# An information foraging perspective on attention differences in autism and ADHD, taking a neurodiversity approach.

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## Declaration

I declare that the work contained in this thesis has not been submitted for any other award and that it is all my own work. I also confirm that this work fully acknowledges opinions, ideas and contributions from the work of others.

Any ethical clearance for the research presented in this thesis has been approved. Approval has been sought and granted by the School of Psychology Ethics Committee at the University of East Anglia.

## Abstract

In this PhD we take the position that the attentional system functions as an information foraging mechanism, seeking information from one source until information is depleted and then moving to another source. Research shows that this system is sensitive to individual variation. Based on the above we proposed to test that Autism Spectrum Disorders (ASD) and Attention Deficit Hyper-Activity Disorder (ADHD) map onto information foraging differences. Specifically, in accordance with neural gain models, ASD and ADHD might present as opposing profiles in their information seeking behaviours. We test this theoretical proposal using two paradigms, one employing Electroencephalogram (EEG) and the other Eye tracking, both in healthy adults, as well as in a sample of adolescents with and without an Autism diagnosis. Across all the samples, we also adopt a multi-dimensional traits approach.

The EEG experiment uses changes in neural-signals over time in response to dynamic repeating and novel stimuli and taps into attentional engagement. Through repeated stimulus exposure, this paradigm stimulates information depletion from a video source. From the adolescent sample, we report that our results find support for the neural gain model in relation to ADHD traits but not ASD traits. The eye-tracking experiment is an adaptation of the gap-overlap paradigm to behaviourally measure attention disengagement. Again, information depletion is achieved by repetitions of the central stimulus. In our adolescent sample we demonstrate a linear decline in disengagement cost as central stimuli are repeated, this finding however was not modulated by individual differences.

We demonstrate that while information gain may modulate attention and engagement, it's interaction with neurodiversity is more complex. Findings from the adolescent samples, particularly as it relates to ADHD were consistent with neural gain models, however conceptualisation of ASD as the attentional opposite to ADHD was not supported.

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# Acknowledgements

بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

In the name of Allah, the most merciful the most compassionate, who has made everything easy. Peace and blessings upon the Messenger, through whom all good has reached us.

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# CHAPTER 1:

## 1. Introduction

## Goal of chapter

*This first chapter provides an overview of both classical and contemporary models to aid our current understanding of the attentional system, and to outline its role as an information-foraging mechanism. We do this by first outlining the classical understanding of the attentional network, describing the three separate networks as first delineated by Posner and Petersen and the research that supports the classical understanding of attentional models. Then we outline the attentional network's evolution into a decision-making and information-seeking framework. This chapter also describes the role of the dopaminergic system in attention as a whole, but also more specifically as it relates to information foraging, explore/exploit trade-offs and individual differences. Autism Spectrum Disorders (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) are then positioned within this framework.*

*We examine literature that point towards ASD and ADHD representing opposing ends of a continuum with regards to their attentional system and find support for this theory from the dopaminergic account and neural gain model. We propose two experimental paradigms to test these theoretical accounts, using EEG and Eye-tracking in adolescent and adult populations and give general predictions based on the reviewed empirical and theoretical literature.*

# 1.1 Background literature review

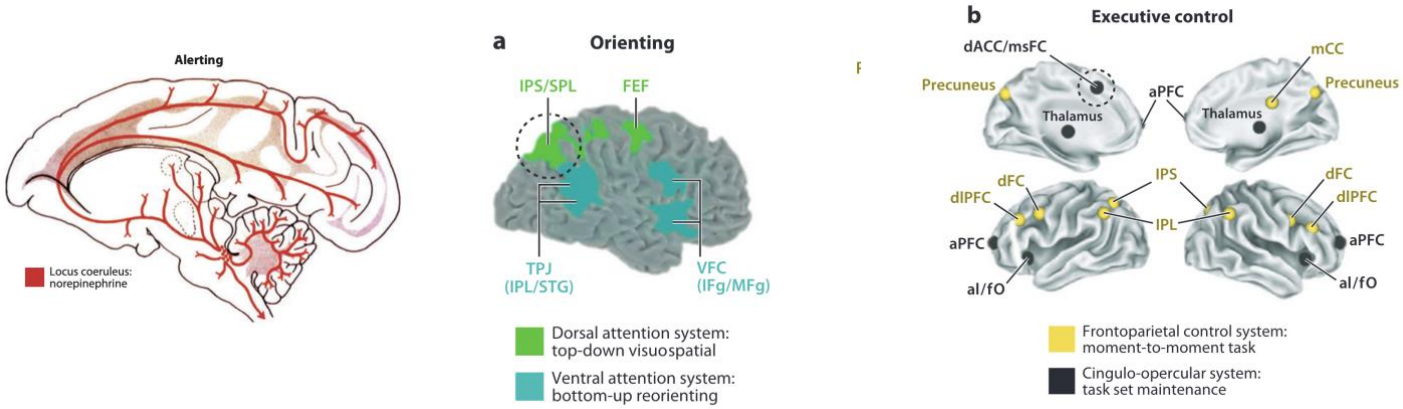
## 1.1.1 Attention: classical models and components

Attention is a fundamental component of our cognition and plays a role in most if not all other cognitive domains such as memory (Chun & Turk-Browne, 2007) and perception (Boynton, 2005). It is a heavily researched topic, and is present in many forms, in almost every behaviour. William James defined it as the "focusing of the mind" in the 1890's and remarked that "everyone knows what attention is". Over-time our understanding of attention has evolved, in order to better align with findings from experimental research and theoretical accounts of cognition. Attention is now more likely to be defined as "the set of evolved brain processes that leads to adaptive and effective behavioural selection" (Krauzlis et al., 2021).

When we process complex environments, a filtering system is needed whereby certain stimuli are prioritised over others. An effective attentional system will result in an individual being able to select relevant stimuli from irrelevant stimuli in their environment, sustain focus on those task-relevant stimuli, and shift efficiently between different stimuli where needed. Over the decades, models of attention have been put forth, explaining attention as a combination of interrelated, but distinguishable processes. Posner and Petersen (1990) famously explained these emergent behaviours as the workings of three major attentional system networks, each representing different processes and distinct neural networks in the brain. According to their proposal, the *orienting network* prioritises incoming sensory information such that relevant stimuli are attended to, and non-relevant stimuli are disengaged from. This network also modulates shifting between different stimuli. The *alerting network* plays the role of maintaining vigilance and performance during tasks, which changes throughout the day (tonic alertness) and through specific task demands (phasic alertness). Finally, the *executive network*, which detects and monitors conflict between competing mental processes to effectively detect errors, make decisions, and other functions that relate to higher-level processing and executive control. These attentional processes are sensitive to motor input and interact with various sensorimotor processing systems in order to achieve multi-sensory integration of incoming information (Rizzolatti et al., 1987; Pineda, 2008; Matusz et al., 2015).

Posner and Petersen's account of the attentional system has been widely accepted due to it being both a mechanistic and a biological model of attention. Three functionally and anatomically distinct networks have specific roles associated with them. For example,

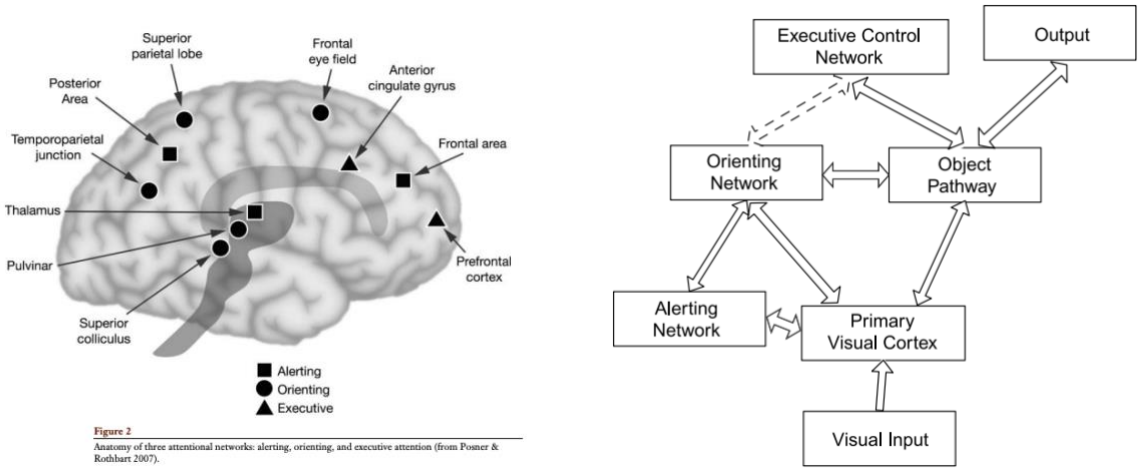
alerting is modulated by the locus coerleus and right parietal cortex and thalamus (Petersen & Posner 1990; Aston-jones & Cohen, 2005), orienting is modulated by the posterior parietal cortex (Petersen & Posner 1990), and frontal eye fields (Petersen & Posner, 2012; Corbetta et al., 1998). The executive network is associated with neural networks in the medial frontal cortex and anterior cingulate (Petersen & Posner, 2012). The biological mechanisms of the three attentional systems are presented in *Figure 1.1,* and in *Figure 1.2*.




*(a)* *(b)* *(c)*

*Figure 1.1. (a)* The Alerting network adapted from Aston-Jones & Cohen (2005) showing the locus coeruleus projections on macaque brain. The diffuse connections interact with other more strongly localized systems. *(b)* The orienting network adapted from Power et al., (2011) showing the dorsal and ventral networks. The dorsal attention network consists of frontal eye fields, and the superior parietal lobe. The ventral attention network consists of regions in the temporoparietal junction and the ventral frontal cortex. *(c)* The executive control system adapted from Dosenbach et al., (2007) show the two networks of the executive control system.

In addition to Posner and Petersen's mechanistic account, other classical models have been proposed, such as by Sohlberg and Mateer (1987, 1989). Here, the attentional system is divided into: focused attention, sustained attention, selective attention, alternating attention, and dividing attention. This model presents a hierarchical structure where subtypes of attention are presented as increasing in complexity (starting with focused attention, and ending at divided attention).




*(a)* *(b)*

*Figure 1.2. (a)* The anatomy of the three attentional networks: alerting, orienting, and executive attention, adapted from Posner and Rothbart, (2007). *(b)* The conceptual sketch of the connectionist model of the attentional networks where each box represents an anatomic or functional neural network adapted from Wang and Fan (2007).

#### *1.1.1.1 Executive network*

The executive network within the attentional system involves a series of processes relating to executive function, including working memory, inhibitory control, cognitive flexibility (also referred to as set-shifting). More specifically, this network monitors rewards and conflict in experimental set ups (Posner et al., 2014). Neuroanatomically, the executive control network is primarily associated with dorsolateral prefrontal cortex and the anterior cingulate cortex (ACC) (Seeley et al., 2007; Niendam et al., 2012). These regions work in unison where the ACC is crucial for conflict monitoring and error detection – signalling when competing responses are present or when outcomes deviate from expectations (Botvinick et al., 2001) and the dorsolateral prefrontal cortex implements more top-down control by biasing processing in favour of task-relevant information and goals (Miller & Cohen, 2001). In the next subsections sustained attention, alertness, and orienting are explored in more depth due to a more direct role in information foraging and seeking.

#### *1.1.1.2 Sustained attention and Alertness*

Our attentional focus is not constant but rather fluctuates on a moment-to-moment basis, shifting between optimal and suboptimal focus (Esterman & Rothlein, 2019). Alerting refers to state of readiness whereby the brain is prepared to process stimuli, it also primes the

brain for higher-order control functions such as sustained attention. In fact, Oken, Salinsky and Elsas (2006) argue that alertness is a prerequisite for sustained attention. Posner and Petersen's model distinguishes between, tonic and phasic alertness. The former refers to a general state of arousal where an individual is maintaining readiness to process or respond to stimuli, and the later refers to brief, often sharp increase in response to a stimulus, usually triggered by a cue or warning signal in experimental set ups. Tonic alertness is modulated by the locus coeruleus and phasic alertness by the right parietal cortex and thalamus.

Sustained attention is the ability to maintain engagement or focus on a stimuli or task over a period of time (Peterson & Posner 1990, 2012). This sub-domain of attention has real world implications across academic performance, social communication, and mental health in adults (Kalechstein, Newton & van Gorp, 2003) and in children (Gallen et al., 2023 for review). Though alerting and sustained attention may rely on distinct neural mechanisms, Oken et al (2006) likened it to a state of high alertness within a task. Indeed, in experimental set ups, higher levels of alertness and sustained attention present as heightened arousal that enhances processing of incoming information in a task and improves task performance.

Esterman and Rothlein (2019) (presented in *Figure 1.3*) propose a framework that outlines distinct cognitive processes relevant to this fundamental process, this includes levels of arousal, cognitive control, opportunity cost, and a perspective on information processing. Their integrated model explains how attention to a task changes moment-to-moment and the impact of this on the information we process in that task. They posit that during an optimal attentional state, task-related information processing outweighs task-unrelated information processing, and this can be traced to an optimal level of arousal, and an effective control system. According to this model, periods of suboptimal attention (periods of low accuracy for example) could be a result of multiple distinct causes, this includes hyper- or hypo-arousal, external distraction or mind wandering.

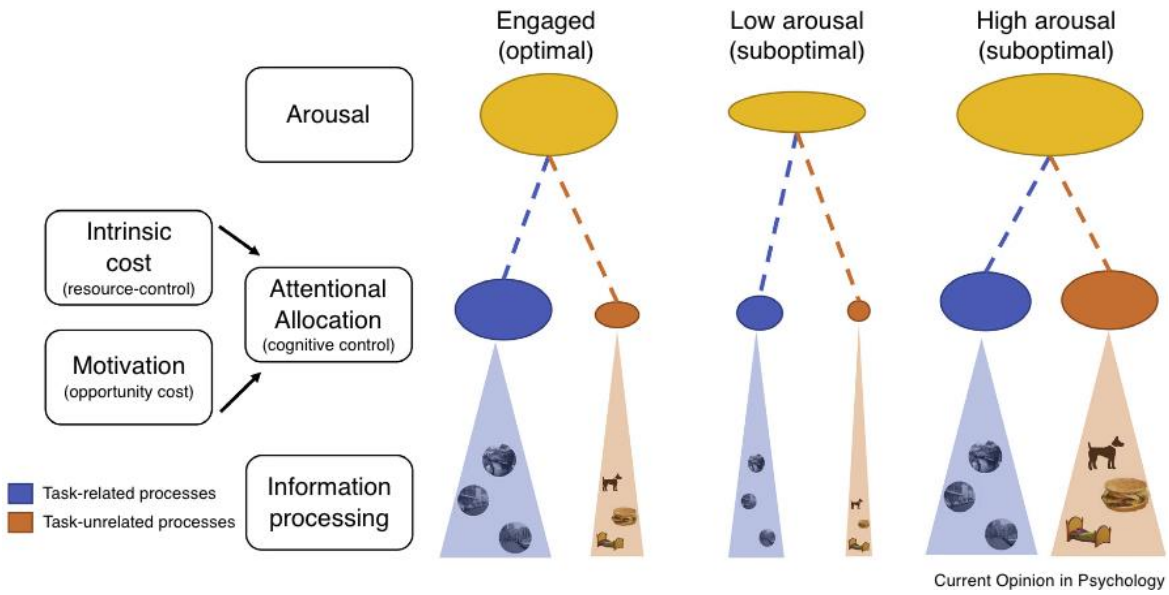



*Figure 1.3.* Model of sustained attention adapted from Esterman and Rothlein (2019) where task performance is modulated by arousal, attentional allocation and information processing. During the different attentional states (optimal, suboptimal or suboptimal), arousal determines the degree of cognitive resources available to be allocated, and cognitive control will determine the proportion of those resources to be dedicated to the task. The information processing (whether it is more task-related or more task-unrelated) is also impacted by the intrinsic cost of control and motivation.

In support of this model, evidence for the presence of optimal and suboptimal attention state was found by Yamashita et al (2021). In their fMRI study, they suggest that attentional states were identified on the basis of brain activity alone, irrespective of behaviour. One state characterised by activation in default mode network and the limbic networks, corresponded with behaviourally optimal behaviour (lower reaction time variability, and higher accuracy). The other state characterised by activation in the dorsal attention network, salience network, somatomotor network, and visual networks corresponded with suboptimal behaviour (higher reaction time variability, lower accuracy). Interestingly, they note the influence of Attention-Deficit Hyperactivity Disorder (ADHD) on time spent between these two states, specifically that those with ADHD spent significantly higher time within the suboptimal brain state compared to the non-ADHD controls.

#### *1.1.1.3 Visual orienting / attention shifting*

The brain does not process every detail of our surroundings; for example, visual processing may be prioritised at specific location by foveating the eyes or by the directing

attentional resources to a given region. This results in facilitation of target detection in the selected location while reducing target detection to other regions in the environment. Posner (1980)'s proposed orienting network implied three functional mechanisms: disengagement of attention from a specific stimulus, shifting attention towards an alternative stimulus, and then engaging attention. The posterior parietal cortex, pulvinar thalamus, and frontal eye fields are implicated as the biological foundation for these functions respectively.

Furthermore, two systems that modulate orienting (disengagement, transfer, and engagement) were outlined (Posner 1980, Posner & Petersen, 1990). The endogenous system is under the influence of individual's intentions or goals, and the exogeneous system is controlled by external variables such as object salience, outside the individual's control. These two systems can also be represented as the overt (endogenous and top-down controlled system), and covert (exogeneous and bottom-up controlled system) systems which act as two independent but interacting attentional systems (Posner & Petersen, 1990). Distinct biological mechanisms are responsible for the two orienting systems, dorsal frontoparietal network for top-down control and a ventral frontoparietal network for stimulus-driven orientation (Corbetta & Shulman, 2002).

The process of disengagement, transfer and engagement of attention is also a costly process. In experimental settings as well as in daily life, our brain needs to balance between selecting relevant information to orient towards for further processing while ignoring irrelevant distractors. However, complete inhibition of distractors would be maladaptive, as the brain must also keep track of unexpected dangers or unexpected rewards. It is well established in the literature that when environments are volatile, i.e. unpredictable, there are more rapid shifts towards novel cues, higher reaction time variability, and more frequent errors (Nassar et al., 2012; Behrens et al., 2007). When individuals are processing information from their surroundings, volatile environments introduce additional cognitive load (Nassar et al., 2012), where valid and invalid cues will be tracked. Neuroimaging studies reveal the role of dorsal frontoparietal networks for coding, selecting, and therefore orienting to relevant locations in space (Corbetta & Shulman 2002; Corbetta, Patel, & Shulman, 2008). In the case where individuals re-orient to relevant events in unexpected locations, more activation is seen in the ventral frontoparietal network (Shulman et al., 2007).

This dichotomy is well represented in research, however, has more recently been described as an incomplete system. Critiques by Awh, Belopolsky & Theeuwes (2012) for

example outline that our environments contain too much information for the orienting system to simply be driven by physical salience of the stimuli or by internal goals of the agent. They first draw on evidence for selection history impacting visual orientation, even when unrelated to current goals (Wang et al., 2005) and secondly, from literature on reward associations, where visual selection occurs due to an association with *previous* rewards, even when reward contingencies have changed (Anderson et al., 2011a; 2011b). They thus present a contemporary model integrating three sources of selection: the observer's current selection goals, selection history, and the physical salience of the items completing for processing. This presents as a model with continuous interaction between top-down and bottom-up drivers and is presented in *Figure 1.4*. Furthermore, Gottlieb & Oudeyer (2018)'s work also agrees with inclusion of 'selection history' as a key component of orienting and emphasises the role of attention and orienting as a dynamic process that samples for information in the environment in order to learn and reduce uncertainty. In more contemporary accounts, visual attention, and more specifically visual orientation is shown to operate as an active decision-maker, whereby the brain selected specific regions to progress and others to inhibit. In natural environments, a vast amount of information is processed (i.e.: attended to) to guide movement and decisions, as well as gain information and reduce uncertainty about the surroundings. We present and adopt this view of visual attention throughout our work, supported by Jacqueline Gottlieb's work presented in section **1.2** of this introduction.

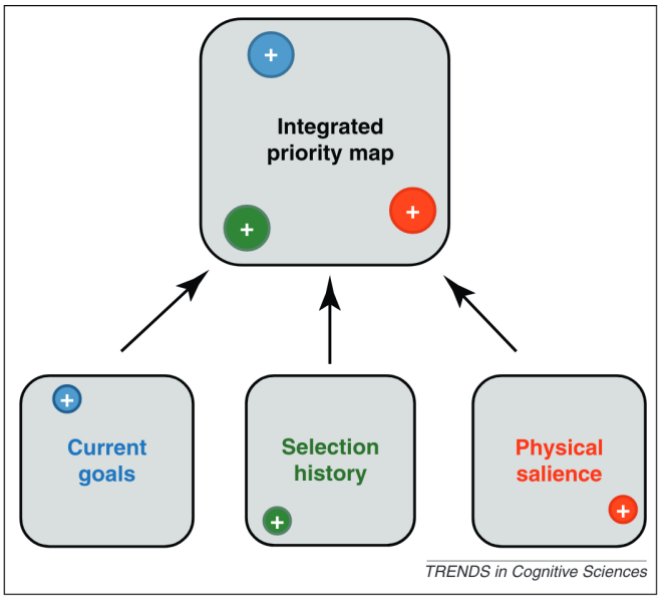



*Figure 1.4.* A schematic representation of a priority map developed by Awh, Belopolsky & Theeuwes (2012). This map integrates the three sources of selection bias: the observer's current selection goals, selection history, and the physical salience of the items competing

for attention. Although these three effects could in principle operate in a coordinated fashion, various studies have demonstrated that they can also work in direct opposition to one another. This suggests that they are distinct sources of selection bias.

In essence, the role of the of the brain is to identify meaningful or behaviourally relevant information in the environment in order to process and effectively move between sources of information. Various contextual features and internal states modulate what we select for processing, how long we stay to process it, and when we orient towards something else. Research based on classical models of attention, such as those proposed by Posner and Petersen, have revealed important mechanisms modulating orientation to different types of targets (for example known versus unknown (Posner, 1980) or predictive versus nonpredictive (Jonides & Irwin 1981)). However, according to Gottlieb (2012; 2023) the core questions about what unifies these phenomena in explaining attention control remains relatively unexplored. Her proposal is that attentional is fundamentally a mechanism for seeking out information based on its expected value for guiding future decisions, and actions (in addition to satisfy curiosity, Gottleib et al., (2013)). This "information-seeking" approach provides a suitable candidate where attentional control is not simply a selection of salient stimuli, but a strategic sampling of regions in the environment in order to reduce uncertainty. In support of this, she brings studies of *information demand* in which participants freely observe and process stimuli, as a way to investigate drivers of attentional control that modulate how we engage with and disengage from information.

