ERPs more closely, comparing them to feedback responses elicited during static tasks. Other methods such as single-trial analysis or and sub-averaged waveforms could be useful for investigating within and between-trial changes in the processing of outcomes which could provide further insight into the FRN may be related to risky decision-making.

**Measurement and functional significance of Inhibition and Feedback-related ERPs**

The examination of ERPs during partial inhibitions in a modified go/nogo task and feedback presentation in during the BART task provided insight regarding the measurement and significance of these processes. With respect to inhibition, it was demonstrated that measuring partial inhibition was an effective way to increase the detection of errors, improving accuracy measures (Amieva et al., 2002; Chevalier, Kelsey, Wiebe, & Espy, 2014; Cragg, Fox, Nation, Reid, & Anderson, 2009). Two key ERP findings were found. Firstly, observed N2 differences between partial and successful inhibitions indicated that partial inhibitions were processed as errors, highlighting the need to identify and separate these trials. Secondly, the observed absence of an enhanced nogo P3 during partial inhibitions highlights the role of nogo P3 in response inhibition, differentiating it from the novelty frontocentral P3. This modulation could indicate a lack of inhibition or an evaluated failure to completely suppress the response. However, there are several unknowns about the processes that underlie partial inhibitions which require further investigation.

Firstly, differences in motor-related activity between go and nogo trials has been previously identified as a potential confound, although further investigation has concluded that it does not completely account for the nogo P3 effect (Smith, 2011; Smith, Jamadar, Provost, & Michie, 2013; Smith, Johnstone, & Barry, 2008). Nevertheless, P3 modulations
during partial inhibitions are also consistent with this account and future research should revisit this idea. This can be investigated by examining modulations in ERPs to partial and successful responses in a context where a motor response is required in both conditions. Secondly, due to a lack of trials, we were unable to examine unsuccessful inhibition of nogo trials. Although previous studies have identified both unsuccessful and partial inhibitions (Cragg, Fox, Nation, Reid, & Anderson, 2009; De Jong, Coles, Logan, & Gratton, 1990), research has yet to examine whether processes underlying unsuccessful and partial inhibition are distinct. Underlying differences between these conditions would provide insight regarding the suppression of an ongoing motor response. However, a key consideration for undertaking this research is to develop a paradigm that is able to elicit a sufficient amount of unsuccessful and partial inhibitions reliably. Some possible solutions may include using a more difficult inhibition task (e.g. stop-signal) and/or more sensitive response measures (e.g. EMG sensors and force transducers).

With respect to feedback processing during the BART, two distinct positive peaks following the FRN were identified. The observation of an earlier frontocentral positivity and a later parietal positivity is similar the subcomponent structure found in response to task-relevant stimuli and to erroneous responses. However, the frontocentral feedback P3 it is not commonly reported in response to feedback. Recent studies have reports have demonstrated there is some consistency in the frontocentral feedback P3, warranting attention to this component in future studies (Mathewson, Dywan, Snyder, Tays, & Segalowitz, 2008; Sambrook & Goslin, 2015; Walentowska, Moors, Paul, & Pourtois, 2016). Sambrook and Goslin (2015) identified that this component is sensitive to outcome likelihood and is more prominent in tasks where the participant has more control. In line with this finding, a recent study by Walentowska et al. (2016) reported that frontocentral P3 was sensitive to outcome probability and whether outcomes were informative. Consistent findings were also reported in a non-probabilistic learning task where Mathewson et al. (2008) reported an enhanced frontocentral P3 to negative feedback greater on test compared to learning phase of the task. The sensitivity of the frontocentral P3 to outcome probability, task control and feedback relevance could suggest a role in the signalling a violation of expectancy or internal models of the task. However, further research is required to investigate this idea and examine whether it is functionally distinct from the FRN and parietal feedback P3. Future research
should examine whether the context in which the frontocentral feedback P3 is elicited and whether this response is functionally distinct from the FRN and the parietal P3.

**Limitations on the current approach to studying risk-taking**

While this thesis provides insight into the development of inhibitory and feedback processing and the functional significance of the associated ERPs, there are some notable limitations relating to separate measurement of inhibitory and feedback processes, the breadth of measures to provide context for behavioural differences, and the use of a non-clinical sample. The discussion of these limits is followed by suggestions for future research.

Firstly, based on the proposed idea that heightened adolescent risk-taking is partly driven by discrepancies in the development of inhibitory and feedback processing systems, this thesis investigated the combined role of these systems by separately examining their state during the adolescent-adult transition. The expectation was to demonstrate differences in response inhibition and not feedback monitoring highlighting ongoing and early development of these systems, or differences in both systems in a pattern suggesting the coupling of an immature cognitive control system with a hyperactive reward processing system.

The implicit assumption of this approach is that there are deviations in the state of one or both systems which contribute additively to risk-taking propensity. While important developmental differences in P2 were observed, ERPs implicated in inhibition and feedback processing were largely similar across the adolescent-adult transition and between low and high drinking groups. It is possible that developmental contribution of the two systems lies in the interactive engagement of the two systems, rather than discrepancies in individual systems. As such, the current approach may not be optimal for capturing the interaction between the two systems.