*Interim summary:*

In our daily life, we make use of all elements of the attentional network – orienting, alerting and executive control – in order to process the information from our surroundings. For example, while walking outside, a salient item in your path (such as a large stone) would be orientated to. This region will be selected for processing for a period of time (it may be a fraction of a second it takes to gather enough information about the placement of the stone), following this, when you have reached a threshold and have gathered sufficient information (you were able to adjust your path to avoid the stone), you disengage your attention and orient your attention elsewhere in your visual scene. Here, the executive control system would mediate between competing stimuli and execute higher-order functions: such as switching between intention to avoid the large stone and instead deciding to step on it in

order to avoid stepping in a muddy puddle instead. An impaired control of visual attention and control can result in failure to orient to potentially harmful changes in the environment and inability to modify responses to external stimuli.

All organisms function on limited resources, it is impossible to attend to all information in our surroundings, all the time. In such information rich environments, Gottleib (2023) crucially describes information processing (and information seeking) as resource-limited process rather than a data-limited process. Meaning, it is constrained by the *internal* availability of cognitive resources to process information instead of the being constrained by the *external* quantity/quality of the information itself. This entails that the brain must strategically prioritise and allocate its limited capacity in order to sample information from locations that are expected to be the most valuable. How do human agents make decisions on what locations are most valuable when navigating environments? For example, when to look towards and process one lamppost before reaching a threshold of enough information and moving onto processing the next lamppost and so on. Do individuals differ in what this threshold is, i.e. how much information is sufficient?

While the classical understanding of attentional system discussed above can describe the functional components of attention, they do not give insight into the decisions human agents make. Particularly with regards to when they disengage from a stimulus. More contemporary models of attention present the attentional system as an active, goal-directed sampling of information, reducing uncertainty and guiding future actions. This is a contrast to previous frameworks which present the system as a passive filtering system.

### 1.1.2 Gottlieb and information seeking: Attention in a decision-making framework

Animals and humans are constantly seeking knowledge and information through their behaviours, at low levels (for example looking at a new storefront), at higher levels (such as exploring the depths of the ocean), and everything else in between (reading books, watching documentaries). Over the last decade, Gottlieb's work has expounded on the relationships between attention, information seeking, and decision making. The central idea to the framework is that instead of intuitively defining attention as a 'focusing' of the mind (as more classical literature would have described it (James, 1980)), we should attempt to understand it as a distinct form of cognitive decision. The cognitive decision to gather information involves two separate but interrelated systems of attention, namely: 'attention for action' and 'attention

for learning' (Gottleib et al., 2013). The former of which directs resources to familiar reliable cues in order to guide current and future actions, and the latter which preferentially weights novel, surprising, and uncertain cues in order to alter the agent's epistemic state, which *may* or *may not* guide future actions. Behavioural and neuropsychological studies in rats have found evidence for these two distinct attentional systems (Holland & Maddux, 2010; Pearce & Mackintosh, 2012).

Additionally, similar to how we describe the visual orienting system through endogenous and exogenous orienting, motivations behind information seeking can be intrinsic or extrinsic. For example, extrinsically motivated information gathering can be to look at the traffic crossing light at a busy intersection (Tatler et al., 2011). Examples of intrinsically motivated gathering can be reinforcement learning tasks or other reward maximising tasks. Although an interesting note here is that some studies show that processes required to compute informational value differ from those in reward maximising decision making tasks (Tatler et al., 2011; Gottlieb, 2012). Specifically, that for the former, the motivation behind a shift in attention is not value per say, but the need to learn (i.e.: update one's predictions through new information). Along the same lines, we also know that humans show intrinsic curiosity and gather information without an apparent ulterior motive, which suggests that the brain is naturally able to generate intrinsic rewards, and assigning value to the information being sought and gathered (Berlyne 1960; Redgrave et al., 2008; Bromberg-Martin and Hikosaka, 2009). Support for this idea comes from observations of the dopaminergic system, which modulates reward processing in the brain, this is further discussed in subsection **1.1.4** of this introduction.

The ocular system's role in gathering information is critical, saccades for example are specialised for sampling information to assist ongoing tasks and in a goal-directed fashion. This implies that the brain has mechanisms for assigning priority to sources of information based on demands of the task. Given that gathering information about our environment is crucial for survival, this system has evolved to be highly efficient; Najemnik and Geisler (2005) for example, show that visual search in humans is nearly optimal compared with a computed "ideal searcher" that uses precise statistics to maximise search efficiency in a visual scene.

In the integrated model proposed by Gottleib and Oudeyer (2018), as presented in *Figure 1.5,* presents attention in itself is an action aimed at sampling information and is

guided by an agent's beliefs about the value of information. More recent work by Gottlieb (2023) expands on this and proposes a view of attention as a mechanism for optimising prediction accuracy; in other words, we selectively sample stimuli based on the expected information gain (EIG). Stimuli that are more reliable predictors of resolving prior uncertainties are therefore more likely to be selected. Here again Gottlieb presents attention as an active, goal-directed process which takes into account costs associated with information seeking (eg: effort, opportunity costs) in order to decide where attentional resources should be allocated to maximise the utility of the information being sought. Gottleib's framing of attention as a tool for sampling information in the environment and as a tool for gaining predictive accuracy places the attentional processes at the centre of decision-making, learning, and information seeking. Another model from their work is presented in *Figure 1.6* which presents the idea of predictive information gain based on prior and posterior observations. Essentially, by framing attention as a tool for predictive accuracy, the theory highlights its role in decision-making, learning, and adaptive behaviour, extending beyond traditional views of attention as enhancement of selected stimuli.

Mechanisms of information gathering are widely distributed, involving interactions between frontoparietal areas involved in orienting attention and gaze (Bisley & Goldberg, 2010), executive control involving frontal areas (White at al., 2019), and the neuromodulory dopaminergic and norepinephrine systems (Bromberg-Martin & Hikosaka 2009).

The original classifications of the attentional system by Posner and Peterson present the attentional system as anatomically separate from processing systems. Attention, according to these models, therefore, was not necessarily a component of decision-making, learning and adaptive behaviours. Contemporary representations of attention (such as those put forth by Gottlieb, Anderson, 2011, and Krauzlis et al., 2021) incorporate important cognitive processes within their descriptions of attentional models. By highlighting attention as a tool for predictive accuracy, information sampling, and behavioural selection, the newer theories build on classical theories (such as proposed by Posner and Peterson), and incorporate broader understanding of cognitive neuroscience, illustrating a more complex role attention plays in learning, choice, and behaviour.

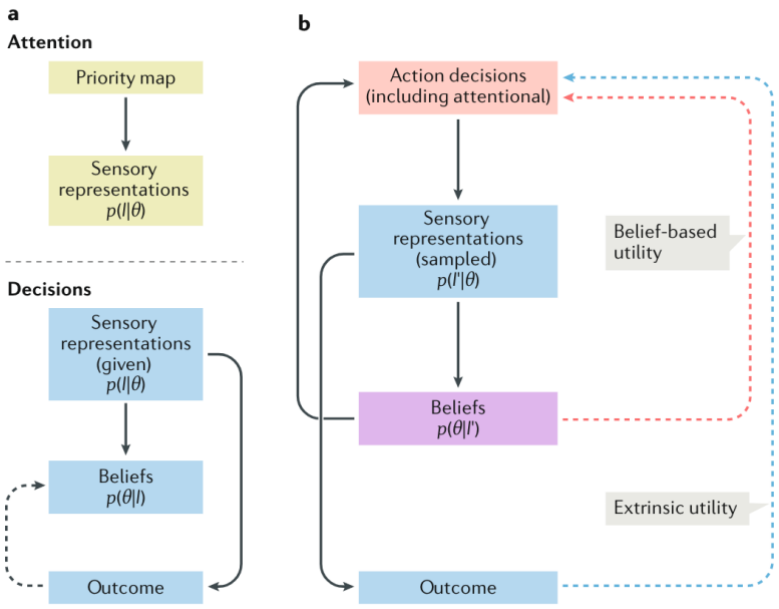



*Figure 1.5.* The proposed architecture of attention and decision making based on the current literature, adapted from Gottleib and Oudeyer (2018). Attention is envisioned as starting with a priority map that specifies the to-be-attended feature or location and modulates the sensory representations in earlier visual areas. Decision-making on the other hand operates on given sensory representations. An integrated attention and decision-making model proposes that attention in itself is an action aimed at sampling information. This is guided by an agent's beliefs about what information is valuable. The value of the information is determined by its utility in achieving external goals as well as intrinsic cognitive value (which drives curiosity and exploration).

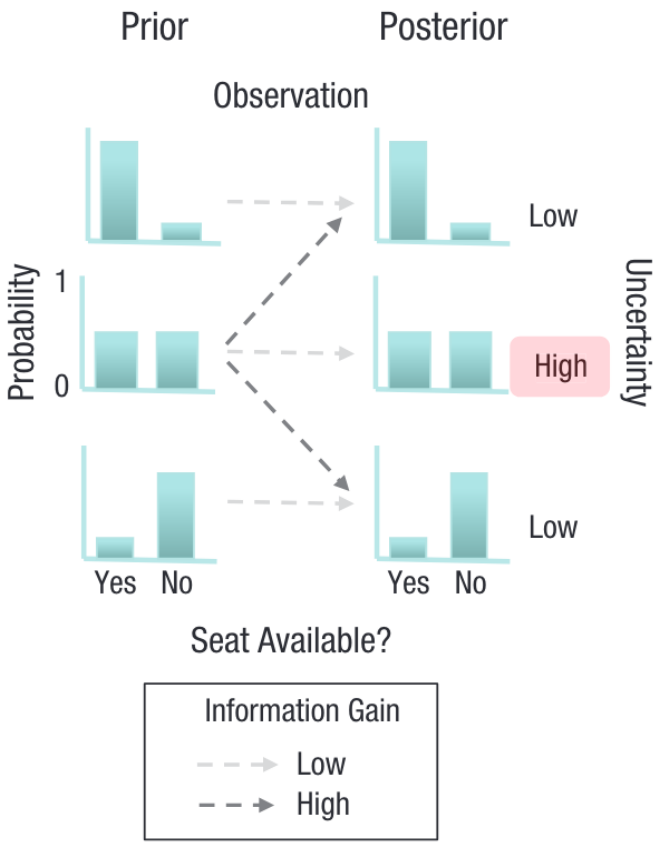



*Figure 1.6.* Adapted from Gottleib (2023) where information gains are defined in terms of uncertainty in an example where a restaurant goer is inquiring about availability of a restaurant seat where the seat may or may not be available. The probabilities before and after obtaining an observation are referred to as, respectively, the prior and posterior beliefs. For both distributions (prior and posterior), uncertainty is high if the alternatives are equally likely (middle row, pink) and lower if one alternative is more probable (top and bottom rows). The dashed arrow shows information gain, which is high for transitions from a high prior uncertainty to a lower posterior uncertainty and low otherwise.

### 1.1.3 Attention as Information Foraging

The attentional networks summarised above, work together in a coordinated fashion in order to select and process the information in our surroundings and make decisions on when to engage with stimuli and when to disengage in favour of other stimuli. One of the theoretical frameworks put forward that explains the interplay between attentional systems and information seeking is the foraging hypothesis.

Foraging has been extensively studied in animals (Pyke, Pulliam, & Charnov, 1977; Stephens, Brown, & Ydenberg, 2007; Hills, 2006) and has also been experimented on in human psychology (Kolling et al., 2012). Regarding animal foraging, organisms in the wild seeking resources such as food, shelter, and mates, must strike a balance between harvesting (and therefore depleting) a known source, or venturing out towards a new source (which may have unknown rewards). Typically, an animal exploits a known resource (also referred to as a patch or cluster), until their acquisition rate falls below a threshold, at which point they are may seek a new patch. Elements such as danger, rate of depletion, and source richness in the environment may all play a role in regulating individual decisions (Stephens, Brown & Ydenberg, 2008).

The foraging hypothesis as a model of attention suggests that seeking information (or reward) from our surroundings and seeking resources such as food requires a similar mode of action; such that we harvest or exploit our current source of information until the source depletes. When this source depletes, the effort needed to continue extracting information outweighs the relative benefit. It is at this point that a shift of attention might occur to seek information elsewhere (Calhoun and Hayden 2015; Hills et al., 2015). There is also evidence of individual variation in prioritization of different information sources (Desimone and Duncan, 1995) and because the visual orienting system is under the influence of the dopaminergic system, it has been suggested that novel incoming information are assigned a reward value (Snyder, Blank, and Marsolek, 2008).

Concepts of optimal resource acquisition are well-studied in animal foraging. The marginal value theorem (Charnov, 1976) for example, attempts to mathematically demonstrate how long a foraging animal should stay within a resource patch while the resource is depleting, before moving to another potentially richer source. Strategies can change depending on external dangers in the environment, uncertainty and patch richness. In psychology research, the multi-armed bandit problem is one that taps into a similar decision-making paradigm where a decision-maker must choose between multiple options with known and unknown rewards in order to maximise rewards over time. In other words, participants must decide between exploiting a known reward sources (usually monetary rewards or points-based rewards) and exploring unknown sources in the environment which may result in higher reward. Gittins & Jones (1974) have also developed an optimal choice strategy for this task naming it the Gittins Index.

Optimal choice strategies in multi-bandit tasks provides a useful framework that can be applied to human information foraging. Human seeking of information involves a similar balancing between exploiting known sources and exploring more uncertain sources. Hills et al., (2016) describes how the entire cognitive system can be characterised by trade-offs between exploration and exploitation, applying to low level cognition such as spatial navigation, and to higher level cognition such as problem solving.

A key point to note here is that foraging for food involves an agent deciding between continuing to engage with the current source which is depleting or to move off and search or engage with another source. The component of 'depletion' is rarely incorporated in experimental paradigms such as the multi-bandit tasks. The concept of 'depletion' in information seeking is crucial as it necessarily exists in the real world. Just like a move towards a new patch is likely to occur when acquisition rate from the first patch drops below a threshold, one could expect that attentional focus shifts to a new location when the information gain from a location drops below a threshold.

#### *1.1.3.1 Exploration and Exploitation*

Exploration and exploitation are two foundational strategies by which we process and seek information in our environment. They represent opposing sides in a decision-making paradigm such that exploration relates to choice selection with unknown outcomes or unknown information, and exploitation relates to choice selection based on known outcomes or known information. Typically, within experimental paradigms a balance is sought between exploratory and exploitative strategies taking into account uncertainty of the environment, reward contingencies, and other contextual factors (Cohen, McClure, & Yu 2007). People were shown to arbitrate between exploration and exploitation in all avenues of behaviour, and the correct decision between the two depends on a combination of environmental demands (van Dooren et al., 2021; Chang, Jara-Ettinger & Baskin-Sommers 2022; Brown et al., 2023), cognitive capacity (Hills & Pachur 2012), personality traits and past experiences (Gonzalez & Dutt, 2011; Mulder et al., 2012). These modulators are present in human and animal behaviour (See Mehlhorn et al., 2015 for review).

The exploration-exploitation trade-off literature is vast, covering biological mechanisms (Cohen, McClure & Yu, 2007), judgement paradigms (Gureckis & Love, 2019), behavioural economics (Wilson et al., 2014), and computational neuroscience (O'Reilly, 2013) just to name a few. Wyatt et al (2024) identified three key behavioural choice

paradigms that are typically used to study explore-exploit decision making in psychology: Foraging paradigms (different to foraging as the theoretical framework), reinforcement learning, and information search. An overview of the exploration-exploitation trade-off is presented in *Figure 1.7* and shows that in many experimental paradigm's decisions vary exploration and exploitation. Crucially, a balance must be struck between when to explore and when to exploit as this is the most advantageous and flexible decision-making strategy (Addicott et al., 2017). Imbalances or extremely biased explore-exploitation decision making is disadvantages in natural environments but also within most experimental paradigms (Addicott et al., 2017).

*Foraging* here refers to the search and gathering of resources by searching within a single 'patch' (a resource rich area) or between multiple 'patches'. *Reinforcement learning* relates to paradigms in which agents learn the probabilistic associations between actions and rewarding outcomes through trial-and-error feedback over time in order to maximise resource gains (Sutton & Barto, 2018). *Information search* refers to paradigms in which individuals actively seek out knowledge or reduce uncertainty by sampling the environment for information from multiple potential sources before identifying a final target or committing to a decision (Gottlieb & Oudeyer, 2018). The paradigms used in this thesis are conceptualised within the framework of information foraging.

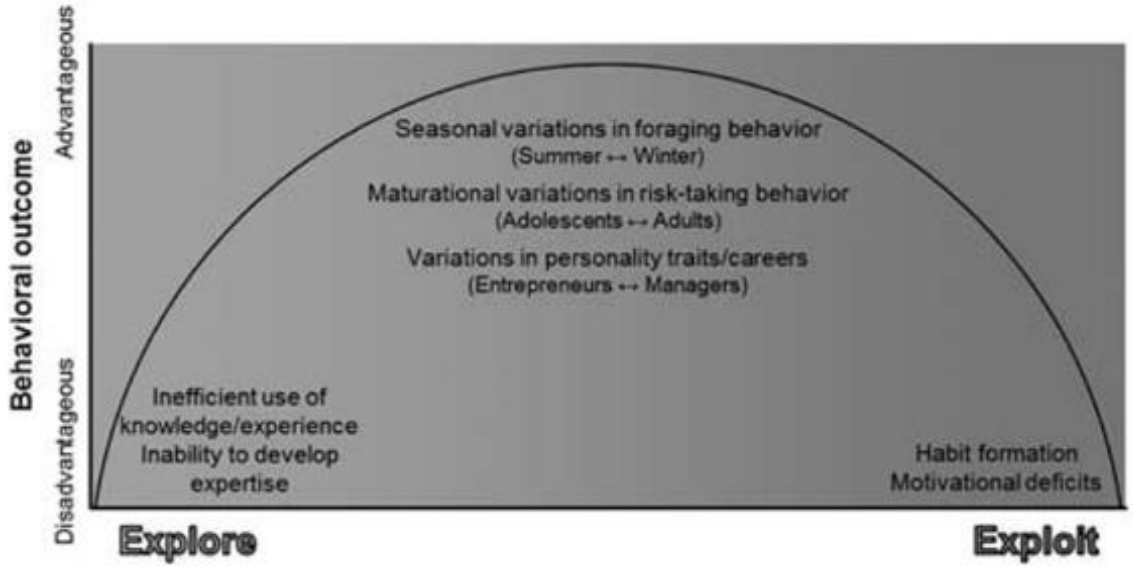



*Figure 1.7.* Conceptual overview of the explore/exploit trade-off adapted from Addicott et al., (2017). Decisions may vary along a continuum between exploration and exploitation with the most advantageous behaviours occurring around a point of balance between the

two. Around this balance, there may be slight externally or internally driven biases towards one decision of the other.

### 1.1.4 Involvement of the dopaminergic system

Research is also beginning to untangle the neural mechanisms that underlie exploratory and exploitative behaviours. Midbrain dopaminergic activity is generally implicated in processing of predictions and rewards (Schultz, Dayan & Montague, 1997), as well as playing a role in the prioritization of different information sources (Desimone & Duncan 1995). Hills (2006) describes the key role of dopamine in goal-directed behaviour in animals and humans, including foraging behaviour such that higher levels of dopamine generally associate with less exploration (or less novelty seeking), and lower levels associate with more exploration (more novelty seeking). These can also be mapped onto various conditions associated with atypical dopaminergic systems, such as Schizophrenia, Obsessive-compulsive disorder, Parkinsons, Autism, and Attention deficit hyper-activity disorder, as presented in *Figure 1.8*.

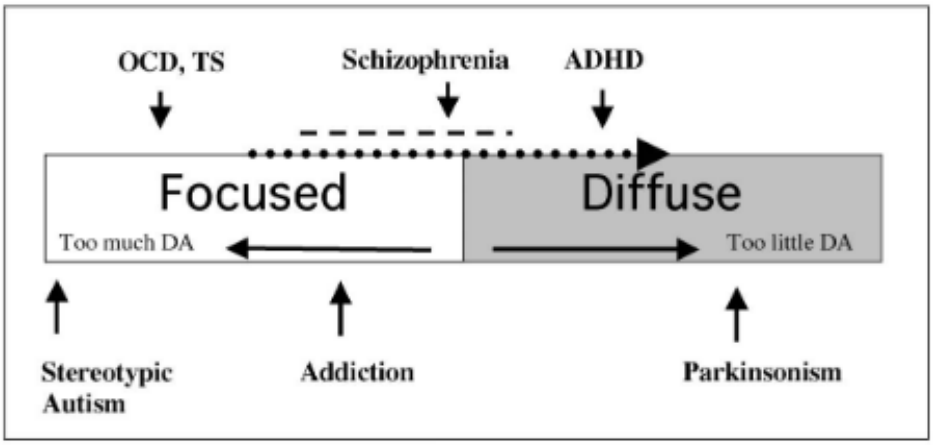



*Figure 1.8.* A visual characterisation of goal-directed pathologies along a continuum of dopaminergic activity adapted from Hills (2006). The placement of the pathologies is not perspective, but based on general descriptions and prevailing theories of the underlying mechanisms. Though dopamine is a key control factor in these pathologies, other mechanisms are still involved. OCD is obsessive-compulsive disorder and TS is Tourette's syndrome.

The dopaminergic system plays a key role in the attention (Clark, Geffen & Geffen, 1989; Schneider et al., 2014; Anderson et al., 2016) and thus is implicated in literature relating to foraging (Le Heron et al., 2020) and exploration (Costa et al., 2014; Chen et al., 2024; Kayser et al., 2015). In addition, this system is especially relevant to cognitive control and executive function (Floresco and Magyar, 2006; Cools, 2016). At a basic level, activation of dopamine neurones, correlates with detection of more salient stimuli (Schultz et al., 1997). Disruption to this system such as hypo- and hyperactivation of dopamine neurones correlates with dysfunction of behavioural processes associated attention, for example the disruptions that are seen in Parkinson's disease (Dujardin et al., 2013), and in Schizophrenia (Braff, 1993; Braff & Light, 2004).

The neuromodulatory role of dopamine in motivation, reward prediction, and learning is well established (Schultz, 2016) and is also linked to its role in the visual orienting system. In other words, individuals associate a particular reward value to stimuli during visual processing, and this value association is dopamine modulated (for review see: Vitay and Hamker, 2007). In more complex experimental set ups involving decision making, the influence of dopamine function is still apparent in both short-term prediction of rewards (phasic response) and in long term learning of average rewards (tonic response) (Schultz 2016).

It is also unsurprising then that reward-based decision-making involving deliberating between exploration and exploitation are also modulated by the dopaminergic system. Generally, exploitation relates to preference for known/predictable reward contingencies, whereas exploration is associates with novelty and unpredictable reward. Typically, dopamine in the striatum region of the brain, associates with signalling existing and predicting reward contingencies (Schultz, Dayan, & Montague, 1997; Bayer & Glimcher, 2005) and therefore genes associated with striatal dopamine signalling tend to associate with exploitation biases (Frank et al., 2009). Exploration importantly provides information about the environment for future decisions (Cohen, McClure, & Yu, 2007; Daw et al., 2006; Mehlhorn et al., 2015) and the implicated genes are related to prefrontal dopamine function (Frank et al., 2009; Krebs et al., 2009). A more recent investigation by Cremer et al (2023) explains that dopamine plays a significant role in directed exploration whereas noradrenaline modulates more random explorations.

In many experimental set ups, administering variants of dopamine agonists and antagonists result in altered exploration and exploitation of choices. When individuals are administered cabergoline, a dopamine agonist, their foraging behaviour altered such that it led to participants leaving bad patches much earlier and richer patches only slightly earlier and therefore performing closer to computed predicted optimal behaviour (Heron et al., 2020). Here, cabergoline's role was attributed to altering D2 receptor activity in the mesolimbic dopaminergic system. In addition to this, Chakroun et al., (2020) found that participants on L-dopa were more likely to exploit known options with higher rewards and presented significantly fewer directed explorations. Here, L-Dopa's role is attributed to increasing dopamine signalling particularly in the striatum. It is also known that novel stimuli elicit an excitatory response from dopaminergic neurones and activate cortical regions receiving dopaminergic input, the reward here being reducing uncertainty about the novel stimuli (Bunzeck and Düzel, 2006; Costa et al., 2014).

The findings mentioned above along with others (Beeler et al., 2010) demonstrate that exploration and exploitation may be relying on distinct neural systems (striatal dopamine for exploitation and prefrontal dopamine for exploration), however the causal links between dopamine transmissions, underlying neural mechanisms, and exploration and exploitation are not entirely clear. What we do know is that the dopaminergic system plays an important role in deliberating between rewarding outcomes and likely through this mechanism, drives exploratory and exploitive behaviours. Either by (a) associating uncertain options with higher rewards or (b) associating known reward contingencies with higher rewards. Although this does not fit in with the more simplistic model proposed by Hills (2006) (*Figure 1.8*), it may be the case that the Hills model is more aligned with dopamine synaptic resistance proposed by Landry (2021) (which is further explained in subsection **1.1.5.3**) or with research showing lower dopamine transporter availability and lower D2/D3 receptor densities in the mesocorticolimbic pathway (Fusar-Poli et al., 2012). Based on this, the role of the dopaminergic system is clearly important, however the specific mechanisms by which it affects behaviour, decision-making, and attention, is not as straight-forward. This is touched upon again in section **1.1.5.3.**

It has previously been suggested that the underlying neural system in reward seeking, also supports information-seeking (Bromberg-Martin & Hikosaka, 2009; Bromberg-Martin & Hikosaka, 2011; Blanchard et al., 2015, Ligneul et al., 2018), the implication being that maximising rewards in a paradigm is similar to maximising information due to their potential

of helping us in decision making (Daw et al., 2006; Nakamura 2006; Nakamura & Komatsu, 2019; Costa et al., 2014). This is evidenced by early neuroscience studies of information seeking in monkeys which report that the reward prediction error system had similar signals for information and for rewarding outcomes. In other words, dopamine signalling for higher than predicted rewards (e.g.: more water than predicted) is similar to dopamine signalling in an informative cue (e.g.: more information than predicted) (Bromberg-Martin & Hikosaka, 2009; Bromberg-Martin & Hikosaka, 2011). The anticipation of acquiring new information activates dopaminergic pathways, often in the mesolimbic and mesocortical dopaminergic pathways (Schultz 2016). This activation, not only motivates the rewarding outcome (gain of information) but also enhances learning and encoding of that information for later decision making (Cools, 2011).

*Interim summary*:

The dopaminergic system primarily driven by the neurotransmitter dopamine, plays a critical role in the attentional system, guiding it to behaviours that increase rewards and information about the surroundings. This system encodes and reinforces behaviours with rewarding outcomes, and these associations become important for motivation and decision making. When stimulus-reward associations have been formed, they persevere even in the case that the reward has been devalued (Changizi, Mcgehee & Hall, 2002).

When it comes to information seeking, this same system has been suggested to modulate responses to novelty, driving curiosity and exploratory behaviour (Bromberg-Martin & Hikosaka, 2009; Bromberg-Martin & Monosov, 2020). The dopamine system is thus necessary for survival because it underlies key behaviours such as seeking food, and information about our surroundings.

Several lines of research have found that there are individual differences that modulate dopaminergic functioning. It is subject to variability, which impacts how individuals process and anticipate rewards and therefore how individuals process information around them. Individual differences in dopamine receptor density, dopamine synthesis, and dopamine reuptake efficiency associate with variations in how people experience reward, motivation and cognitive engagement (Dreisbach et al., 2005). In the Dreisbach et al study, both blink rates and dopamine gene polymorphisms were used as a measure of dopamine variation. These differences have also been seen to relate to different personality traits and cognitive styles (Cohen et al., 2007; Smillie & Wacker, 2014).

### 1.1.5 Neurodiversity and information seeking

#### *1.1.5.1 Autism*

Autism or autism spectrum disorders (ASD) is a heterogeneous condition and presents on a wide range of scales and severities, however, are typically characterized by difficulties in social communication, repetitive or restrictive behaviours and interests, and co-occurrence with other conditions (Masi et al., 2017; Simonoff et al., 2008). These difficulties have a significant impact on daily life, affecting communication that is in line with social norms, such as speaking with an appropriate volume, interacting at appropriate physical distance, and detecting and adapting communication in response to gestures and facial expression (Hirota & King, 2023, for review). In relation to restrictive behaviours, repetitive movements (such as hand flapping) or repetitive speech is common, it also manifests as requiring others to speak or behave in a specific way and insist on adhere to prescribed schedules or activities (Hirota & King, 2023, for review). The apparent increase in the prevalence of ASD has also become a topic of great debate. In Briton, Bertnand et al., (2001) investigated the prevalence of ASD in the population, and reported that rates were significantly higher than what was noted 30 years earlier. This is also the case in various other countries, including United States of America, Canada, Germany, France, Sweden, Norway, and Finland (Honda et al., 2005 for review). This increase may be attributed to various factors, for example: changes in diagnostic criteria over time, introduction of new assessment instruments, increase in inaccurate diagnoses, and increase in awareness and acceptance of Autism (Matson & Kozlowski, 2011).

Autism is believed to be present at birth and usually diagnosable by 18 months of age (Kuban et al., 2009). Genetic research also shows that ASD has a strong heritable component, from twin studies estimating heritability between 64-91% (Tick et al., 2016). Advanced genome association studies have identified potential risk genes (Satterstrom et al., 2020), however strong evidence indicates both genetic and environmental factors (Kim and Leventhal, 2015; Newschaffer et al., 2007). Commonly, individuals diagnosed with Autism present with comorbid psychiatric conditions, including social anxiety disorder, ADHD, and intellectual disability (Simonoff et al., 2008; Mannion and Leader, 2016).

Findings relating to core symptom clusters (i.e.: social communication and repetitive, restrictive behaviours) are robust, however, there are often mixed findings in relation to cognitive differences. Some research support accelerated abilities (Muth, Hönekopp, &

Falter, 2014; O'Riordan et al., 2001; Plaisted, O'Riordan, & Baron-Cohen, 1998), whereas others report impaired abilities (Keehn et al., 2010; Minshew & Goldstein, 1998).

Visual attention is one area that has received interest, findings are not completely clear however research does point towards presence of differences between typically developing individuals and those on the autism spectrum (Allen & Courchesne, 2001; Courchesne et al., 1994; Johnson et al., 2007; Keehn et al., 2010; Renner, Klinger, & Klinger, 2006). Visual attention is of particular importance, being an early-developing aspect of cognition, and playing an integral role in the development of social functioning through mediating joint attention (Mundy & Jarrold 2010), which is consistently found to be impaired in ASD (Leekam, Lopez, & Moore, 2000; Dawson et al., 2004). There is some emerging evidence for difficulties in shifting attention (Keehn et al., 2013; Landry, Mitchell, & Burack, 2009), however not consistently reported (Dichter et al., 2010). Keehn et al., (2013) review various literature related to attentional networks (i.e.: alerting network, orienting network, and executive control network) in Autism research and conclude that attentional networks are atypical from early on, suggesting that attentional disengagement may underlie all other differences and symptoms. The summary of their review on attentional dysfunction in ASD is presented in *Figure 1.9*.

Although research relating to attention and cognitive function in autism is vast, still with many unanswered questions, here we adjust our focus to attention as a mechanism for information selection and processing. There has been some interest in understanding developmental conditions such as autism through the lens of foraging and exploration. For example, in a large-scale foraging study by Pellicano et al., (2011) where children were asked to search for target locations in a 4x4m laboratory set up, they reported that participants with autism had search patterns that were less optimal with fewer systematic searches compared to typically developing children. They also reported higher proportion of revisitations in children with ASD. Another investigation into exploratory behaviour in ASD, used social and non-social information found that participants aged 2 to 18 preferred social stimuli in comparison to non-social stimuli (Elison et al., 2012). Importantly, the authors reported that the number of images visited per unit of time increased with age, however this increase was steeper in typically developing participants in comparison to ASD participants. In young children, decreased exploratory behaviour was reported in a task where children were expected to find hidden objects different containers, in addition, children with ASD spent less time in 'active exploration' and opened fewer containers (Pierce & Courchesne, 2011).

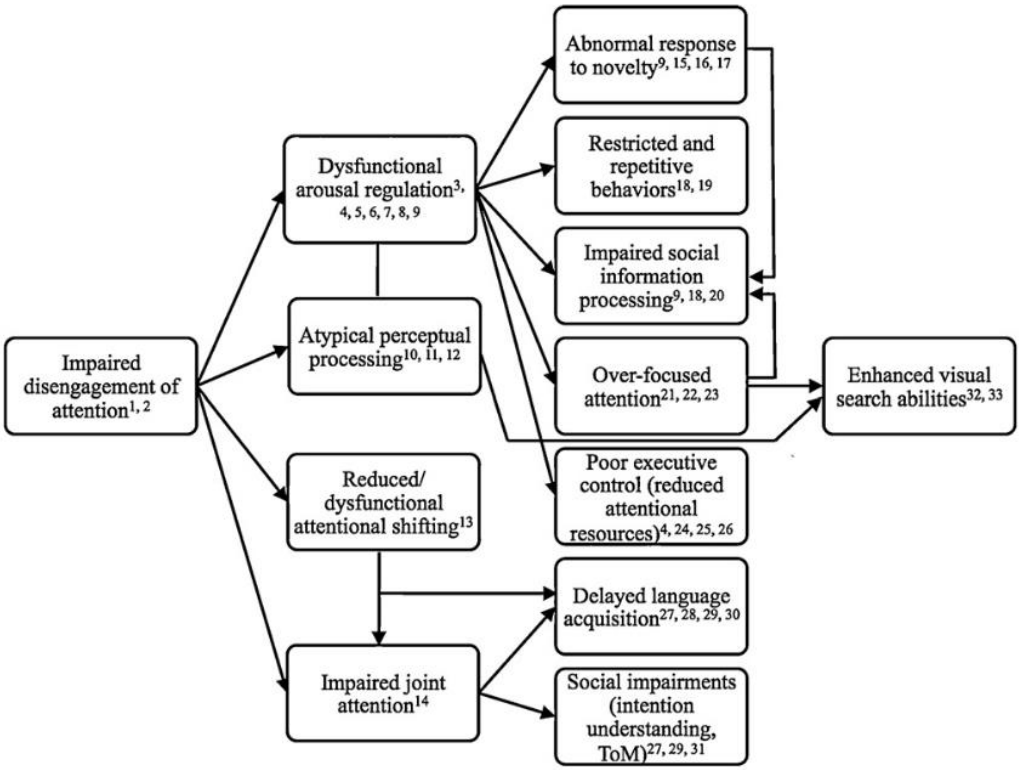



*Figure 1.9.* Graph adapted from Keehn et al., (2013) on the contribution of attentional dysfunction to the autism phenotype based on literature reviewed in Keehn et al., (2013).

#### *1.1.5.2 ADHD*

Attention-deficit/hyperactivity disorder (ADHD) is a condition, as the name suggests with notable alternation in attention and hyperactivity that interferes with typical functioning and development. The three core symptoms include (but are not limited to): inattention, hyperactivity, and impulsivity (APA, 2013). Inattention manifests as difficulty in sustaining focus, failing to give close attention to details, and being easily distractible. Hyperactivity/impulsivity may involve excessive fidgeting, inability to stay seated, interrupting others, and feelings and actions associated with restlessness. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) the diagnosis of ADHD requires the presence of these symptoms that are out of proportion to what is expected for an individual's age, developmental level, and intelligence. These symptoms should also be present in different settings, eg: home, school, or work. Like autism, ADHD is a neurodevelopmental condition and therefore is typically diagnosed in childhood, but individuals remain affected by their ADHD symptoms into adulthood (Biederman et al.,

2012). Individuals with ADHD have lower rates of high-school graduation and completion of postsecondary education (Loe & Feldman, 2007), and poor peer relationships (Hoza, 2007).

Following the DSM-5 criteria, ADHD can be classified into three main subtypes: predominantly inattentive subtype (ADHD-I), predominantly hyperactive/impulsive subtype (ADHD-H), and combined subtype (ADHD-C). These subtypes are typically differentiated from one another on inattention symptoms, associated features, and responsiveness to stimulant medication (Geurts et al., 2005; Carlson, Shin, & Booth, 1999). However, the presence of separate and distinct subtypes is not always clear-cut. Specifically with regards to executive function (EF), where some research reports that only ADHD-C and ADHD-H associates with EF related deficits (Barkley, 1997; Houghton et al., 1999; Nigg et al., 2002), and others find no difference in EF between ADHD-C and ADHD-I (Chhabildas et al., 2001; Guerts et al., 2005). Gender further adds a layer of complexity in the expression of different subtypes of ADHD (Nigg et al., 2002).

Similar to ASD, the root causes of ADHD are not clear, however both genetic and environmental factors play a role (Faraone et al., 2005; Thapar et al, 2013). It has high heritability with twin studies showing a heritability of 76% (Faraone et al., 2005) and several genome association studies showing risk genes (Thapar et al., 2005; Park et al., 2005). A large body of research has generally implicated fronto-striatal network abnormalities as critical contributors to ADHD symptomology (Casey, 1997; Bush et al., 2005) as well as several other areas including visual and default mode network (Liddle et al., 2011). Both cognitive performance and motivated behaviour have been linked to catecholamine signalling, primarily in dopamine in the mesocorticolimbic brain pathway (Aarts, van Holdstein, & Cools, 2011). ADHD is associated with lower dopamine transporter availability and lower D2/D3 receptor densities in the mesocorticolimbic pathway (Fusar-Poli et al., 2012).

Findings relating to the core symptom clusters of inattention, hyperactivity, and impulsivity are robust (Wender, Wolf, & Wasserstein, 2006; Luo et al., 2019). However, research on cognitive differences reveal a complex picture, with some studies reporting impairments in certain domains while others find no significant differences, or even accelerations in specific contexts (Kofler et al., 2020; Sjöwall et al., 2013). For instance, Kofler et al., (2020) conducted a meta-analysis and found that on average, children with ADHD do show significant deficits in working memory, inhibition, and vigilance. An

important note from this analysis was that these effects were often on very small scales and ADHD tended to be characterised by substantial heterogeneity. Some lines of research have also identified potential cognitive accelerations, for example superior performance on creative tasks (Boot, Nevicka, & Baas, 2020; White & Shah, 2006).

Visual attention is one area that has received considerable interest in ADHD research. While findings are not entirely uniform, research points towards the presence of differences between typically developing individuals versus those with ADHD. For example, there is evidence for differences in sustained attention, as previously stated from Esterman & Rothlein (2019)'s framework of sustained attention, where individuals with ADHD spend a disproportionate amount of time in suboptimal attention states. Children with ADHD have demonstrated weaker alerting and executive attention ability, but matched orienting ability in a study using a modified version of the adult attention network test (ANT-I) (Mullane et al., 2010).

Individuals with ADHD also exhibit altered foraging and information seeking behaviour. Specifically, that ADHD associated with exploration-biased foraging strategies, where individuals shift between tasks, rewards, and information sources more frequently, (Hauser et al., 2015). Hauser et al., (2014) investigated decision-making and learning mechanisms in adolescents with ADHD and reported that learning rates were largely preserved in the ADHD sample, however the adolescents with ADHD were significantly more exploratory during probabilistic reversal learning task. Patch-leaving paradigms also show that individuals with ADHD abandon resource patches sooner than neurotypical controls, which suggests a lower threshold for switching and potentially due to impulsivity or to heightened sensitivity to diminishing rewards (Addicott et al., 2021). ADHD is also associated with novelty preferences in studies related to learning (Sethi et al., 2018).

1. *1.1.5.3 Dopaminergic system in Autism and ADHD*

Atypical dopaminergic functioning has been implicated in a range of conditions, for example Parkinson's, Schizophrenia, OCD, Autism, and ADHD. Hills (2006) describes dopamine as the dominant modulator of goal-directed behaviour and characterized pathologies of goal-directed behaviours along a continuum of dopaminergic activity (*Figure 1.8*). Essentially, when dopaminergic activity is high, behaviour is streamlined and in extremes, it is stereotypic. Whereas when dopaminergic activity is low, behaviour lacks focus and does not persist. It is notable that based on the proposal by Hills (2006), ADHD and

Autism seem placed on opposing sides of the spectrum with regards to goal-directed search behaviour. While autism and ADHD are complex conditions, atypical dopaminergic functioning has been proposed as one contributing factor to some presented traits (Paval 2017; Hauser et al., 2016). There is, however, no unifying mechanism that has been used consistently to describe how alterations in the dopaminergic system of individuals with autism and with ADHD can present as differences in information seeking behaviours.

Paval (2017) for example, proposes that several dopaminergic pathways are implicated in ASD, including the mesocortical, mesolimbic, and nigrostriatal pathways. Disruptions in these pathways present as core features of ASD, namely restricted repetitive behaviours and social communication difficulties. It was also highlighted that hyperdopaminergic activity in the striatal pathways lead to repetitive behaviours and hypodopaminergic activity in the mesolimbic pathways may play a role in social reward processing. Summary of this proposed model is presented in *Figure 1.10.* Evidence for the former is also established by De Risi et al., (2021) where they reported autistic-like behaviours in other diseases such as mucopolysaccharidosis (metabolic disease caused by malfunction in breaking down glycosaminoglycans) was due to striatal hyperdopaminergic activity.

*Table 1.1*

*Summary table based on literature review by Landry, 2021 on the cue-induced attention and task switching in ASD and ADHD. Behavioural patterns 1-5 represent areas where ASD and ADHD present opposing traits, 6-8 represent areas of overlapping traits. Additional column for how these behaviours might present in foraging paradigms is added where **Behavioural Patterns** directly relate to foraging.*

| **Behavioural Patterns** | **ADHD** | **ASD** | **Justification** | **Predicted foraging behaviour** |
|---|---|---|---|---|
| *1. Attention-switching* | Frequent | Infrequent | Overall attention-switching frequencies are higher in ADHD (Taylor, 1998) and less frequent in ASD (Zwaigenbaum et al., 2005). These contrasts are present in sensory modalities such as auditory processing (Kilpelainen et al., 1999; Lepisto et al., 2005) and also in internal diversions such as mind wandering which is frequent in ADHD (Franklin et al., 2014) and rigid-thinking patterns reported in ASD (Peterson et al., 2009). | |
| *2. Task-switching* | Frequent | Infrequent | Hyperactivity – as evident by high rate of task shifts is more commonly observed in ADHD (Roberts, Ray, & Roberts 1984; Taylor 1998) in academic tasks and between different toys presented by examiner. Inflexible behaviour is commonly observed in ASD, such as writing on a blackboard for hours (Kanner, 1943), and continuing to run circles after a game of tag is finished (Korn-Bursztyn, 2011). | High ADHD = shorter patch staying time or early departure from source of information, potentially before sufficient information gain. |
| *3. Risky behaviour* | High | Low | Risky behaviour is reported in various domains of everyday life for adults with ADHD, including in recreational, health, transport-related, and | High ASD = longer patch staying time or |

| | | | | |
|---|---|---|---|---|
| | | | financial decision-making (Brown & Casey, 2016). In ASD on the other hand, there are low levels of risky behaviours in consumer purchasing decisions (Baas & van Raaji, 2010) and generally reported to have an intolerance of uncertainty and fear of unknown (Matson & Love, 1990; Wigham et al., 2015). | persistence with source of information, potentially after value has significantly depleted. |
| *4. Delay aversion* | High | Lower | Children with ADHD, in comparison to children without ADHD show high aversion to delay, as exhibited by extreme discounting of future rewards in points earned in computer games, or cookies hidden under cups (Critchfield & Kollins, 2011; Paloyelis, Asherson & Kuntsi, 2009). This is also the case when comparing with children with ASD (Antrop et al., 2006). Additionally, use of dangerous substances such as tobacco and alcohol are particularly common in ADHD and not in ASD (Milberger et al., 1997; Elkins, McGue & Iacono, 2007). | High ADHD = higher sensitivity to information depletion, due to devaluation of incoming information when accounting for cost of waiting.<br>High ASD = less sensitive to information depletion and tendency to give value even with longer waiting |

| | | | | |
|---|---|---|---|---|
| *5. Demand for novelty* | High | Low | The desire for novel stimuli and environments is generally higher in ADHD in comparison to typical developing population (Lynn et al., 2005). *Boredom* is also a highly reported in ADHD (Kass, Wallace & Vodanovich, 2003) meanwhile it is less often reported in ASD (Happe & Frith, 2009). Less visual exploration of novel environments is reported in ASD (Zwaigenbaum et al., 2005) and higher tendency to avoid novel stimuli and situations (Kootz, Marinelli & Cohen (1982). | High ADHD = exploration-biased foraging. High ASD = exploitation-biased foraging. |
| *6. Repetitive motor behaviours* | | | Repetitive motor behaviours which are a diagnostic feature of ASD (DSM-5, p.50), are also common in ADHD (Martin et al., 2014) and can be classed under *fidgeting*. Unlike fidgeting in ADHD however – which is often characterised as "off-task" behaviour (Fischer & Newby, 1998) – repetitive behaviours are generally more purposeful such as ordering objects or repeatedly touching the same object (McDougle et al., 1995). | |
| *7. Not listening when spoken to* | | | A tendency in ADHD to not listen when spoken to generally reflects challenges in sustained listening where individuals find it difficult to continue following lectures, meetings and conversations (Ramsay & Rostain, 2005). In ASD however is likely reflects failure to initiate | |

| | | |
|---|---|---|
| | listening, where individuals show no response to the sound of their own names (Nadig et al., 2007). | |
| *8. Hyperresponsiveness to sensory cues* | Sensory hyperresponsiveness in ASD reflects an aversion to novel sensory cues in ASD and the opposite in ADHD. In ASD intense negative reactions (eg. tantrums) are reported in response to sensory cues (Leekam et al., 2007), and in ADHD over-responsiveness to cue-induced attention and task switching is commonly observed in research (as mentioned above and in Dunn and Bennett, 2022). | |

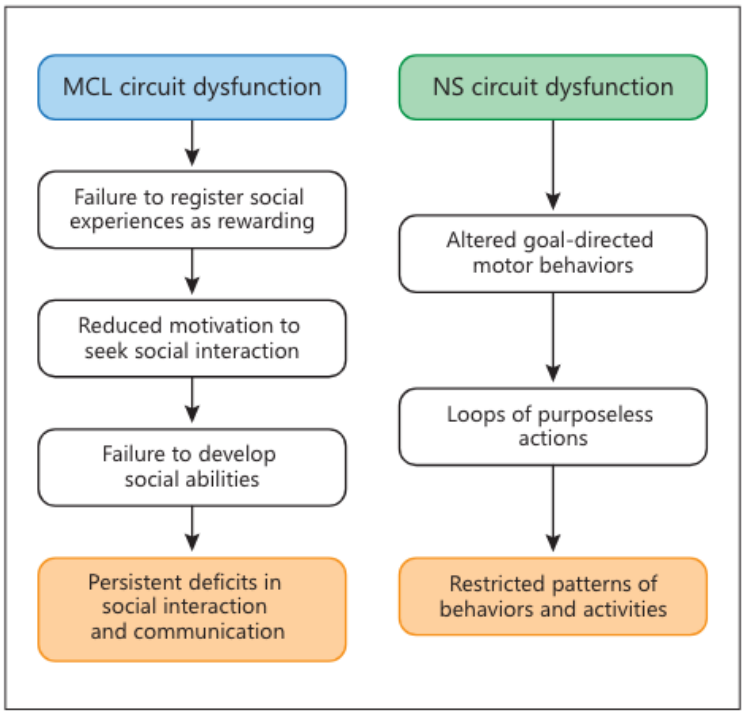



*Figure 1.10.* The proposed mechanism through which dysfunctions of the diffuse dopaminergic modulatory systems of the midbrain could lead to autistic core features adapted from Pavăl (2017).