An alternative approach that closely reflects the interaction proposed by the model is to investigate how the capacity to inhibit responses is modulated by differences in reward and other situational factors. For example, a hybrid task could be used to examine whether potential outcomes influence the ability to suppress responses and whether there are
developmental differences in this effect. Some research has found that inhibition-related ERPs are modulated by emotional context (Ramos-Loyo, Llamas-Alonso, González-Garrido, & Hernández-Villalobos, 2017). It has also been demonstrated that risk-taking behaviour is influenced by peer presence, highlighting the importance of context in which decisions are made (Silva, Patrianakos, Chein, & Steinberg, 2017). Future research could examine whether these factors modulate response inhibition and risky decision-making.

Secondly, the results in Study 4 suggested that differences in risky decision-making between low and high drinkers were not due to underlying differences in inhibitory, feedback and attentional processing, or self-reported personal sensitivity to external feedback. As such, it was suggested that these differences reflect domain-specific risk-taking may be accounted for by transient situational and environmental factors. However, other measures of risk-taking and measures of environmental factors were not obtained. Additionally, it was also suggested that differences in risky decision-making reflected differences in preference for immediate reward. However, without appropriate measures that tap into the temporal aspect of reward preference, we were unable to provide additional support for this idea. These points can be summarised as a limit in the breadth of measures obtained.

Additionally, due to the absence of matching between adolescents and adolescent groups, it is possible that age-group differences studies 2 and 3 reflect a demographic sample difference rather than underlying differences in the development of stimulus- and feedback-related processing. Given that key processes examined were related to performance monitoring and cognitive control, it is possible that individual differences in cognitive ability (e.g. executive functioning) may have explained the developmental effect.

The thesis makes a few recommendations for future research investigating risk-taking and alcohol use in typically functioning young adults. Given the acknowledgement of domain specificity in risk-taking, situational factors such as peer presence and current evidence suggesting that heightened risk-taking was driven by external factors, researchers should focus on obtaining appropriate measures to enable commentary on this pathway towards risk-taking. For example, one could take advantage of modern smartphone technology to collect detailed information about the pattern and context in which risky
drinking occurs (Poulton, Pan, & Hester, 2016). One could also implement measures of attitudes towards risk-taking in various contexts (Domain specific risk-taking scale, Blais & Weber, 2006) and recording reported engagement in other risky behaviours would provide insight about the specificity of risk-taking. In addition to demographic variables often measured such as cognitive ability, education and socioeconomic status, future research should also obtain measures of situational and external factors that have a reported relationship with the behaviour of interest. For example, research investigating alcohol use in young adults should obtain information about living arrangements and social affiliation which have been shown to be related to alcohol use in this sample (Harford, Wechsler, & Muthén, 2002; Harford, Wechsler, & Seibring, 2002).

Relating to the use of dynamic risk-taking tasks such as the BART, based on findings from the delayed discounting task, it was suggested the reduction in number of adjusted average pumps in high risk groups could index a preference for immediate reward. These findings give precedent to the notion the BART may not only sensitive to risk-taking propensity but also to sensitivity to delays in reward. As such, future research examining using the BART, dynamic risk-taking tasks or other which the ability to delay rewards should consider whether there are differences in this dimension by obtaining measures of self-report such as the delayed gratification inventory (Hoerger, Quirk, & Weed, 2011) or behavioural measures such as the delayed discounting task (Reynolds & Schiffbauer, 2004).

**Conclusion**

To summarise, the thesis provides various insights regarding neurodevelopment, functional significance of inhibition and feedback-related ERPs, and risk-taking. It provides evidence for the ongoing development of early attention during the adolescent-adult transition whilst highlighting the relative maturity of inhibitory and feedback processes. Based on our findings, it appears that adolescent-adult differences are due to differences in the approach to the task (impulsive responding and emphasis on external feedback) and not deficiencies in processes involved in response suppression and feedback processing.

Relation to the functional significance of ERPs, the developmental studies highlighted the importance accounting for attentional processes reflected by the P2. The use
of the modified go/nogo task demonstrated the utility of measuring partial inhibitions both as a way to improve task sensitivity and to investigate the significance of inhibition-related ERPs. The use of the BART, outlined the potential and limitations associated examining ERPs using dynamic risk-taking tasks.

The examination of individuals differences in risky decision-making and alcohol use provides insight into the conceptualisation of risk-taking and how underlying cognitive processes may contribute. It demonstrated that variation in risk-taking in typically functioning young adults are not necessarily associated with deviations in inhibitory control or feedback processing. While previous evidence reports abnormalities in substance dependent and other risky groups, it is possible that these individuals may not be on the same continuum as typically functioning young adults. These findings suggest that differences in risk-taking may be domain specific and future research is encouraged to obtain measure risk-taking behaviours and attitudes broadly and consider the role of situational and environmental factors.
References