Generally, research has consistently shown that excessive exploration and novelty seeking is a mechanism underlying ADHD (Addicott et al., 2020; Hauser et al., 2014; Hauser et al., 2016), and the noradrenaline and dopaminergic system are often implicated as valid candidates modulating this behaviour (Dubois et al., 2022). Hauser et al (2016), presented in *Figure* 1.11 for example, proposed that ADHD is characterised by impaired neural gain. Neural gain reflects the brain's ability to effectively amplify relevant signals and filter our irrelevant signals. High neural gain is dominated by the most prevalent signals that carry information. In the high gain states, prominent signals are strengthened and weak signals are attenuated, making processing less susceptible to noise. This state is most beneficial in conditions where the brain needs to avoid distractions. ADHD is described as manifesting states of low neural gain, where the system is not dominated by prevalent signals and is therefore likely to process weaker signals, potentially carrying less relevant information. There are advantages to low neural gain, for instance the ability to detect a predator in an unexpected location. Hauser et al (2016)'s model is in line with previously mentioned Cremer

et al (2023) and Aarts, van Holdstein, and Cools (2011) that it is the catecholamine transmitters – both dopamine and noradrenaline – that modulate ongoing neural activity. Dopamine function as mentioned above has strong projections to prefrontal and striatal regions (Schultz et al., 1997), and noradrenaline similarly mainly intervenes in prefrontal regions, and to a lesser extent in striatal areas (Aston-Jones & Cohen. 2005).

Furthermore, Landry (2021) introduces synaptic resistance as a mechanism which can explain specific behaviours in both autism and ADHD. They state that synaptic resistance – defined as the degree to which synaptic connections resist changes in response to dopaminergic signals – can vary between individuals, which then impacts how individuals process rewards, allocate attention, and switch between tasks. Specifically, Landry argues that individuals with low synaptic resistance would exhibit higher sensitivity to dopamine, making them more prone to distraction and impulsivity (in line with key ADHD traits), and vice versa, high synaptic resistance would exhibit lower sensitivity to dopamine, potentially contributing to rigid and repetitive behaviours (in line with key ASD traits).

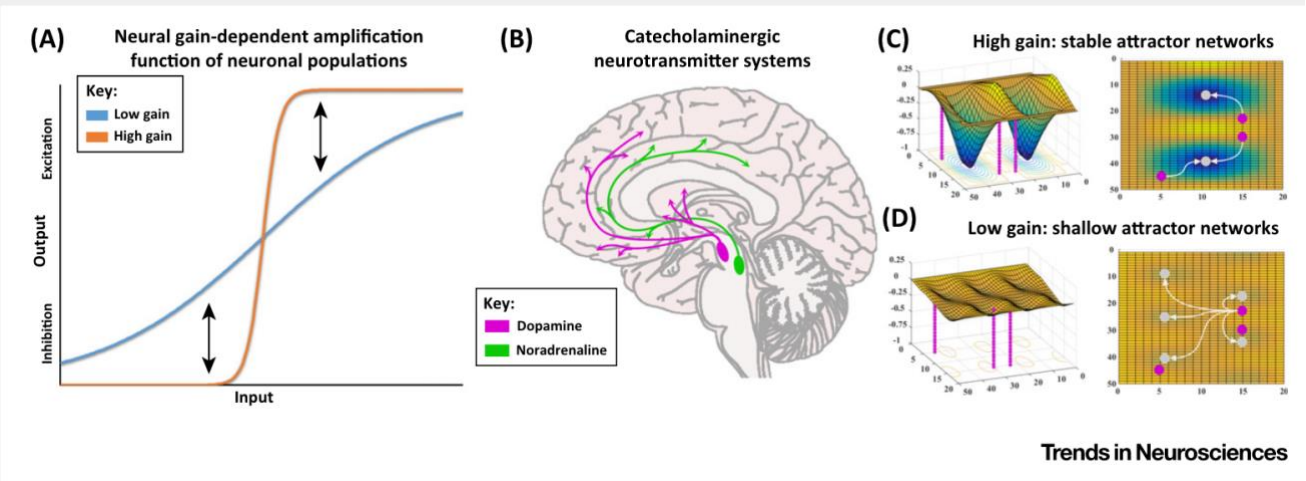



*Figure 1.11. (A)* Neural gain has an amplifying effect on neuronal signals by boosting strong inputs. *(B)* Catecholamine systems are crucial for modulating brain-wide neural gain. On a network-level, *(C)* high gain leads to stable attractor states and thus consistent outputs and behaviours, whereas *(D)* low gain causes unstable and shallow attractor states.

Based on the literature described by Landry (2021) – some of which is summarised in *Table 1.1* – dopamine modulates novelty seeking, which is supported by evidence that shows novel stimuli excite dopamine neurones across humans, non-human primates, and animals. In contrast, ADHD is a condition characterised by low levels of dopamine (or low synaptic

resistance or low neural gain), resulting in behaviours such as exploration and novelty seeking in order to stimulate dopaminergic response from a hypoactive system. ASD therefore, is characterised by higher levels of dopamine (or high synaptic resistance or high neural gain), which result in more stable but repetitive behavioural patterns.

There are several models that have been identified, building on different elements of the dopaminergic system, however the mechanisms are multifaceted and context-dependent. Whether autism and ADHD are purely a condition of dopaminergic dysfunction is unlikely, however in both these cases pharmacological interventions often involve dopaminergic treatments. Specifically, antidopaminergic treatments in autism in order to overcome stereotypic behaviours (Posey and McDouggle, 2000) and sympathomimetics in ADHD which stimulate the release of dopamine (Markowitz, Straughn & Patrick, 2003).

*Interim summary*:

There are two levels of theory that have been explained above. Information foraging is a broad conceptual framework that likens attentional sampling of the environment to that of animals foraging resources. Here the system balances between exploring new sources, potentially with unknown values and exploiting known sources likely to deplete over time. The neural gain model originally proposed by Hauser et al (2016), is an added layer, offering a neurocomputational mechanism by which changes between exploration and exploitation can be conceptualised. In particular, that low gain states lead to unstable or shallow attractor states and therefore frequent switches, closely identifying with exploratory behaviours, and high gain states lead to stable attractor and perseverative states, closely identifying with exploitive behaviours (*Figure 1.11*). The information foraging framework is a well-known account of the attentional system which informs our understanding of how the attentional system engages in sampling information and thus is used in selecting and designing the experimental paradigms in this work. However, in this thesis, our aim has been to specifically test the neural gain theory in explaining individual differences in ASD and ADHD with regards to how information is sampled in the paradigms.

### 1.1.6 Development of information seeking

In their most basic forms, visual attentional processes of alerting and orienting are present as early as infancy. Newborns, for instance, demonstrate the ability to orient to more salient-coloured stimuli instead of greyscale (Bornstein, 1976), and prefer facial patterns to most other patterns (Pascalis and Slater, 2003). This then develops rapidly within the first

year of the infant's life, alongside the development of the executive network allowing them suppression of competing information (Amso & Scerif 2015 for review). Exogenously motivated orienting therefore, emerges early and remains stable throughout childhood (Ristic and Enns, 2015). In contrast, endogenous – more intentional – orienting develops slower and well into adolescents (Crone, 2009).

As for executive control of orienting, evidence from an antisaccade paradigms show that toddlers and young children between 8 to 38 months of age are able to exhibit executive control of saccades (Scerif et al., 2005). This top-down control of saccades continues to develop, until adult like performance at 14 years of age (Luna et al., 2004). In fact, one of the most salient characterizations of development into adolescence is the steady increase in executive control abilities (Crone, 2009). This development of the attentional system from infancy through to adolescents is also highlighted through various longitudinal structural neuroimaging studies, revealing the dramatic changes in local structure and connectivity across development (Gogtay et al., 2004; Sowell et al., 2004; Raznahan et al., 2011).

Specifically with regards to visual foraging, there is limited understanding of its development, especially within the context of information seeking. Generally, research shows that children explore more than adults, especially as it relates to more broad exploration of the environment (as opposed to systematic exploration mediated by the prefrontal cortex), even as it leads to losses (Schulz et al., 2019). In highly simplified tasks, where there was little variability in choice outcomes, 4-year-old children would still explore at extreme levels in comparison to adults. Computational models show that by age 6, children are able to adjust their exploration and exploitation, although with less efficiency than adults (Nelson et al., 2014). This tendency to explore, decreases with age, and around adolescence the pattern of exploration and decision temperature tends to stabilise (Giron et al., 2023). Although some research still show adolescents to still present heightened exploration in comparison to adults (Spear 2000; Steinberg, 2008; Lloyd et al., 2020).

The developmental trajectory of information foraging and neural gain mechanisms may differ across age groups. It is clear that basic exogenous orienting is present from infancy (Ristic & Enns, 2015), however the endogenous control systems that underpin more strategic information sampling (such as the trade-off between exploration and exploitation) continue to develop through adolescence into adulthood (Luna et al., 2004; Crone, 2009). This developmental perspective is critical for interpreting findings across age groups, as the

neural gain model (Hauser et al., 2016) may manifest differently depending on the maturity of underlying frontostriatal and dopaminergic circuits.

Recent work on patch foraging across development has demonstrated that from between the three stages of childhood, adolescence and adulthood, there is a systematic decrease in exploration choices in foraging decision-making problems (Lloyd et al., 2023; Gopnik et al., 2017; Giron et al., 2022; Wiegand et al., 2019). In youth, a bias to explore when faced with explore/exploit choices can support the individual to learn the structure of their environment, preparing them to effectively exploit rewards later in adulthood (Lloyd et al., 2023). It is important to note that this developmental transition may be disrupted by psychopathology, where there are biases in explore/exploit choices in mental health problems. In patch-foraging tasks, adults tend to over-exploit (staying too long in one patch) and children over-explore (leaving too early), whereas adolescents display the most optimal balance (Gil-Gómez de Liaño et al., 2022; Gopnik et al., 2017). This suggests that the adolescent period represents a unique developmental window where exploration is calibrated to support learning and environmental mastery, consistent with models describing adolescence as an adaptive period of heightened experience-seeking necessary for the specialisation of brain systems supporting cognitive control (Lloyd et al., 2021; Lloyd et al., 2022). Compared to adults, adolescents also exhibit improved decision-making in *n*-armed bandit tasks, which is driven by adolescents' optimal exploratory tendencies (Eckstein et al., 2022).

This developmental perspective is particularly relevant when considering the neural gain model (Hauser et al., 2016) and its implications for ASD and ADHD. The catecholaminergic systems undergo significant reorganisation during adolescence (Luna et al., 2015; Crone, 2009), and therefore it is possible that the expression of low-gain and high-gain states may differ between adolescent and adult populations. Moreover, the neural circuits supporting information foraging and exploration-exploitation decision-making continue to refine through adolescence, with prefrontal and striatal regions showing protracted maturation into early adulthood (Luna et al., 2004; Lloyd et al., 2023; Gil-Gómez de Liaño et al., 2022; Gopnik et al., 2017).

#### *1.1.6.1 Adolescents*

It is understood that the first few years of life relate to a sensitive but rapidly developing period of learning and brain maturation (Shonkoff and Garner, 2012) and research

from this early period of development has contributed significantly to global practices and policies (Black and Hurley, 2014). It is established that the first 18 months of life, infants experience an over-production of synaptic connections (Huttenlocher, 1990), however a similar stage of synaptic over-production occurs during early adolescents (Giedd, 2008). Adolescence represents a critical neurodevelopmental period, marked by rapid biological, cognitive and social changes (Dahl et al., 2018). This period also coincides with progression from primary school into secondary school, where academic performance becomes more relevant. As described in the previous section, it is during this time that various cognitive functions, particularly those relating to attention begin performing closer to adult-like levels.

Crucially, it is during adolescence that core attentional and executive functions reach adult-like levels of proficiency, though the underlying mechanisms are still developing (Luna et al., 2004; Crone, 2009). While more basic processes like alerting and exogenous (bottom-up) orienting are present early in life and remain relatively stable, more complex endogenous (top-down) control – mediated by the prefrontal cortex – shows a protracted development well into the early adulthood (Ristic & Enns, 2015; Crone, 2009). For instance, performance on antisaccade tasks, which require inhibiting a reflexive glance towards a cue (a measure of executive control over orienting), improves steadily throughout childhood and only reaches full maturity around age 14 (Luna et al., 2004). This maturation is supported by longitudinal neuroimaging studies showing dramatic changes in cortical thickness, white matter connectivity, and functional network organization throughout the adolescent brain (Gogtay et al., 2004; Sowell, Thompson, & Toga, 2004).

In the United Kingdom, adolescents are technically defined to be between the ages of 10 and 19 (WHO, n.d.), however this is a large time window encompassing various stages of development, both psychologically, biologically, and academically. Research may split participants into younger adolescents (usually 11-14) and older adolescents (15-18) (for example, in the research by Dubois et al., 2022), however for the current work, we focus on the middle-adolescent period (ages 13-17) as this age reflects entering into secondary school and is the appropriate age for participants to be able to complete self-report cognitive/trait measures.

## 1.2 Experimental approach

To effectively test whether the neural gain model, operating within the broader information foraging framework, can explain attentional difference between ASD and ADHD, a multi-method approach is essential. The following paradigms are suggested for their ability to capture dynamic processes of engagement, disengagement, and ability to simulate information depletion of information sources. In all our proposed paradigms, information depletion is simulated by repeating stimuli with the understanding that at the early presentations the stimuli is treated as novel information, and processed as "information-rich" sources, however when the same stimuli are repeated a number of times, it progressively reduces the richness of the source and is then treated as familiar information. We expect that repetitions of the same stimuli elicit slow progressive changes in information affordances that can be tracked over time. We are also proposing the use of two separate neural and behavioural measures which can obtain a more comprehensive picture of information foraging.

It is important to note that while ASD and ADHD may present as opposing ends on certain attentional dimensions (eg., variability in goal allocation) (Landry, 2021) this does not preclude high rates of co-occurrence. Both conditions are heterogeneous and multidimensional, and comorbidity can arise from shared features (eg., executive dysfunction, sensory sensitivities) even when attentional profiles differ (Landry, 2021; Becker & Fogleman, 2020). The neural gain framework should therefore be understood as describing a specific dimension of attentional functioning rather than a comprehensive model of either condition. Comorbidity rates between ASD and ADHD are estimated between 14% and 75% in some reports (Becker & Fogleman, 2020; Simonoff et al., 2008). However, the "opposing ends" theory applies specifically to information foraging and attentional mechanisms, not to the entire clinical phenotype.

### 1.2.1 EEG and neural engagement

The paradigm adapted from Piccardi et al. (2021) is a paradigm in which the trade-off between sustained engagement with a depleting information source (indexed by frontal theta power) and the orienting response to a novel, salient distractor (indexed by the P1 component of the visual evoked potential) is measured. This design directly operationalizes the core foraging dilemma: to continue exploiting a current source or to explore a new one. Theta oscillations (4-8 Hz) are a robust neural correlate of cognitive engagement, information

encoding, and active learning (Begus & Bonawitz, 2020; Klimesch, 1999), making them an ideal metric for tracking information sampling over time. This paradigm allows for the testing of neural gain hypotheses (Hauser et al., 2016) by examining how the theta/P1 trade-off is modulated by individual differences in ASD and ADHD traits. Moreover, this paradigm operationalises the core foraging trade-off discussed in section **1.1.3** such that more exploitation would be associated with sustained engagement with ongoing stimulation (measured by theta response), and more exploration associated with less stable engagement with ongoing stimulation, in favour of incoming stimulation from the checkerboard presentations (measured by P1 peak amplitude and relationship between P1 amplitude and theta band power).

### 1.2.2 Eye-tracking and attentional disengagement

The gap-overlap paradigm (Saslow, 1967) is a classic tool for measuring the cost of disengaging attention from a central stimulus to orient to a peripheral one. To adapt it for foraging research, the informational value of the central stimulus can be manipulated through repetition, simulating a transition from a novel, information-rich "patch" to a familiar, depleted one over the repetitions. The key prediction is that disengagement cost will be highest when leaving a novel stimulus and will decrease as the central stimulus becomes familiar. This paradigm provides a direct behavioural measure of the disengagement process that is central to foraging theories and can reveal whether individuals with ASD and ADHD differ in their sensitivity to information depletion when deciding to switch attention (Landry, 2021; Fischer et al., 2014).

A key selection criteria for these suggested paradigms is their relative passive nature; they measure neural and ocular responses without requiring any additional motor responses, taxing executive system, or being tested on memory. This is a deliberate choice to avoid common confounds in cognitive research within neurodiverse populations. Many traditional tasks that rely heavily on motor responses or explicit memory recall can disproportionately disadvantage individuals with ASD or ADHD, not necessarily due to cognitive challenges but due to co-occurring challenges with motor coordination, processing speed, difficulty understanding instructions, or working memory (Bishop, 2019; Karalunas at al., 2014). By measuring changes in neural signal and eye movements directly, these paradigms provide a more direct measure into core mechanisms of attentional engagement and disengagement, minimising the influence of other factors that could obscure performance on these tasks.

## 1.3 Research questions and aims of thesis

Building upon the theoretical foundations and experimental approached outlined above, this thesis aims to address several critical research questions that remain unresolved in the literature. Firstly, to test the validity of the neural gain hypothesis in neurodiverse populations as outlined by Hauser et al (2016) and later supported by Piccardi & Gliga (2022). We aim to investigate this model and whether it serves as a valid framework to distinguish between the attentional profiles of ASD and ADHD. Specifically, whether 'low-gain' and 'high-gain' states are strongly associated with varying ASD and ADHD traits.

Moreover, we aim to understand whether ASD and ADHD may be presenting on opposing ends of an attentional spectrum, as suggested by some dopaminergic accounts (Hills, 2006; Landry, 2021). Within our experimental paradigms we expect that behaviours and neural measures in high ASD traits and low ADHD traits would present similarly, and vice versa, that low ASD traits and high ADHD traits would present similarly. Individuals in the former group will show more stable engagement and in the latter group would show more erratic engagement behaviours.

An important goal of this thesis is to adopt a multidimensional traits approach with which we hope to capture richer individual differences data, including natural covariation between the different traits. This will allow us to better represent neurodiverse samples and determine whether a specific neural or behavioural pattern is better associated with ASD traits versus ADHD traits in clinical groups and in the general population. Both ASD and ADHD are marked by their heterogeneity, a dimensional approach allows us to maintain full range of variability.

# CHAPTER 2:

## 2. Information trade-offs in adolescents with Autism and ADHD traits.

### Goal of chapter

*This experimental chapter provides a framework to test the theoretical framework proposed in the previous chapter using an adapted EEG paradigm. Here I re-visit some of the aforementioned literature, in addition to presenting more specific EEG related literature to support the design of this study.*

*The experimental paradigm is based on a previous study by Piccardi et al., (2020) where change over-time in neural signals are measured in response to dynamic repeating and novel stimuli. The neural signals targeted here are frontal Theta band oscillations and P1 peak amplitude of the ERP component. Through repeated stimulus exposure, this paradigm simulates information depletion from a video source where each additional viewing of the video contributes progressively to reduce the amount of information available to be acquired/gathered.*

*In this chapter I explore the results of this paradigm in a typically developing adolescent sample as well as neurodiverse sample including both Autistic and ADHD diagnosed young people. I report that our results demonstrate support for the neural gain model proposed by Hauser (2016) and supported later by Piccardi and Gliga (2022), however this is only present along the ADHD traits scale and does not extend to ASD traits. Implications for this trend in the data is discussed along with findings that lends to alternative baseline engagement in relation to ASD traits.*

# 2.1 Introduction

## 2.1.1 Background literature

Attention is one of the earliest developing processes (Amso & Scerif, 2015) that is essential to learning and the development of other cognitive domains such as memory, language, motor control, and executive function (Cuevas & Bell, 2013; Posner, 2011; Song, 2019). An effective attentional system allows individuals to navigate surroundings, making sure relevant information are processed and irrelevant information is ignored. Foraging theory, which is one of the models of attention suggests that seeking information from your environment and seeking resources such as food requires a similar mode of action, such that we exploit our current source of information until the information depletes and therefore the effort needed to continue extracting information outweighs the relative value. It is at this point where a shift of attention might occur to seek information elsewhere in our environment (Stephens, 1987; Calhoun & Hayden, 2015; Hills et al., 2015).

The visual orienting system, which is critical in this account of attention, is under the influence of dopaminergic system, which is implicated in reward and motivation (Schultz, Dayan & Montague, 1997; Wise, 2004). The relationship between visual orienting system and dopaminergic system can be explained by the brain prioritising information by assigning it a reward value (Snyder, Blank, & Marsolek, 2008). There is also evidence that this mechanism is susceptible to individual variation, for instance individuals with autism are traditionally thought to have higher levels of tonic dopamine, which disrupts reward and motivation neurochemical systems, leading to less exploration and less novelty seeking (Hills, 2006). Hills (2006) further describes how various conditions associated with atypical attentional systems, such as Schizophrenia, Obsessive-compulsive disorder, Parkinsons, Autism, and Attention deficit hyper-activity disorder, map onto differences in dopaminergic systems as presented in *Figure 1.10.*

It has also been proposed that seeking of information relies on the same neural system as seeking of rewards (Bromberg-Martin & Hikosaka 2009; Bromberg-Martin & Hikosaka 2011). Here, *information* is suggested to have inherent value, even in cases where information that is acquired in an experimental paradigm is non-instrumental (i.e. that it does not inform later decisions) (Bromberg-Martin & Hikosaka 2009). This aligns with foraging accounts of attention, where instead of sampling the environment for rewarding resources

(essential for survival), humans sample the environment for rewarding information (essential for reducing uncertainty) (Gottleib, 2013; Manohar & Husain, 2013; Hills et al., 2015).

Expanding on from Hills (2006)'s account of dopamine, recent theoretical accounts of autism spectrum disorders (ASD) and attention deficit hyperactivity disorders (ADHD) points towards the dopaminergic system playing a key role in the expression of many traits commonly seen in ASD and ADHD (Hauser et al., 2016; Paval, 2017; Landry, 2021). Pavăl (2017) for example explains how social deficits and restricted behaviours can be traced back to dysfunction of the dopaminergic modulatory systems (particularly in the midbrain region). On the other hand, Hauser et al., (2016) postulates that ADHD is characterised by 'low gain' states indicating either low Dopamine release or low dopamine receptors in the striatum. This presents as unstable goal selection and unstable allocation of attentional resources. This view is also supported by Piccardi and Gliga (2022) where they suggest low visual sensory seeking (a trait highly prevalent in children and adults with ADHD; Little et al., 2018; Bijlenga et al., 2017) also maps onto low gain states described by Hauser et al (2016). The dopaminergic model explained by Pavăl (2017) is presented in *Figure 1.10* and the account by Hauser et al., (2016) in *Figure 1.11*.

These theoretical accounts are supported by animal models where alterations to the dopaminergic system presents behaviours that are stereotypically related to either Autism or ADHD; such as repetitive behaviours in Autism and impulsivity in ADHD (Banerjee, Bhat, & Riordan., 2014; Russell., 2011; Dougnon & Matsui., 2022). Studies that consolidate the dopaminergic systems involvement in both Autism and ADHD are few. One report that attempts to summarise research relating to the involvement of the dopaminergic system and its role in ASD and ADHD is Landry (2021). They argue that behavioural patterns related to the attentional system presented in ASD and ADHD tend to present at opposite ends of the same spectrum and that some of this can be traced back to the dopaminergic system. Some examples of this in the report include Attention-switching – frequent in ADHD, infrequent in ASD; task-switching – frequent in ADHD, infrequent in ASD; delay aversion – high in ADHD and lower in ASD. This is in line with other well documented areas such as visual sensory seeking, where ASD and ADHD present in opposing fashion such that novelty preference is higher in ADHD and (Lynn et al., 2005) and lower in ASD (Minshew & Hobson., 2008).

It is also understood that co-occurrence of ADHD and ASD is anywhere between 14% to 75% in some reports (Becker & Fogleman., 2020), however, few studies compare groups with Autism and ADHD or include groups with co-occurring Autism and ADHD. Although overlapping traits are not uncommon (such as hyperresponsiveness to sensory cues (Leekam et al., 2007)), the dopaminergic accounts propose a cognitive attentional system that should theoretically be performing in opposing fashion between ADHD and ASD groups. It also raises questions about the behavioural profile that may be present in individuals who have a dual diagnosis, or who have varying levels of ASD traits and ADHD traits within their diagnosis. We and others (Klin et al., 2002; Xavier & Cohen, 2020) suggest that a multidimensional approach that measures ASD traits and ADHD traits in all participants will provide us an understanding of where traits associated with the two neurodivergent groups converge or diverge with respect to their attentional systems.

Adolescence represents a critical neurodevelopmental period, marked by rapid biological, cognitive and social changes (Dahl et al., 2018). This period also coincides with progression from primary school into secondary school, where academic performance becomes more relevant and it is during this time that various cognitive functions, particularly those relating to attentional control begin performing at adult-like levels (Crone, 2009; Luna et al., 2004).

### 2.1.2 The Proposed Study

To test the theoretical accounts of opposing attentional systems represented in seeking of information, we use an adapted paradigm from Piccardi et al (2021). This paradigm captures the trade-off between allocating attention resources to ongoing stimulation – a repeating video representing a depleting source of information over time and flashes of a high-salience distractor image. Using electroencephalogram (EEG), a video clip from an animated cartoon was repeated while being consistently interrupted by a black-and-white checkerboard image, presented for 100ms. Frontal Theta activity (4-6Hz) to the video was used as a measure of information processing as the video is repeated. Visually evoked potentials (VEP) evoked by the presentation of checkerboard distractor represent early visual sensory processing. The selection of these two neural signals was theoretically and methodologically well suited for the aims of the Piccardi et al (2021) study, as well as the aims of the current research.

Frontal theta band is defined as oscillations in frequency range of 4hz to 8hz in adults and 4hz to 6hz in infants. Activity within this range has been shown to associate with various cognitive processes including mental arithmetic (Ishii et al., 2014), working memory (Kawasaki & Yamaguchi., 2013), and learning (Clarke, Roberts, & Ranganath., 2018) (for review: Klimesch, 1999; Karakas, 2020). Similarly, in infants, processes such as exploration, information seeking, and are attributed to this range (Begus, Southgate, & Gliga., 2015, 2016; Piccardi et al, 2021). Begus, Southgate and Gliga (2015) for example show a predictive association between theta-band oscillations and learning, such that modulations of frontal theta-band oscillations were found to predict infants' encoding of objects' features. A recent review of the role of theta oscillations in learning in infants and in adults by Begus and Bonawitz (2020) argues that this frequency band is uniquely suited to investigate mechanisms underlying active learning and engagement. In essence, it is believed that theta activity in the frontal regions reflect pre-frontal hippocampal information-processing and seeking loops (Klimesch, 1999; Begus & Bonawitz, 2020). In the Piccardi et al study, as well as our proposed study, activity within the theta-band range is used as a neural measure indexing information seeking and engagement.

Conversely, the P1 component of the VEP was chosen as a measure of the participant's sensory processing of the high-salience distractor. The P1 is an early positive deflection in the event related potential (ERP) that occurs approximately 100ms following stimulus presentation. The P1 component is sensitive to faces (Itier & Taylor, 2004), and more importantly, is known to be affected by the participant's attention (processing of) the stimulus presented and the task demands (Mangun, 1995; Taylor, 2002). This component is reliably generated in and around the visual cortex (Di Russo et al., 2001; Luck 2014). In the context of this paradigm, a larger P1 response to the checkerboard can be interpreted as a stronger orienting response, reflecting a shift of engagement from the repeated video towards the brief checkerboard presentation.

Piccardi et al (2021) results revealed three major findings: Theta oscillatory power changing with the repetition such that there is an initial increase (from time-bin 1 to time-bin 2), followed by decrease in power (from time-bin 2 to time-bin 3), and then stabilising (time-bin 3 to time-bin 4). This was proposed to reflect the progressive encoding of information, followed by depletion of information and thus encoding. The power of theta oscillatory amplitude was negatively correlated with the P1 peak amplitude of the VEP. In other words when theta power in the frontal regions was high, P1 peak amplitude was low and vice versa.

This was taken to mean that there was a trade-off in attention/information sampling between the video and the checkerboard. As the information from the video is depleted (theta decreasing), attention is switched to the checkers, resulting in a larger peak P1 response. Finally, there was individual variation in this trade-off. Using scores on the Infant-Toddler sensory profile (ITSP) (Dunn, 2002) infants were classified as high or low visual seekers. Lower sensory seeking in infants relate to later lower Deprivation-type curiosity traits (Piccardi & Gliga, 2022). Deprivation-type curiosity measures persistence on tasks or obtaining information and is inversely correlated with ADHD traits such as inattention and hyperactivity (Pietrowski et al, 2014). Low seekers had a shallower increase in P1 as theta decreased, corresponding to a low gain profile, characteristic of ADHD.

The relationship between these two signals, rather than their absolute values alone, is central to the hypotheses. The trade-off between processing of ongoing information (repeated video) and processing of incoming information (checkerboard) acts as an index of the efficiency of attentional resource allocation over time. Importantly, we expect that this trade-off can accurately map onto the neural gain model as described by Hauser et al (2016) and be susceptible to individual differences. The gain model suggests information processing is a matter of navigating and balancing competing signals according to situational and individual demands. Higher gain individuals remain engaged with information (the video) for longer but then switch sharply as it is farther depleted. On the other hand, low gain individuals disengage with ongoing information earlier and in an unstable fashion (potentially shifting back and forth between the sources of information). This then presents as a shallower slop (F*igure 2.1*).

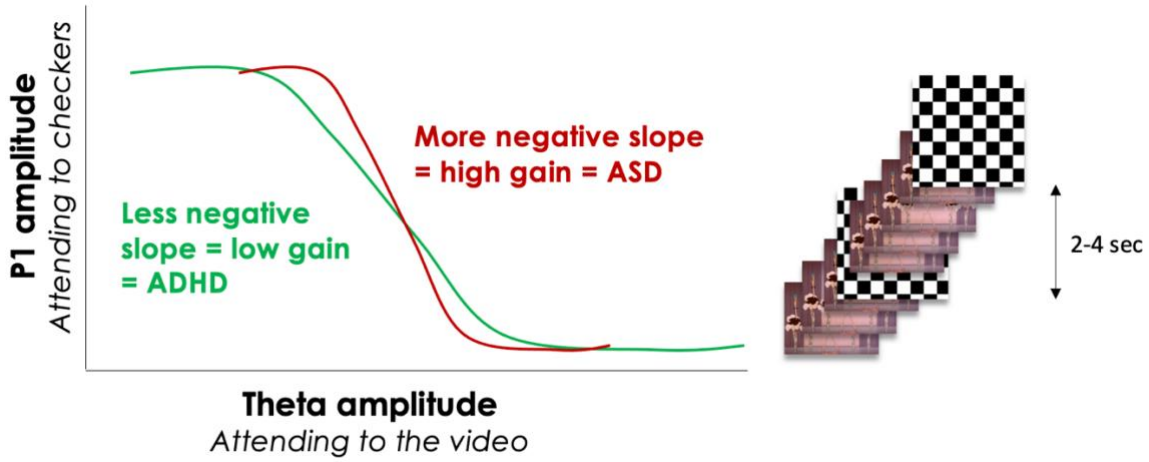
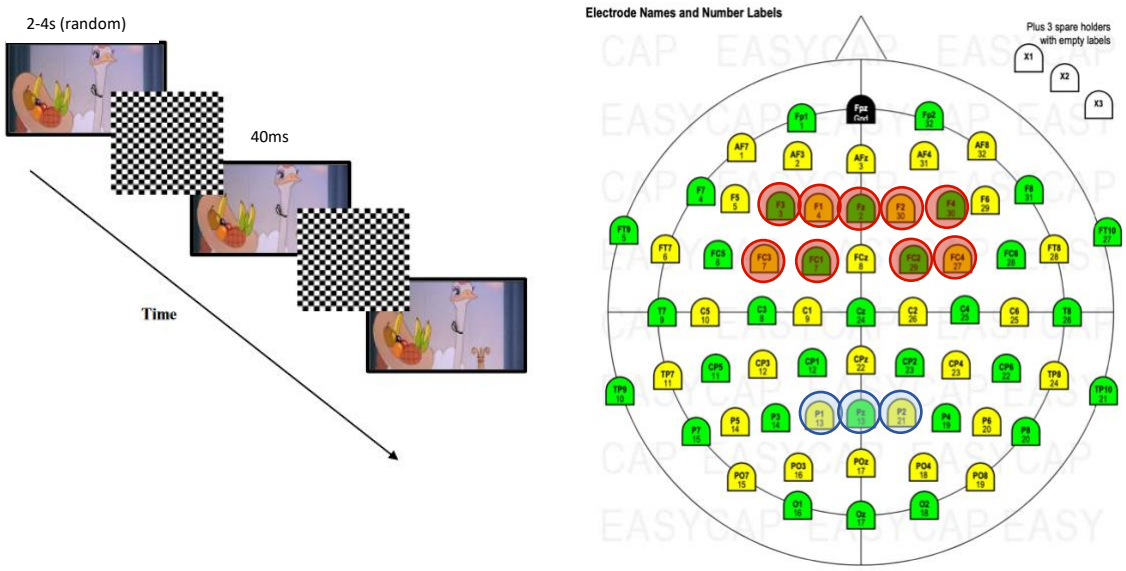


*Figure 2.1*. Adapted from Piccardi and Gliga (2022). Here the likelihood of disengaging attention from ongoing stimulation in order to engage with new stimulation depends on the former. Importantly, the steepness of this attentional switch from ongoing source of information to incoming source of information, is expected to be modulated by high-gain and low-gain states which we propose are mapped onto ASD and ADHD respectively.

We aim to use an adapted version of the Piccardi et al (2021) paradigm, using the same stimuli and measurements. While the previous study used proxies of ADHD traits (using ITSP to measure sensory seeking) and did not measure autism traits, we employ a multi-dimensional traits approach and include clinical samples in order to appropriately test whether neural markers of information seeking differ according to individual differences in ADHD and ASD traits.

In this adapted version we use a longer clip (1 minute 30 second clip as opposed to 40 seconds in Piccardi et al, 2021) and present the checkerboard on screen for 40ms instead of 100ms. These adjustments are intended to increase trial numbers for analysis thus increasing EEG signal/noise as well as cater to the characteristics of older participants. We repeat the main clip 11 times and intersperse three additional clips from different parts of the movie (each one of them different) within the sequence. The experimental sequence and the channels of interest are presented in *Figure 2.2.* Novel videos in the sequence provides additional insight into the function of theta power and its relationship with information gain and engagement. If an increase in theta power serves as an index of information uptake (Begus, Southgate, & Gliga., 2015, 2016; Piccardi et al, 2021; Piccardi & Gliga, 2022), we would expect to see decline in theta power as the main clip is repeated, representing familiarity with the video and information depletion. Following this, a novel video should result in recovery of theta power, indicating the necessity to encode new information. In total there are 14 video clips. The previous study split data into 4-time bins, however as our video clips are longer than the previous study, we analyse data in each video separately. Within each video there are an average 30 instances of checkerboard.




*(a)* *(b)*

*Figure 2.2.* (a) Represents the sequence of events occurring in the experimental paradigm. Here a brief flash of checkerboard (40ms) – shorter than used by Piccardi et al., (2021) (100ms) – is presented every 2-4 seconds at random while the videos are being watched by the participants. Each video is 1 minute 30 seconds long (b) Representation of the channels of interest, the nine Red coloured channels in the frontal-medial region are selected for frequency analysis and the three Blue coloured channels in the parietal region are selected for VEP/ERP analysis.

Hypotheses

1. We predict that frontal theta oscillatory power to the repeated video will decrease with video repetition, due to information depletion from the video.

2. We expect the P1 amplitude to be inversely related to frontal theta amplitude.

3. We predict that the degree of modulation between theta and P1 (i.e., the trade-off between engagement with the video and disengagement with the video) will differ according to both ADHD and ASD traits. Specifically, we expect that higher ADHD traits should be associated with a shallower theta-P1 modulation slope (less negative, or closer to 0), reflecting a low-gain, more erratic attentional state. ASD traits on the other hand, should be associated with a steeper modulation slope (more negative), reflecting a high-gain, more stable trade-off, in line with hypothesis of opposing ends of an attentional continuum.

4. If theta indeed captures information depletion, we predict to see a recovery in theta band activity for the novel videos in the sequence, compared to the repeated videos.

# 2.2 Methods

## 2.2.1 Participants

Forty participants took part in the EEG study (13 females, M age = 14.1, SD age = 1.05). One participant's data was not included in the final analysis due to excessive data loss from the EEG recording and therefore the total number of participants included in this report is thirty-nine (13 females, M age = 14, SD age = 1). Minimum number of participants required was determined through an a priori power analysis based on Piccardi et al (2021). In accordance with Cohen (1988) a medium to large effect size is 0.5 and, with an estimated power of 0.9, we expect a sample size of 48 to detect an effect of video repetition (through a repeated measures between factors ANOVA).

The first sample of adolescents (N=20) were recruited from posting on local SEND parent and online support groups. We advertised to recruit young people between the ages of 13-16 who were already diagnosed with either Autism or ADHD (or both) or were on pathway to receiving diagnosis for either Autism or ADHD (or both). Fliers encouraging research participation were also placed at the local library and a small selection of participants were recruited through this. The second sample of adolescents (N=20) were recruited from the volunteer database (University of East Anglia), where parents of young people (between the ages of 13-16) received an email requesting participation in the study. There was no exclusion of participants with ASD/ADHD or on pathway to receiving a diagnosis. In the final sample we report 17 participants had received a diagnosis of ASD, 1 participant had a diagnosis of ADHD, 3 participants with a diagnosis of both ASD and ADHD, and 4 participants on a pathway to receiving a diagnosis of either ASD or ADHD.

All participants resided in Norfolk, and the procedure is approved by the Research Ethical Committee of the University of East Anglia, Norfolk, United Kingdom.

It is important to note that our recruitment strategy aimed to include a balanced clinical sample with both ASD and ADHD diagnoses. However, due to recruitment challenges within the available timeframe, the final sample was asymmetrical: we successfully recruited a substantial number of participants with ASD diagnoses (n=17) but only a limited number with ADHD-only diagnoses (n=1). We utilise data from this sample of participants in the current chapter and in Chapter 4.

## 2.2.2 Design

Participants view a sequence of 14 video clips (11 of them repetition of the same video, intermixed with 3 novel clips) from *Fantasia*. Each clip is 1.5 minutes long and interrupted by 40ms checkerboard overlays at random intervals (2– 4 s). To capture engagement with the videos, we extract absolute power of Theta band activity from the frontal medial region of the scalp, and to capture engagement with the checkerboard (i.e. disengagement with the video), we extract P1 peak amplitude component from the ERP. The order of videos presented to the participants was pre-selected. To keep participant's interest, they were asked content-related questions after 6 randomly selected clips (Appendix A).

This experimental design employed a repeated-measures design and mainly uses individual differences measures (ASD and ADHD) on continuous scales. The within-subject independent variable was video repetition (with 10 levels for repeated video, as one video was excluded due to missing data). ASD traits (continuous, z-scored ADOS-2 total score) and ADHD traits (continuous, z-scored Conners-4 ADHD-index composite score of self and parental report) were entered as covariates in the models.

Additionally, we analysed individual differences as between-subject independent variables in some analysis by implementing a median split on ASD and ADHD trait scores. Although we could have split participants into ASD diagnosed and non-ASD diagnosed, we instead took advantage of our dimensional approach in which we obtained ASD and ADHD trait scores from every participant. We then split them according to their actual lab-measured traits, not diagnosis status. This preserves the continuous nature of the data while permitting supplementary group-level analysis based on what traits participants present regardless of diagnosis status, including importantly the groupings for ADHD for which we did not obtain a sufficient diagnosed group.

There were three dependent variables in this study design, the first one being the absolute power of theta band activity recorded from the fronto-medial region of the scalp, time-locked to video segments free from checkerboard presentations. The second dependent variable was the P1 peak amplitude, recorded from the parietal region of the scalp, occurring between 70-130ms post checkerboard presentation. The third variable was derived from the slope of the correlation between theta power and P1 peak amplitude across the 10 repetitions for each participant. This represented the trade-off between engagement with the video and disengagement with the video.

### 2.2.3 Materials

*2.2.3.1 Measures of individual differences*

**Conners Rating Scales – Forth Edition (Conners, 2023)** was used as a measure of ADHD traits which included self-report and parental report surveys. This is a norm-referenced measure of ADHD commonly used to screen for ADHD in children and young people ages 8-18 to aid diagnosis and assess behaviours that co-occur with ADHD. The total number of items in the self-report is 115 and 114 for the parental-report. The items are rated on a 4-point scale from 0 to 3, where 0 represents *not true at all*, 1 represents *just a little true*, 2 represents *pretty much true*, and 3 represents *completely true*. The items from Conners-4 can also be combined into subscales of inattention, hyperactivity, impulsivity, emotional dysregulation, depressed mood, and anxious thoughts.

Total scoring for each participant is generated automatically through MHS Access – an online platform hosting the online versions of Conners-4 survey. Several scores are generated through the platform including scores related to inattention/executive dysfunction, hyperactivity, impulsivity, functional outcome scales, DSM specific symptom scales, and the Conners 4 – ADHD index. The ADHD index score in the Conners rating scale scores items that distinguish between general population and ADHD cases. There is strong evidence for reliability, and inter-rater reliability. Importantly, unlike other subscale scorings from the Conner's scale, there are no meaningful variance or group differences in relation to gender, race/ethnicity, county of residence, or parental education level. Due to the relatively smaller sample of participants, and the focus of this research being specifically related to ADHD symptomology, the ADHD-index from the Conners-4 survey is an appropriate measurement to use as the individual differences measure in ADHD traits. The lowest score on the ADHD index is 0 and the highest score is 40, scores in our sample in *Table 2.1* shows a varied distribution in the scores with the lowest score being 1.5 and the highest being 38.

The ADHD index scores for self-report and for parental report are extracted, after which these scores are averaged and converted to z-scores in order to normalise distribution of individual differences.

**The Autism Diagnostic Observation Schedule (ADOS-2)** was used as a measure of ASD traits performed by trained researcher (myself) alongside an assistant note taker. ADOS-2 is a reliable and valid diagnostic assessment designed to explore social responses and behaviours in naturalistic conversation-based settings. In this study, module 4 was used as it is aimed at young people and adults that can use fluent speech. Each module in the ADOS-2

is scored with a diagnostic algorithm comprised of communication, social affective behaviour, and repetitive behaviours. In order to represent the full ASD phenotype for all participants, we used the composite score across all categories of measurements in the module 4 of the ADOS, instead of the diagnostic algorithm score which comprises of a selection of scores.

Similar to the ADHD traits, this total composite score is z-scored in order to normalise the distribution of individual differences.

Additionally, the **Behavioural Rating Inventory of Executive Function (BRIEF)** was used as a measure of Executive Function. The BRIEF is a behavioural rating measure of everyday executive skills of young people, with items specifically designed to assess everyday environments, including home and school (Gioia et al., 2000). Both the parent form and the self-report form was used from the BRIEF and both surveys comprise of 86 questions, rated on a 3-point scale 0 represents *never*, 1 represents *sometimes*, and 3 represents *often*. The BRIEF is composed of two major index scales as well as a total score from them, which are the Behavioural Regulation Index (BRI) and the Metacognition Index (MI). The BRI comprises of items relating to inhibition, shifting, and other control related subscales, whereas the MI comprises of items relating to initiative, working memory, and other planning related subscales. It is worth noting that Gioia et al. (2000) report that children with ADHD had significantly higher scores on all subscales of the BRIEF and Gilotty et al. (2002) report elevated scores in several subscales for children with autism.

*2.2.3.2 Stimuli*

Experimental stimuli in the EEG session consisted of clips from the Disney movie Fantasia by Walt Disney. The video clip resolution is 1280 x 720 px. There are repeated videos and novel videos in the sequence of videos, all of which are 1 minute and 30 seconds long. The repeated video clip is repeated 11 times. In addition, three novel clips taken from the same movie at different timepoints are used. These depict sections of the movie the participants have not yet viewed and are the same length as the repeated video. The three novel videos are presented once at set positions in the sequence and do not appear again. The total number of videos participants will view are 14 videos; 11 of which are one video repeated, and three novel videos. The presentation sequence is as follows (where video B, C, D represent novel videos):

| Sequence | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Video | A | A | A | A | B | A | A | C | A | A | A | A | D | A |

Throughout the presentations, the video is interrupted by brief overlays of a black-and-white checkerboard image. This image is 2251 x 2248 px and overlays the entire video for approximately 40ms at random intervals between 2 to 4 seconds during the repeated and the novel videos alike.

For the purpose of keeping participants engaged and attentive within the experiment, they were asked questions about video content at the end of some of the video presentations. The questions required button press of YES/NO or TRUE/FALSE answers and were related to the content of the videos they had just finished watching. Examples of questions include: "Did all the hippos have four teeth each?", or "Was the colour of the curtains on stage pink?" Out of 14 videos, questions appear at the end of 6 random pre-selected videos. These are presented in *Appendix A.*

*2.2.3.3 Apparatus*

We used the Brain Vision actiCAP system with 64 active electrodes (Brain Products GMbH). The electrodes are set in an elastic mesh cap that fits around the whole scalp. One electrode is placed under the left eye to monitor eye movements and blinks. The EEG channels are connected through BrainAmp modular amplifiers which allows for high-resolution data acquisition.

Participants were sat approximately 70cm from a 61cm monitor (resolution 1920 x 1080 px) in a dark room with a small light source behind the participants in order to avoid sitting in complete darkness. Participants responded to questions on the screen using a keyboard that was placed on the table between them and the monitor.

### 2.2.4 EEG pre-processing

Continuous EEG signal was recorded at 500hz sample rate using FCz as a reference electrode and pre-processed using EEGLAB (Delorme and Makeig, 2004) and ERPLAB (Lopez-Calderon & Luck, 2014) in MATLAB®. First, Finite Impulse Response (FIR) is used for band-pass filtering continuous EEG data 0.1 to 40hz, and then sampling rate is changed to 250hz. Breaks in the data – where recording has started before the experiment, or where a participant takes a longer break after a video presentation resulting in large sections of noise

indicating movements, these are manually removed from continuous data before further cleaning.

Channels which are consistently noisy are identified with visual inspection and removed from the data (to be interpolated at a later step). This is followed by Independent Component Analysis (ICA) where components corresponding to eye blinks were removed.

Using Binlister function in ERPLAB, EEG triggers (sent at the onset of the videos) are used to bin data into separate video bins. Each checkerboard within these video-bins, also has an assigned EEG trigger (sent at the onset of the checkers) which are used to segment the data for frequency and ERP analysis. Following this, data are segmented as described below. Firstly, continuous data are segmented from -500 to 1000ms, where 0 represents the onset of the checkerboard, to compute VEPs time-locked to the checkerboard presentations (total segment 1.5s). A baseline correction is applied to these segments, ensuring stable reference point. Secondly, the continuous data are segmented from -1500 to 0ms relative to checkerboard trigger to be used in computing frequency analysis of Theta band. These segments are free from checkerboard presentation. This is to hinder potential influence of the VEP in the oscillatory data (total segment 1.5s). Baseline correction is not applied to these segments.

Artifacts are rejected across both sets of segmented data using moving window peak-to-peak threshold method with a voltage threshold of 100μV and time window of 200ms. Data is then visually inspected for any clear artifacts not detected with the moving window method and rejected manually. Channels that were previously removed were interpolated with the spherical interpolation function from EEGLAB. Finally, the data is re-referenced to the average of all 64 channels.

### 2.2.5 EEG analysis

#### *2.2.5.1 VEP analysis*

For VEP analysis, 3 channels with the highest activity were chosen and clustered from the parietal regions commonly associated with attentional processes and where early components of VEPs are apparent (Heitanen et al., 2008; Di Russo et al., 2001). This included channel POZ, PO3, and PO4, and participants must have minimum retention of 2 out of 3 channels non-interpolated for their data to be included.

From data segmented for VEP analysis (-500ms to 1000ms), the peak positive amplitude of P1 from is automatically extracted within a time window of 70ms to 130ms following checkerboard onset. This was determined from a sample of 4 adolescent pilots and 7 adult pilot participants, where we found that the first positive component of the ERP typically manifested within this window. There are approximately 30 instances of checkerboard in each video and therefore averaged waveform is extracted for each video for each participant. Following this, combination bins for the repeated videos are created such that bin1 is video 1 and video 2, bin2 is video 3 and video 4, and so on. This is for visualisation purposes.

*2.2.5.2 EEG frequency analysis*

For frequency analysis, nine channels were selected covering the frontal medial region. These are: Fz, F3, FC1, FC2, F4, F1, FC3, FC4, F2. These channels were selected to correspond with the channels used by Piccardi et al., 2021, and based on research using varying number of channels from this region when recording theta-band activity, i.e. Liang et al., (2021) using 4 channels from this region, and Du et a., (2023) using 12 channels from this region. Participants had to have at least 7 out of these 9 channels non-interpolated to be included in the analysis.

The data segmented for frequency analysis (-1500 to 0ms) was imported from EEGLAB and processed using Fieldtrip toolbox (Oostenveld, et al., 2011). Fast Fourier Transform (FFT) analysis is performed on the segmented data using a Hanning taper window with the frequency band set between 4hz to 30hz in steps of 1hz, generating absolute power ($uV^2$) of for each of the video bins. This is conducted across all channels for each video bin. From this, the frequency range of Theta (4hz to 8hz) is extracted for the range of channels that make up the frontal medial band. Similar to the VEP analysis, combination bins for the repeated videos are created for visualisation purposes.

### 2.2.6 Procedure

Participants parents or primary caregivers would respond to our recruitment email or to fliers posted indicating their interest in participation or receiving further information about involvement (recruitment material in *Appendix B*). They would receive an email outlining all components of the session, including relevant detailed information where it may cause discomfort for young people with sensory needs (such as gel in the hair during EEG capping). All participants received two information sheets prior to attendance, one targeted towards an

adult caregiver, and one written in more child-friendly language targeted towards the young person.

When participants arrive, they were briefed on what is expected of them in the experiment and what they will experience. This includes a summary of all the tasks they will take part in during the 2.5-to-3-hour session: an EEG study, followed by filling out ADHD and Executive function surveys on the computer, then a separate eye-tracking session (unrelated to the aims of this study), and finally the ADOS-2. The order of the sessions was sometimes changed in order to suit the participant's needs (for example, the surveys on the computer were done at two separate time points to allow for additional breaks). Participants were offered small snacks and drinks throughout the session, and breaks were offered between all parts of the session.

After briefing, both the caregiver of the participant and the participant signed separate consent sheets, agreeing to take part in all parts of the session. The participant information and consent sheet directed to the caregiver is presented in *Appendix C.1* and the participant information and consent sheet directed to the participant in *Appendix C.2* which uses more age-appropriate and easy to understand language. Following this, we set up the EEG system with the caregiver in the room and explained the steps as we performed them in accessible language. Head circumference was measured for the participant, and an appropriately sized cap was fitted onto their heads. Next, EEG electroconductive gel was carefully injected into each electrode to bridge the gap between the scalp and the electrode. This takes approximately 30 minutes, depending on hair texture and quality of EEG recording. Participants were given instructions to watch the videos presented on the screen and answer the questions when they come up by pressing the RIGHT or LEFT buttons on the keyboard. The end of each video clip could be taken as a break, in case the participant wanted (for example to stretch or have a drink), otherwise they could press SPACE to continue playing the next video clip. Caregivers were offered the option to stay with their child or to sit outside the recording room with the researchers.

The total length of time participants will be watching the video is 25 minutes, and the full EEG session including setting up and washing up afterwards is approximately 1hr and 30 minutes.

Following the EEG recording, participants and their respective caregiver were asked to sit at two computers placed next to each other on a desk and answer the questions from the

BRIEF survey and the Conners-4 survey. This was presented to them on separate computers next to each other and took approximately 10-30 minutes to complete. They were asked to answer the questions from their own perspectives and were encouraged to ask the researcher in case any questions caused confusion.

# 2.3 Results

## 2.3.1 Descriptives

*Traits measures*. Independent samples t-test shows a significant difference between ADOS-2 and Conners-4 scores between the ASD sample and the non-ASD sample ($t$ (37) = 3.37, $p$ <0.01 for ADOS-2 and $t$ (37) = 5.02, $p$ <0.001 for Conners-4) such that the ASD sample scored higher in their ADOS-2 scores and in the Conners-4 survey. This is presented in *Table 2.1.* There was a significant positive correlation between ASD z-scores and ADHD composite z-scores ($r$ (37) = .41, $p$ = .01). Normality checks using the Shapiro-Wilk test on raw ADOS scores and ADHD (self and parental combined) reveals both to be normally distributed (W (39) = .97, $p$ = .30 for ASD, and W (39) = .98, $p$ = .71 for ADHD).

*Data quality.* Additionally, data quality checks show that individuals in the non-ASD group had slightly better retention of data. Information on the number of channels interpolated and number of trials retained are present in *Table 2.2.*

*Table 2.1*

*Descriptive statistics of individual differences measures for ASD sample versus non-ASD sample*

| | ASD n = 19, Female = 6 | | | | Non-ASD n = 20, Female = 7 | | | |
|---|---|---|---|---|---|---|---|---|
| | *Mean* | *Std. Dev* | *Minimum* | *Maximum* | *Mean* | *Std. Dev* | *Minimum* | *Maximum* |
| **Age** | 14.04 | 0.97 | 13 | 16 | 14.05 | 1.15 | 13 | 17 |
| **ADOS-2** | 24.89 | 10.7 | 10 | 44 | 13.85 | 9.7 | 1 | 34 |
| **Conners-4 ADHD** | 24.37 | 7.62 | 11.5 | 38 | 12.95 | 6.56 | 1.5 | 24 |

*Table 2.2*

*Data quality and cleaning record for EEG data in ASD and non-ASD group.*

| | ASD n = 19, Female = 6 | | Non-ASD n = 20, Female = 7 | |
|---|---|---|---|---|
| | *Mean* | *Std. Dev* | *Mean* | *Std. Dev* |
| **Average number of channels removed** | 3.7 | 1.9 | 2.3 | 1.8 |
| **Average number of channels of interest*** | 0.5 | 0.5 | 0.3 | 0.6 |

| | | | | |
|---|---|---|---|---|
| **Retention of data following artifact rejection** | 85.5% | 10.9 | 92.4% | 5.6 |
| **Number of trials for VEP** | 429 | 50.3 | 460.7 | 23.1 |
| **Number of trials for Frequency analysis** | 411.8 | 58.5 | 450.6 | 26 |

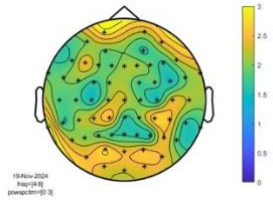
*channels of interest include the 9 channels from the fronto medial region (Fz, F3, FC1, FC2, F4, F1, FC3, FC4, F2) and the three channels of interest from parietal regions (POz, PO3, PO4)

In our sample three participants had missing data for the final video – which was a repeated video – due to participants ending experiment early, or due to all trials in that video being flagged as artifact. For this reason, any further analysis relating to the repeated videos will be using 10 videos (where all participant's data is present) instead of 11 videos.

Overall, Theta power and overall P1 were not correlated with any individual difference's measures, however age was a significantly correlated with overall strength of neural signal. Correlation matrix showing this is present in *Table 2.3*. This likely relates to skull thickness, where thinner skulls (in younger participants) show stronger neural markers (Frodl et al., 2002; Hagemann et al., 2008).

### 2.3.2 Change in Theta-band power with repeated videos

A repeated measures ANOVA was conducted to establish any changes in Theta power (4 to 8 Hz) across the 10 repeated videos. Mauchly's Test of Sphericity revealed the assumption of sphericity to be violated and therefore Greenhouse-Geisser correction is used to reduce risk of Type 1 error. This showed that there was a significant decline in Theta power across the 10 repeated videos ($F$ (2.1, 79.3) = 3.67, $p$ =.03), and that this decline also presents as a linear contrast ($F$ (1, 37) = 4.31, $p$ =.05).



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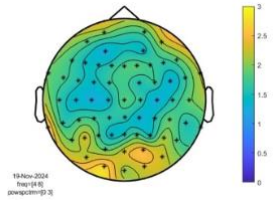

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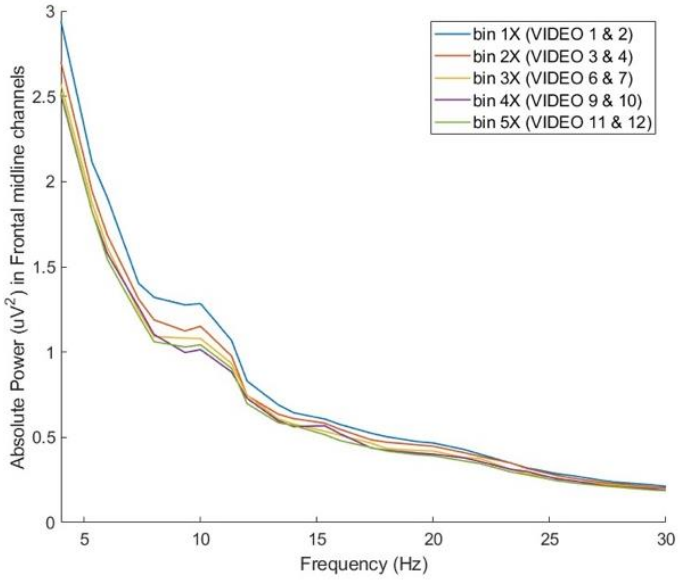


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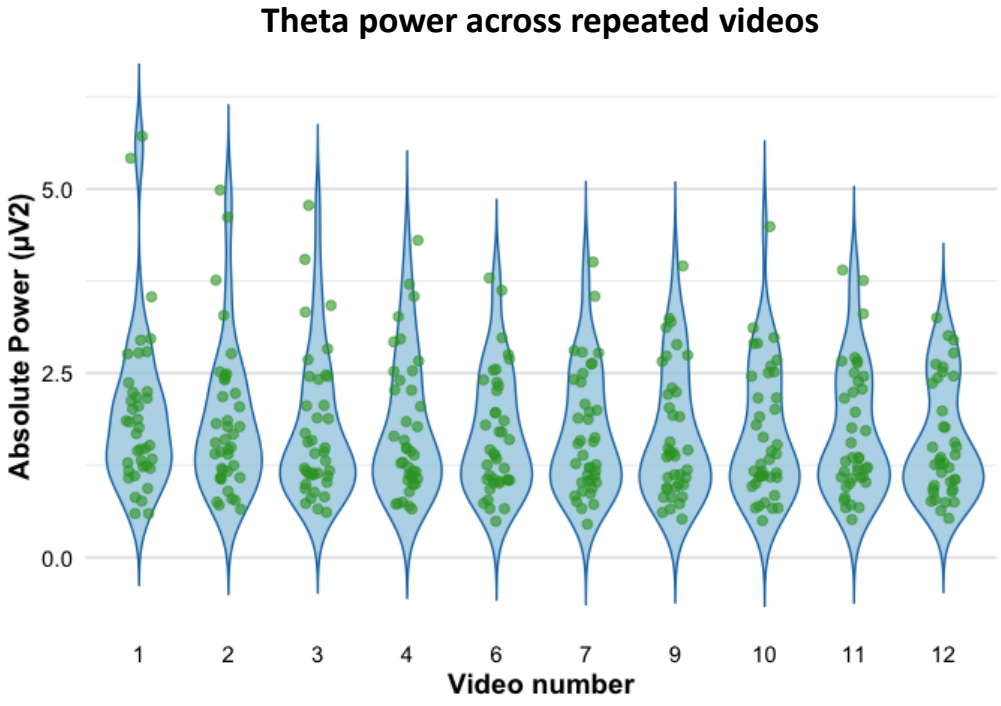



*(d)*

*Figure 2.3. (a)* Topographic representation of absolute theta band activity (4hz-8hz) across the whole scalp for combined time period of the first two videos; video 1 and video 2. *(b)* Topographic representation of absolute theta band activity across scalp for time period of final two videos; video 11 and 12. *(c)* Broadband frequency signal from 4hz to 30hz in the frontal-medial channels of interest where the videos are combined into bins; each bin

containing two of the repeated videos. *(d)* Violin plots showing change in theta band activity across repeated videos only.

*2.3.2.1 Individual differences*

ASD and ADHD traits were added as a covariates into a repeated measures ANCOVA to see any effects of their scores on changes in Theta power. Firstly, using Greenhouse-Geisser corrections, the repeated measures ANCOVA revealed that the main effect of change in theta power is still a significant when controlling for ASD and ADHD traits ($F$ (2.1, 75.7) = 3.70, $p$ = .026), however no significant interaction of ASD traits ($F$ (2.2, 75.7) = .90 , $p$ =.42) nor ADHD traits ($F$ (2.2, 75.7) = .17, $p$ = .76) on the change of Theta power across the video repetitions. Main effects of ASD and ADHD on theta band power also did not reach significance (($F$ (1, 35) = 3.03, $p$ = .09) for ASD, and ($F$ (1, 35) = 3.13 $p$ = .09) for ADHD)

Individual scores of ASD and ADHD for the sample were also split into and median splits for further analysis. Greenhouse-Geisser corrected repeated measures ANOVA with ASD median groupings and ADHD median groupings as between subjects factor still revealed significant main effect of change in theta ($F$ (2.4, 83.2) = 4.64 $p$ <.01). Importantly, it reveals that ASD but not ADHD groupings have an effect on the pattern of change in Theta across the 10 repeated videos ($F$ (2.4, 83.2) = 5.20, $p$ <.01) for ASD and $F$ (2.4, 83.2) = 1.29, $p$ =.29) for ADHD respectively. *Figure 2.4a* shows the difference between high versus low scoring ASD participants on the power of Theta in the 10 repeated videos, such that the lower scoring ASD participants show an expected decline in Theta whereas higher scoring participants present a flatter change in Theta where decline overtime is less apparent. There was no significant effect three-way repetition x ASD group x ADHD group interaction ($F$ (2.4, 83.2) = 1.14, $p$ = .33) and no significant main effect of ASD groupings or ADHD groupings on overall theta band activity (($F$ (1, 34) = .20, $p$ = .66) for ASD, and ($F$ (1, 34) = .01 $p$ = .92) for ADHD).

Further analysis on the median split ASD groups show that when conducting a repeated measures ANOVA on only the lower scoring ASD groups, there is a significant change in Theta band activity ($F$ (1.8, 34.2) = 5.1, $p$ =.01) and that this change is still significant with a linear contrast ($F$ (1, 19) = 6.16, $p$ =.02). Higher scoring ASD group on the other hand does not show the same significant change in theta ($F$ (4.9, 83.3) = .77, $p$ =.57) and does not present with a linear contrast either ($F$ (1, 17) = .36, $p$ =.56). *Figure 2.4a* shows

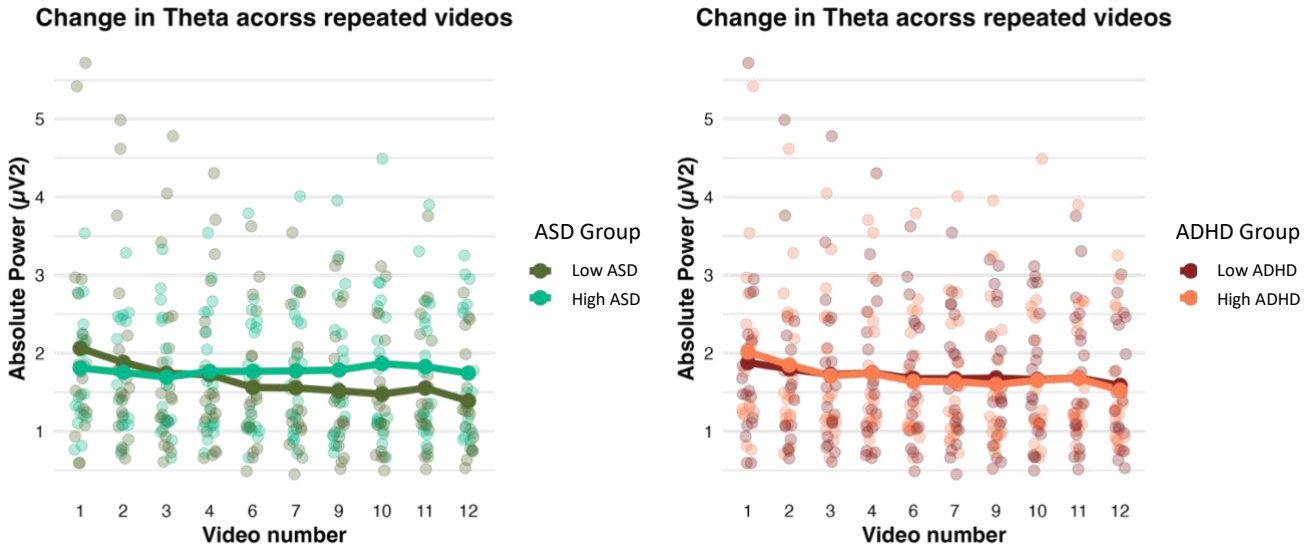
this change in Theta band activity split by median scores of ASD and for median scores of ADHD as a comparison.

*Table 2.3*

*Correlation matrix of individual differences measures and overall average neural measures throughout the experiment*

| | | *Age* | *Overall P1* | *Overall Theta* | *Slope of P1* | *Slope of Theta* | *ASD* | *ADHD* |
|---|---|---|---|---|---|---|---|---|
| **Age** | ***r*** | x | | | | | | |
| | ***p*** | | | | | | | |
| **Overall P1** | ***r*** | **-.33** | x | | | | | |
| | ***p*** | **.04*** | | | | | | |
| **Overall Theta** | ***r*** | **-.41** | **.34** | x | | | | |
| | ***P*** | **.01*** | **.03*** | | | | | |
| **Slope of P1** | ***r*** | .00 | .07 | .09 | x | | | |
| | ***p*** | 1 | .68 | .57 | | | | |
| **Slope of Theta** | ***r*** | -.08 | **-.48** | -.22 | **-38** | x | | |
| | ***p*** | .61 | **.00**** | .19 | **.02*** | | | |
| **ASD composite score** | ***r*** | -.25 | .06 | .20 | .08 | .21 | x | |
| | ***P*** | .12 | .70 | .23 | .62 | .19 | | |
| **ADHD composite score** | ***r*** | -.13 | .01 | -.17 | .23 | .18 | **.42** | x |
| | ***p*** | .43 | .95 | .29 | .16 | .26 | **.01*** | |

**** p< .001, * p< .05**

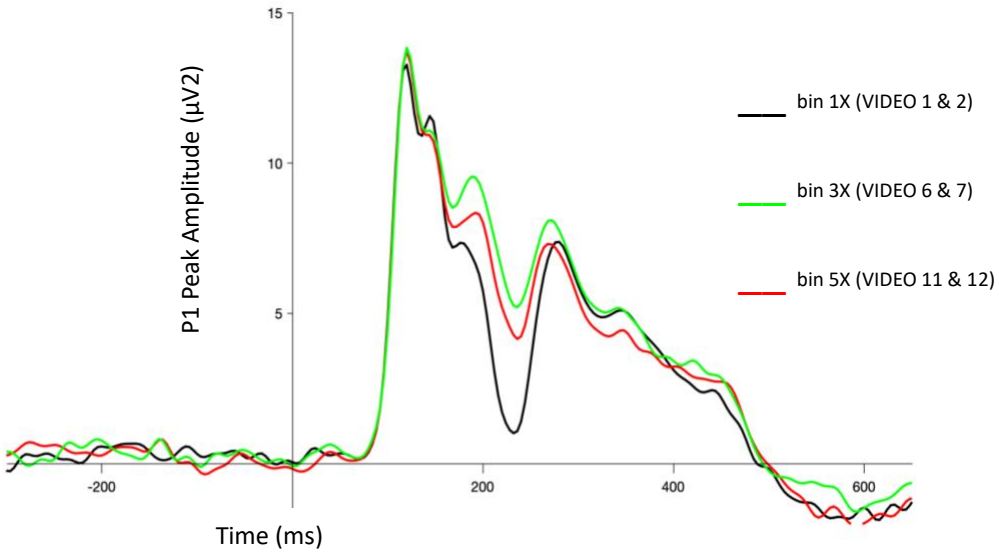



*(a)* *(b)*

*Figure 2.4. (a)* Displays the change in theta band activity across the repeated videos only (video number 5, 8, and 13 which are novel videos are not included) where participants have been median split into high versus low scoring on their ASD traits. *(b)* Displays the change in theta band activty across the repeated videos where participants have been median split into high versus low scoring in their ADHD traits.

### 2.3.3 Change in P1 peak amplitude across repeated videos

Repeated measures ANOVA was conducted on the peak P1 amplitude across the 10 repeated videos, to reveal a non-significant change across them ($F$ (6.4, 239.8) = .90, $p$ = .50).

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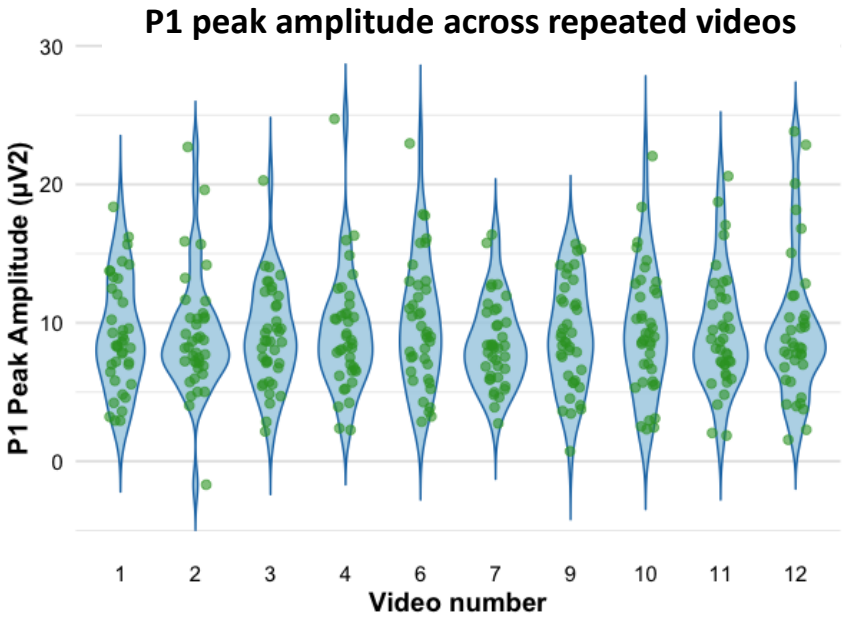



*(b)*

*Figure 2.5. (a)* The ERP/VEP neural signal from the three parietal cluser (PO3, POz, and PO4) where videos are combined into three time bins for where each time bin has 2 repeated videos for vizualisation purposes. *(b)* Violin plots showing change in P1 peak amplitude across repeated videos only.

### *2.3.3.1 Individual differences*

Similarly to Theta analysis, ASD traits and ADHD traits were entered as covariates into the repeated measures model for change in P1 component across the repeated videos. Results from Greenhouse-Geisser corrected ANCOVA reveal the main effect of change in P1

peak component is non-significant when ASD and ADHD traits are controlled ($F$ (6.2, 218.4) = .90, $p$ =.54), and no significant interaction between repetitions and ASD traits or ADHD traits ($F$ (6.2, 218.4) = .99, $p$ =.45) for ASD and ($F$ (6.2, 218.4) = 1.40, $p$ =.21) for ADHD respectively from the same model. There were also no main effects of ASD nor ADHD on P1 amplitude (($F$ (1, 35) = .30, $p$ =.59) for ASD and ($F$ (1, 35) = .09, $p$ =.76).

### 2.3.4 Association between Theta-band change and P1-change

Next, Beta values (representing the slope of change with repetitions) were extracted from regressions conducted on each participant's change in theta and P1 component across the repeated videos. Each participant, therefore, has a Theta band slope and P1 peak amplitude slope. Across the sample, slope of Theta band and slope of P1 peak amplitude showed a significant negative correlation ($r$ (37) = -.38, $p$ = .016). To assess whether this correlation was driven by outliers, we examined Cook's distance and leverage values. No cases exceeded the critical threshold for Cook's distance (Cook's D < 1.0). As for leverage, all values were below the critical threshold of 0.103 (2 x average leverage), except for one participant's leverage score (leverage = .222). However, the correlation remained significant in the expected direction after removing the participant with the highest leverage score ($r$ (36) = -.46, $p$ = .003), confirming the relationship is not an artefact of outliers. Thus, participants whose theta power decreases more with repetitions have P1 values that increase more with repetitions, indicating a trade-off in attention allocation to the video and checkers.

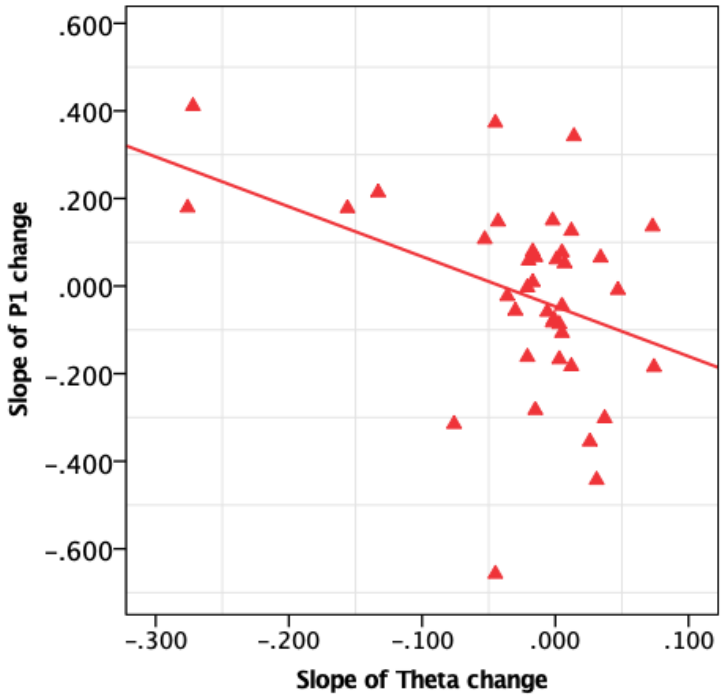


*Figure 2.6*: Represents the relationship between the slope of Theta change against the slope of P1 change. Negative scores here represent a negative change in theta/p1 (i.e. declining over time), numbers closer to 0 reflect a shallower slope whereas numbers further away from 0 reflect the slope becoming steeper.

#### *2.3.4.1 Modulation of theta and P1 with individual differences*

To investigate how ASD traits and ADHD traits are associated with the relationship between theta and P1, at an individual level, we computed correlation between theta-power and P1 amplitude for each individual participant across the 10 repetitions of the video. From the correlation, the **B** (*beta*) values were extracted. A more negative beta indicates a clearer trade-off between when attending to the video versus checkerboard (high gain) and vice versa for positive betas, indicating a less clear trade-off between attending to two different sources of information. Additionally, here, a beta that is closer to 0 indicates a shallow slope. This **beta** value is referred to as slope of modulation for following analysis. Next, correlations are computed to test the relationship between slope of modulation and individual difference scores in ASD and ADHD traits.

From a Pearson's R correlation, we report that ASD z-scores do not significantly relate to the slope of modulation ($r$ (37) = .19, $p$ = .24), whereas ADHD combined z-scores do significantly relate to the slope of modulation ($r$ (37) = .37, $p$ = .02). Essentially, individuals with lower ADHD scores, have more negative slopes of modulation, and individuals with higher ADHD scores, have more positive slopes of modulation. Partial correlations between ADHD traits and slope of modulation while controlling for ASD traits, still reveal a significant positive relationship between ADHD traits and their slope of modulation ($r$ (36) = .33, $p$ = .04).

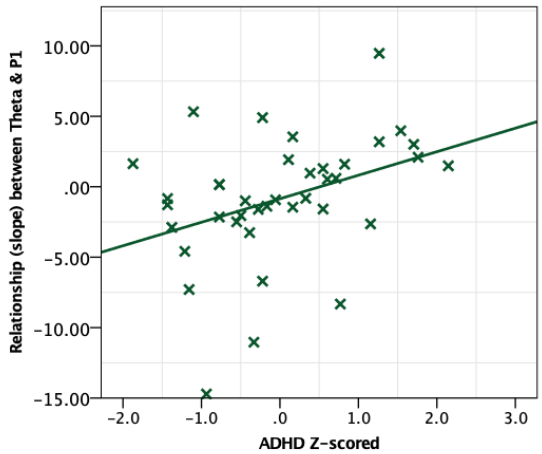
To further investigate this and test the role of ASD and ADHD, and the interaction with the slope of modulation, a regression was carried out with the slope of modulation as the outcome and ASD and ADHD traits as the predictors reveals no significant effect overall (F (3, 35) = 1.98, $p$ = 0.13). *Table 2.4* displays the Betas and significance values for each factor in the model. This demonstrates that only ADHD alone interacts with the slope of modulation.

*Table 2.4*

*Regression model with slope of modulation as an outcome and ASD and ADHD as predictors with an interaction element.*

| | *Estimate* | *Std. Error* | *T value* | *P* |
|---|---|---|---|---|
| **(Intercept)** | -.72 | .75 | -.95 | .35 |
| **ASD** | -.18 | .77 | -.23 | .82 |
| **ADHD** | 1.66 | .79 | 2.11 | .04* |
| **ASD * ADHD** | -.33 | .75 | -.44 | .66 |

For further analysis, in order to check whether the steepness of slope differed between participants who were lower scoring in their ADHD and ASD traits, the participants were median split by their ADHD and then their ASD scores and a one sample *t-test* with 0 as the test statistic was conducted. This revealed that individuals from low-ADHD group were significantly different to 0 ($t$ (19) = -2.42, $p$= .03) and that this difference was negative (i.e. the low ADHD had negative associations between theta and P1). Individuals from high-ADHD group were not significantly different to 0 ($t$ (18) = 1.18, $p$= .25). This was repeated for ASD median split, and we report that nether low ASD nor high ASD groups differed from 0 ($t$ (20) = -1.71, $p$= .10, $t$ (17) = -.17, $p$= .87, respectively).

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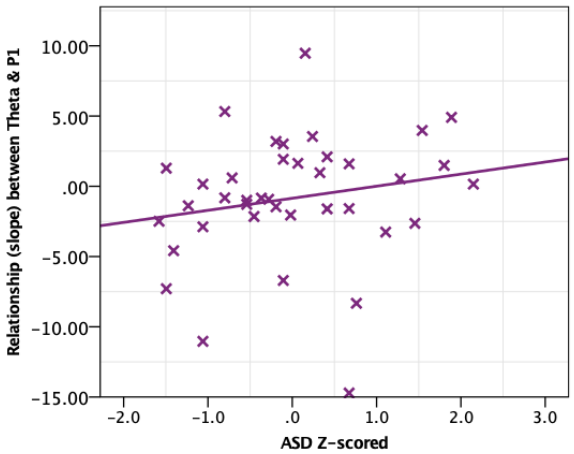


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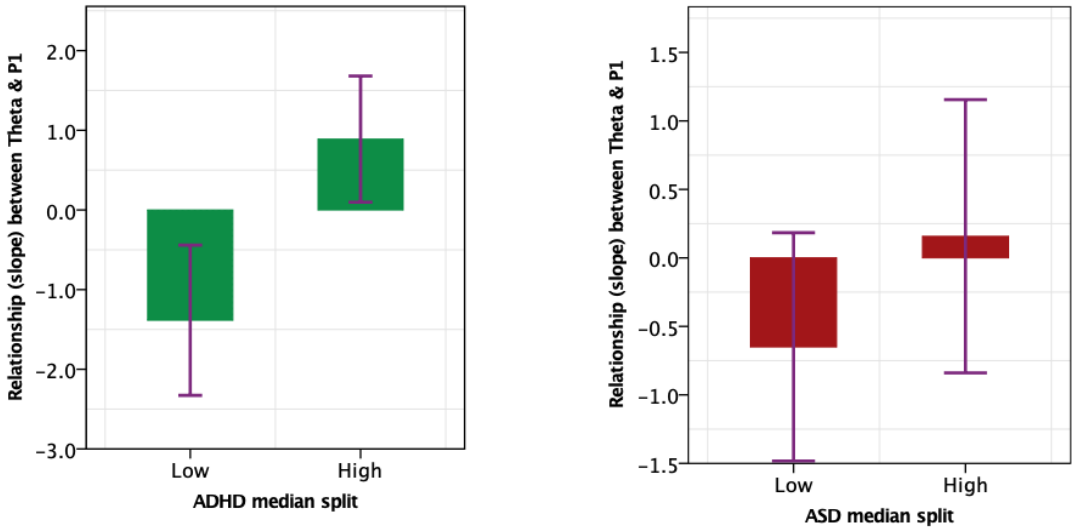



*(c)* *(d)*

*Figure 2.7. (a)* Represents the relationship between ADHD combined Z-scores and the theta/p1 modulation slope. It shoes a positive relationship which is statistcally significant between these two variables. *(b)* Represents the relationship between ASD Z-scores and the theta/p1 modulation slope, which seems to show a similar positive relationship however does not reveal as significant. *(c)* ADHD median split groups and their relationship with the theta/p1 modulation slope indicates that the low scoring ADHD group, has a modulation slope with is significanlty different to 0 (meaning a flat slope), whereas the high scoring ADHD group is not significantly different to 0. *(d)* ASD median split groups and their relationship with the theta/p1 modulation slope. Here neither higher scoring ASD group nor the lower scoring ASD group have modulation slopes that are significantly different from 0.

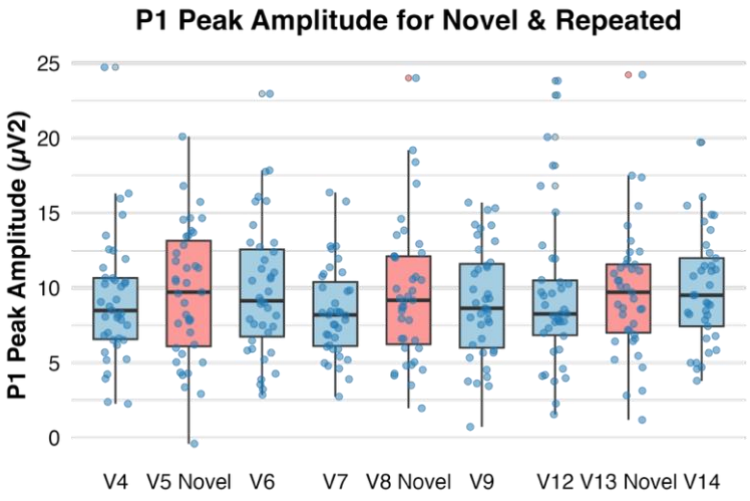
### 2.3.5 Novel video analysis

Dispersed within the repeated videos were three videos that were taken from a different – but related – part of the movie *Fantasia*. These clips appeared as the 5$^{th}$, 8$^{th}$, and 13$^{th}$ video in the sequence of clips. In order to compare changes between the novel video and the video preceding it, and video proceeding it, we first computed an average for theta band activity in video 5, 8, and 13 (the novel videos), and then computed an average for theta band activity in video 4, 7, and 12 (the videos preceding novel videos), and finally an average for video 6, 9, and 14 (the videos proceeding novel videos). The same was completed for P1 peak amplitude. Changes in theta power and p1 peak amplitude for novel videos in comparison to the videos preceding and proceeding it are presented in *Figure 2.8*.

A repeated measures ANOVA on average pre-novel, novel, and post-novel video reveals no significant change in Theta band activity ($F$ (2, 76) = .40, $p$ = .67) nor in peak P1 amplitude ($F$ (2, 76) = 2.1, $p$ = .13). Change in theta power across the three (pre-novel, novel, and post-novel) remains non-significant after ASD and ADHD traits are entered as continuous covariates into the ANCOVA ($F$ (2, 72) = .39, $p$ = .68). There is also no significant main effect by either ASD traits ($F$ (2, 72) = 1.14, $p$ = .33), nor ADHD traits ($F$ (2, 72) = .04, $p$ = .96) in this model.

Similarly, no significant difference was seen in change peak P1 amplitude (across pre-novel, novel, and post-novel) when controlling for ASD and ADHD traits ($F$ (2, 72) = 2.07, $p$ = .13). There was also no significant by either ASD traits ($F$ (2, 72) = 2.01, $p$ = .14), nor ADHD traits ($F$ (2, 72) = .28, $p$ = .76) for ADHD).

Novel videos in the sequence of videos represent new information presented to the participant, and the results here indicate that there is no change in neural signals directed towards the video nor the checkerboard when participants are watching the novel videos in comparison to the repeating and familiar video.

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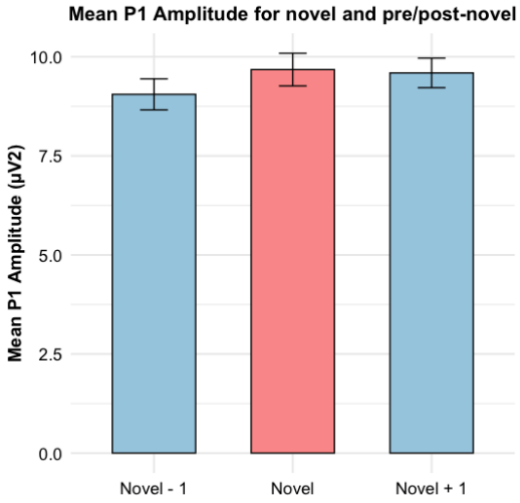


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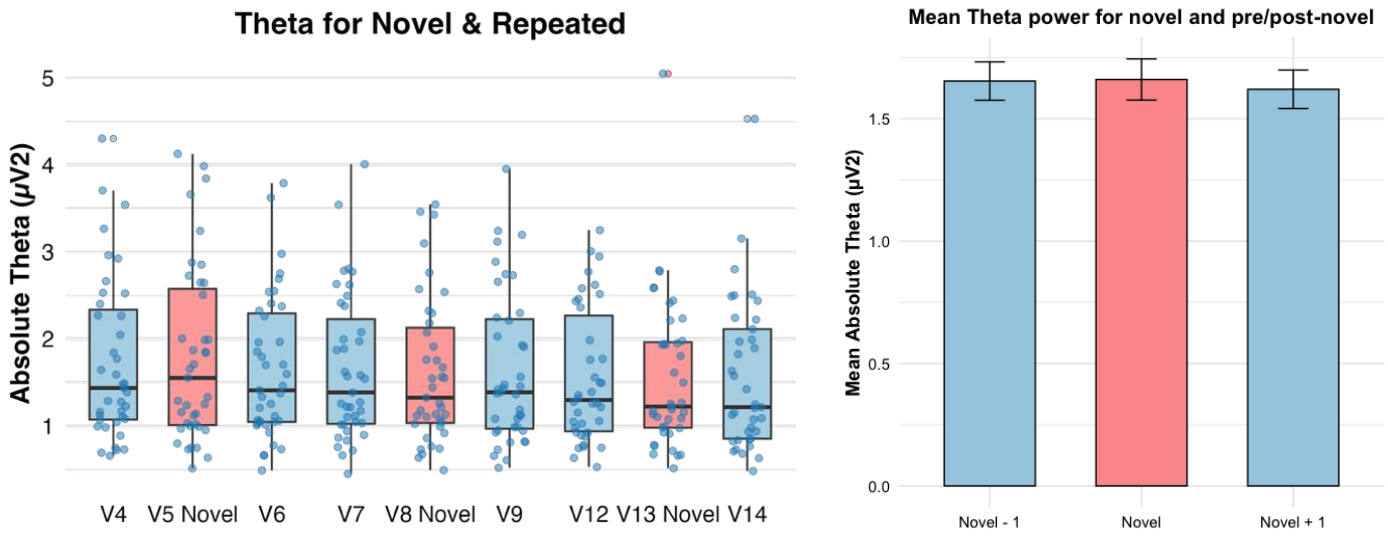



*(c)* *(d)*

*Figure 2.8. (a)* P1 peak amplitude for sections from the video sequence with novel video, video preceding it, and video proceeding it. For example, video 4 (preceding novel video), video 5 (novel video), and video 6 (proceeding novel video). *(b)* The overall P1 peak amplitude for all the videos that precede novel videos (Novel – 1), videos that are novel in the sequence (Novel) and videos that proceed the novel videos (Novel + 1). *(c)* Theta power for sections from video sequence with novel video, video preceding it, and video proceeding it. *(d)* The overall theta power for all videos that precede novel videos (Novel – 1), videos that are novel in the sequence (Novel) and videos that proceed the novel videos (Novel + 1).

### 2.3.6 Executive function

Another individual differences measure that was collected alongside ASD and ADHD scorings was scores on the BRIEF self-report and parental questionnaire. We first average the scores between the self-report and parental report, generating a BRIEF-total score which is then Z-scored to order to normalise distribution of individual differences. Correlations between BRIEF scores and other measured variables in this study are tested and presented below in *Table 2.5*. Significant positive correlations are revealed between BRIEF scores and ASD ($r$ (37) = .48, $p$ = .00) and ADHD traits ($r$ (37) = .92, $p$ = .00). This was expected as previous literature consistently finds executive function difficulties in individuals with Autism and ADHD. The modulation index significantly correlated with the BRIEF-Z however, given the strong correlation between BRIEF and ADHD scores, this suggests these 2 questionnaires capture the same traits.

*Table 2.5*

*Correlation matrix of BRIEF Z scores with other key measures from the experiment.*

| | | Age | Overall P1 | Overall Theta | ASD_Z | ADHD_Z | Theta/P1 slope of modulation |
|---|---|---|---|---|---|---|---|
| **BRIEF_Z** | Pearsons correlation | -.14 | .012 | -.10 | .48 | .92 | .48 |
| | Sig. | .39 | .94 | .55 | .00** | .00** | .00** |
| | N | 39 | 39 | 39 | 39 | 39 | 39 |

## 2.4 Discussion

There were several aims of the study, firstly, to test theoretical accounts of opposing attentional systems in Autism and ADHD with a multi-dimensional approach, secondly, we aimed to track changes in neural markers of information seeking in order to test whether changes and interactions between these markers relate to individual differences in ASD and ADHD traits. We presented four hypotheses at the start of this chapter, firstly that theta band activity (indexing engagement with the repeated video) would initially be high followed by a decline. We found strong support for this hypothesis within our sample. Secondly that the relationship between change in Theta band activity and P1 peak amplitude (indexing sensory response to the checkerboard) would be inversely correlated. We also found clear support for this hypothesis. Thirdly, we hypothesised that the slope of relationship between these two neural markers would be associated with their ASD and ADHD traits. We report partial support for this hypothesis on account of ADHD traits. Finally, we hypothesised that theta band activity would recover at the presentation of novel videos in the sequence of videos. We do not find support for this hypothesis from our data.

Our findings reveal that oscillatory activity in the theta band range does decrease significantly over time, representing a steady decline in engagement with the repeated video. This linear decline in theta power suggests that as the information from the video depletes – due to repetition – fewer cognitive resources are allocated to continue processing the video. This aligns with previous findings reported by Piccardi et al., (2021) in addition to Begus, Southgate and Gliga (2015; 2016), and Begus and Bonawitz (2020) who link activity in the theta frequency to information extraction. An increase in theta band activity is commonly seen when participants receive novel stimulation, for example Lee et al., (2014) reporting a significant increase in theta activity for novel auditory stimulation in an additory oddball task. Additionally, Park et al., (2010) report an increase in frontal theta activity when participants correctly identified different face stimuli in a change blindness paradigm. However, our analysis of neural signals in the novel video presentations show that neither Theta power, not P1 peak amplitude differed when participants were watching the unexpected novel video. These novel videos were used an index of novel information that the participant would engage with following repetitions of the same video. We expected if activity in the theta band was in fact related to information content specifically, we would see a level of recovery during declining theta activity. An important note regarding novel video presentation is that although they differed from the repeating video – showing scenes of different animals

dancing – they were still from the same movie depicting scenes that occur near the scene taken for the repeating video. Indeed, frontal theta activity in young university students was specifically associated with higher uncertainty in reinforcement learning tasks (Cavanagh et al., 2012), indicating that true uncertainty in experience is crucial in probing changes in activity in theta band. It is possible that the general themes in these novel videos are not distinct enough from the repeating video. Nonetheless, it seems more likely that activity within the theta band range (4-8hz) might reflect more general engagement (for example, engagement with the experimental task) instead of more direct information seeking.

While there is no significant increase in the P1 peak component as previously reported by Piccardi et al., (2021), the relationship between the slope of change between theta and P1 is inversely correlated. In other words, for participant's who's change in theta was more negative (sharper decline in engagement), their change in P1 was more positive (sharper increase in sensory response) and vice versa. Furthermore, we report that the slope of modulation between theta and P1 was associated with the adolescent's ADHD traits but not their ASD traits. Below, we discuss how these findings provide partial support for Hauser's (2016) model of neural gain, lending itself to discussion on neural gain that is later explored by Piccardi and Gliga (2022).

Our findings demonstrating a negative association between theta power and P1 peak amplitude corroborates with the results previously reported by Piccardi et al. (2021). Furthermore, we extend the previous paradigm by highlighting the role of individual differences in modulating the relationship between the two neural signals. In the Piccardi et al., study, 10-month-olds who were rated as high visual seekers, their increase in P1 peak amplitude was greater than that predicted by the concurrent decrease in Theta power. In our analyses, we report that individuals who had scored lower in their ADHD traits had a steeper (and more negative slope) theta/p1 modulation slope and individuals who scored higher in their ADHD traits, had a shallower (and more positive) theta/p1 modulation slope. In other words, participants with fewer ADHD traits had clearer trade-offs between change in Theta band and P1 amplitude across the repeated videos. Individuals with higher ADHD traits do not present this trade-off between theta activity and P1 activity indicating less discernment between states of engagement and disengagement with ongoing stimulation.

An important note about Dunn's (2002) sensory seeking conceptualization that is used in the Piccardi et al., study is that it does not directly ascribe sensory hyper/hypo sensitivities

to children with ASD or ADHD. This can be an advantage, that this conceptualization offers a model by which sensory sensitivities can be measured in typical and atypical populations. However, the instruments developed by this model can also present in seemingly conflicting ways, for example, high sensory seeking may tap into active search for novel and diverse sensory stimulation (mapping onto a typical ADHD phenotype), or as preference for repetitive restricted seeking (mapping onto a typically ASD phenotype).

When Dunn's model is taken in combination with Piccardi & Gliga (2022) and Hauser's neural gain model, individuals with ADHD are characterised with low gain, meaning a higher response temperature, and requiring less stimulation to display a behavioural response. This gain model predicts that individuals with low gain (and low sensory seeking) would display a shallower decrease in likelihood of orienting to incoming stimulation, and vice versa for high gain individuals, where we would expect to see a sharper decrease in likelihood of orienting to incoming stimulation. Another way to explain this is that high gain is a state that enables processing of highly informative (or highly specific) ongoing signals at the expense of less informative incoming stimulation, and low gain is the opposite, where the detection and processing of less informative stimulation is strengthened at the expense of highly informative (highly specific) ongoing signals (see *Figure 1.11* for visualisation).

Specifically for this EEG paradigm, gain model would predict that individuals in the low gain state would display a shallower decrease in the likelihood of processing incoming stimulation (checkerboard presentations) while they are engaged with processing of ongoing stimulation (the cartoon videos). In other words, be more likely to disengage from highly informative ongoing stimulation when presented with highly salient incoming sensory information.

In our data we see that the change in theta over time and change in P1 over time is negative overall, this means that overall, there is a trade-off between processing one source of stimulation over the other. Importantly, we see that the relationship between theta and p1 change is modulated by ADHD traits. Individuals who scored lower on the ADHD measurement, had steeper, more negative scores. As ADHD traits increase, the slope becomes shallower, and arguably more positive (statistically individuals who scored low in ADHD, had a slope significantly different from 0 and individuals whereas scored higher had slopes not significantly different from 0). We had expected this finding, however our hypotheses

included ASD traits as playing a larger role in this relationship; namely that the steeper slopes would correspond with individuals who had higher ASD traits. We do not see this in our analysis of the sample.

There is some indication that the change in theta power across the repeated videos differs between participants who scored high in ASD traits versus those that scored lower. The analyses on median split ASD scores revealed that higher ASD scores associated with a flatter decline in theta power. This is an important finding by itself, lending itself to literature that demonstrates atypical patterns of theta band activity (Hornung et al., 2019; Cohen et al., 2008; Wang et al., 2013; Shephard et al., 2017), but also important consideration when our dependent measure is the relationship between the change in Theta and change in P1. It could be the case that the modulation of the theta/p1 slope is not shaped by the ASD traits due to this unexpected pattern in theta band activity. A flatter slope in theta decline for high ASD group likely reflects differences in processing of repetitive stimuli, specifically that there is sustained engagement with the video, even in the case that it is depleting in the information it is offering. Indeed, this is evidenced by findings of weaker early exploratory behaviours in infants at elevated likelihood of ASD (Gliga et al., 2018) as well as in adolescents with autism (Heaton & Freeth, 2016).

Lastly, our tests of executive function show that BRIEF scores were positively correlated with both ASD and ADHD traits, consistent with literature highlighting executive function difficulties in these populations (Gioia et al., 2000; Gilotty et al., 2002). BRIEF scores however did not explain additional neural variability beyond ASD and ADHD traits, suggesting that the observed individual differences relating to ASD traits and ADHD traits are sufficient in explaining the variance in individual differences.

### 2.4.1 Limitations

A limitation of the present sample is the asymmetric distribution of ADHD diagnoses and traits: with only one participant who presented a formal ADHD only diagnosis, and ADHD scores being generally low in the non-ASD group ($M = 12.95$, $SD = 6.56$) compared to the ASD group ($M = 24.37$, $SD = 7.62$). In essence, while we had a good range of ADHD traits in the sample, we had very few participants with high ADHD traits in the absence of high ASD traits. This places a limit on our ability to fully test the hypothesis of opposing ends of an attentional continuum.

Contrary to our hypothesis, we found no recovery of theta power or change in P1 amplitude during the three novel presentations of the videos compared to the preceding and following repeated videos. This null finding raises questions about whether the task successfully manipulated informational novelty. As previously suggested, one possibility is that the novel videos did not represent information that was significantly different from the repeating videos (similar music, similar animation) and thus activity within the theta band continued to decline as if participants were viewing the repeated videos. Alternatively, it is also possible that the videos did differ significantly however activity in the theta band closely associated with general engagement instead of a direct measure of information uptake. Future research should use novel video content that is significantly different to the repeating videos (eg., taken from different movies or different genres) and test for perceived novelty to validate the manipulation. Despite this limitation, the significant decline in theta over repetitions and the predicted theta-P1 trade-off suggests that the repetition manipulation was effective in depleting engagement, even if the novel videos failed to recover it.

### 2.4.2 Conclusion

This experimental paradigm was utilized as it captures a trade-off between attentional engagement to material that is depleting in information and sensory processing of an unrelated stimuli, processing of which marks disengagement with ongoing stimulation from the video. The *information prioritization hypothesis* put forth by Piccardi et al., (2021) and Piccardi and Gliga (2022) posits that high-gain states are associated with stable engagement and disengagement, whereas low-gain states are linked with more erratic engagement and disengagement. We find support for this hypothesis within our sample specifically for ADHD traits, however, the theory of ASD being an attentional opposite of this, with stable trade-off between the two sources of stimulation was not found in our sample. Although ASD traits did not modulate the theta/p1 modulation slope, the flatter decline in theta activity in high scoring ASD participants suggests a distinct attentional profile all together, where neural signals do not demonstrate lessening of engagement as information depletes. Our results show that information processing differences between Autism and ADHD are more complex than the described in Landry (2021). The role of the dopaminergic system is likely therefore, to be more complex. While an altered dopaminergic system in relation to ADHD traits presents as affecting trade-offs between engagement and disengagement with ongoing and incoming stimuli, an altered dopaminergic system in relation to ASD may present as an altered baseline engagement with ongoing stimuli, which is less impacted by incoming stimuli.

# CHAPTER 3:

## 3. Information trade-offs in typical adults with Autistic traits and ADHD traits.

## Goal of the chapter

*The current chapter uses the same paradigm as in Chapter 2, but in a general sample of adult participants. This study aims to extend the findings from the previous adolescent study, adopting the same multidimensional approach in subclinical adult participants in order to test whether theories of neural gain are upheld in this sample.*

*The hypotheses in this study are identical to the previous study, such that we expect to see a decline in theta band activity with the repetition of the video, and a negative relationship between change in theta band activity and P1 activity. We also predict, similar to the previous study, that this relationship would be modulated by individual differences in ASD and ADHD traits.*

*In this small adult participant sample (n= 25) the expected relationship between change in Theta band and change in P1 peak component was not observed. Theta band activity decreased in participants with low ASD and ADHD traits but not in those with high ASD and/or ADHD traits. The results from this sample, as well as the novel finding in relation to Theta band activity are discussed. Chapter 2 and 3 findings are compared from the perspective of using subclinical samples versus clinical samples.*

# 3.1 Introduction

In the previous chapter we utilised the Piccardi et al., (2021) EEG paradigm using neural signals and relationships between these neural signals in order to assess attentional engagement with stimuli. Participants watch a repeated video where the repetitions stimulate depletion of information over time. This depletion of information corresponded with a decline in theta band activity in the fronto-medial regions of the brain which indexes engagement (for review: Klimesch, 1999; Karakas, 2020). Importantly, the results of the experiment revealed that individual differences in ADHD traits modulate the slope of relationship between the two neural signals (theta band power and P1 peak amplitude). Specifically, that individuals with higher ADHD traits have a shallower, and potentially more positive slope of theta/p1 modulation whereas individual with fewer ADHD traits have a steeper, and more negative slope of theta/p1 modulation. In other words, the trade-off between engagement with ongoing stimulation, versus engagement with incoming stimulation, was less apparent in individuals with higher ADHD traits. This aligns with Hauser (2016)'s neural gain model, as well as predictions later made by Piccardi & Gliga (2022). In addition, we saw that in this paradigm, ASD traits did not present as the opposite end of the spectrum with regards to slope of modulation between theta/p1, although there are some indications of differences in how theta band activity changes across repeated videos between those with lower and higher number of ASD traits. However, whether these findings generalise to adults, or whether these trends manifest in sub-clinical samples remains unknown.

Attentional systems undergo maturation from adolescents into adulthood, particularly in the prefrontal and striatal dopaminergic pathways (Luna et al., 2004; Crone, 2009). This brings into question whether engagement and information seeking present in similar ways in young adults who have more matured dopaminergic pathways. While adolescents with higher ADHD traits exhibit a weaker trade-off between theta and P1 activity, it may be the case that adults with higher ADHD traits show clearer trade-offs between theta and P1 activity, reflecting more cognitive control or learned compensatory mechanisms (Francx et al., 2015).

While ASD and ADHD are typically diagnosed in childhood and adolescence, and much research focuses on these early developmental stages, both conditions persist across the lifespan. Growing evidence shows that that Autism and ADHD can be modelled and studied as quantitative traits in the general populations (Happe & Frith 2020, 2021; Stanton et al.,

2020; Constantino & Todd, 2003). This continuum-based perspective supports investigation of these traits in non-clinical samples, since these traits manifest along a broad spectrum in the population. The neurodiversity and multidimensional approach we applied in the adolescent sample has shown to be valuable in adults (Waldren et al., 2024), and therefore we extend the same approach that was applied in adolescents to adult non-clinical samples. We use the same EEG paradigm with two key methodological changes: (1) use of adult non-clinical sample of participants, and (2) use of self-report measures of ASD and ADHD traits.

Adopting the same approach from the previous chapter, we investigate whether similar trends emerge in an adult sub-clinical population. We test whether the neural gain model can be applied to this population to reveal the opposing attentional profiles of ADHD and ASD as initially predicted or whether an alternative pattern will emerge. In adult populations, instead of using clinically validated rating scales (such as the ADOS-2 and Conners-4), we use freely available online surveys that are less time consuming to administer and still offer variation in ASD and ADHD traits within the population. Self-report measures such as the Autism Quotient (AQ), and Broad Autism Phenotype Questionnaire (BAPQ) to measure ASD traits, and Adult ADHD Self-Report Scale (ASRS), and Wender Utah Scale (WURS) to measure ADHD traits allow us to quantify subclinical traits which are sensitive to variation present within the general population.

Subclinical measures such as the AQ and BAPQ are validated, frequently used measures in research into ASD traits in the general non-ASD population. The term ‘broad autism phenotype’ for example is used to reference the presence of neurodivergent traits exist in the general population (Baron-Cohen et al., 2001; Constantino & Todd, 2003). The AQ has been used in research related to visual search (Grinter et al., 2009), gaze responses to social and non-social stimuli (Bayliss & Tipper 2005), and is commonly used as a screening instrument in clinical settings (Woodbury-Smith et al., 2005). The BAPQ has also been used as a clinical screening measure and is has been shown to associate with joint-attention differences as a feature of Autism (Swanson & Siller, 2014). The BAPQ has been validated in adults from general populations as well as in individuals with first-degree autistic relatives (Hurley et al., 2007). Both AQ and BAPQ reliably associate with altered sensory processing and individuals who score highly on either of these measures manifest a number of sensory difficulties (Yadon & Vonarx, 2024; Robertson & Simmons, 2013; Bang & Igelström, 2023).

The ASRS and WURS are similar validated, frequently used measures of ADHD traits in general non-ADHD population. The ASRS is developed by the WHO and is used as a screening instrument in clinical settings in adults (Kessler et al., 2005). The ASRS has been used to explore relationships between ADHD traits and comorbidities such as mood disorders (Katzman et al., 2017), and Anxiety, depression and substance abuse (Simon et al., 2009), both reporting notable associations. The WURS on the other hand is a unique assessment of retrospective childhood ADHD symptoms in adult participants (Ward, Wender, & Reimherr, 1993) and has been effective in distinguishing ADHD patients from patients with major depressive disorder (Ward, Wender, & Reimherr, 1993) and used to identify positive responders to pharmacological interventions (Klee & Garfinkel 1986). Both ASRS and WURS reliably correlate with impulsivity, inattention, and difficulties in executive function, with higher scores predicting increased challenges in daily functioning (Kessler et al., 2005; McCann et al., 2000).

Our hypotheses below are identical to the previous study;

1. We predict that frontal theta oscillatory power to the repeated video initially be high, followed by reduction of power due to information depletion from the video.

2. We expect the P1 amplitude to be inversely related to frontal theta amplitude.

3. We expect a difference in degree of modulation of P1 by theta that is explained by individual differences scores in ASD and ADHD. Specifically, we expect that higher ADHD traits will be associated with a shallower (less negative) theta/P1 modulation slope, reflecting a weaker trade-off between engagement with ongoing stimulation and incoming stimulation. This would replicate the findings from the adolescent sample. Based on the neural gain model, we expect that high ASD traits will be associated with steeper (more negative) modulation slope, however this was not apparent in the adolescent model.

4. If theta indeed captures information depletion, for the novel videos in the sequence, we predict to see a recovery in theta band activity compared to the video preceding it.

# 3.2 Methods

## 3.2.1 Participants

Thirty participants took part in this EEG study (21 females, M age = 22.1, SD age = 5.9). Four participant's data was not included in the final analysis due to issues with EEG recording, and one participant's data was not included due to extreme values exceeding 3 standard deviations above the mean on neural measures of slope of modulation between theta and P1. The final sample consisted of twenty-five participants (18, females, M age = 20.8, SD age = 2.6). In this sample, there were 3 participants who self-reported on having a diagnosis of neurodevelopmental conditions (1 ADHD, 1 ASD, 1 Autism, ADHD and Dyspraxia).

The sample consisted mostly of students enrolled in UEA undergraduate Psychology programme, who participated in exchange for course credits as part of their program requirements. Procedure was approved by the Research Ethical Committee of the University of East Anglia, Norfolk, United Kingdom.

## 3.2.2 Design

In line with the experiment conducted in the adolescent sample, this experimental design employs a repeated-measures design and mainly uses individual differences measures (ASD and ADHD) on continuous scales. The key difference in methodological approach between the adolescent sample and the adult sample is the choice of individual differences measures. In the previous sample we use clinically validated rating scales (Conners-4 rating scale, ADOS-2, and the BRIEF), whereas in this sample we use self-report of subclinical measures of ASD and ADHD traits (detailed in **Section 3.2.3.1**).

Participants view the same 14 clips, where 11 are repetitions of the same clip and 3 are novel videos from different parts of the movie. We measure absolute theta power from the fronto-medial regions of the brain which indexes engagement with the ongoing video. We also extract P1 peak component directed to the checkboard as an index of disengagement with the video. The same content related questions were posed the participants after the same 6 randomly selected clips.

The within-subjects independent variable was the video repetition (with 11 levels for 11 instances of repeated videos), with ASD and ADHD traits acting as continuous covariates in a repeated-measures design. For supplementary group-level analyses, we implemented

median splits on the continuous scores, treating them as between-subjects factors in order to better visualise trends. The dependent variables in this study were the frontal-medial theta band power and importantly, the slope of the theta/P1 modulation across the repeated videos.

### 3.2.3 Material

#### *3.2.3.1 Measures of individual differences*

The Broad Autism Phenotype Questionnaire (BAPQ) is a freely available measure used to capture subclinical ASD traits (Hurley et al., 2007). This survey encompasses variation in traits including social aloofness, pragmatic language, and rigidity. There are a total of 36 items and are scored on a scale between 1 to 6 where 1 is ‘very rarely’ and 6 is ‘very often’. Reverse scored items is for the following items in the questionnaire (1 becomes 6, 5 becomes 2, etc.): 1, 3, 7, 9, 12, 15, 16, 19, 21, 23, 25, 28, 30, 34, 36. These scores are used to compute an mean for each participant which represents their BAPQ score (Hurley et al., 2007).

The Autism Quotient (AQ) is another freely available self-report measure of subclinical ASD traits and has been suggested to be a means of quickly estimating ASD risk in order to guide referrals (Baron-Cohen et al., 2001). There are a total of 50 items and are scored on a 4-point scale where  is "Definitely agree" or "Slightly agree" responses to questions 2, 4, 5, 6, 7, 9, 12, 13, 16, 18, 19, 20, 21, 22, 23, 26, 33, 35, 39, 41, 42, 43, 45, 46 score 1 point. "Definitely disagree" or "Slightly disagree" responses to questions 1, 3, 8, 10, 11, 14, 15, 17, 24, 25, 27, 28, 29, 30, 31, 32, 34, 36, 37, 38, 40, 44, 47, 48, 49, 50 score 1 point. For the total score, all items are summed (Baron-Cohen et al., 2001).

The Wender Utah Rating Scale (WURS) was developed to assess adults’ retrospective account of ADHD related behaviours from their childhood (Ward, Wender, & Reimherr., 1993). The long version consists of 61 items and a shortened version made up of 25 items best able to discriminate between adults with and without ADHD. In this study we use the 25-item freely available survey where items are scored on a scale from 0 to 4. For the total score, all items are summed.

The Adult ADHD Self-Report Scale (ASRS) developed by the World Health Organisation Composite International Diagnostic Interview (Kessler et al., 2005) is a short 6-question survey. The ASRS has a part A with 6 items and part B with 12 items and therefore a total of 18 items, all rated on a 5-point scale. “Often” and “Very Often” responses from participants are automatically scored 1 point. For items 2, 3, 4, 9, 12, 16, and 17, a response of “Sometimes” also scores 1 point. The rest are not allotted any points. For the total score, all items are summed.

To compute one Autism and one ADHD score per participant, a Z-score is calculated for each questionnaire and then the z-scores for the AQ and BAPQ are added, similarly the z-scores for the ASRS and the WURS.[1]

*3.2.3.2 Stimuli*

Refer to Chapter 2: subsection 2.2.3.2

*3.2.3.3 Apparatus*

Refer to Chapter 3: subsection 2.2.3.3

### 3.2.4 EEG pre-processing

Refer to Chapter 2: subsection 2.2.4

Data quality measures are summarised in *Table 3.2* of the **Results** section. The average number of channels interpolated in the adults is higher than that from the adolescent sample, however artifact rejection reveals better trial retention in comparison to adolescent sample. Additionally, the number of trials retained for analysis between VEP and frequency analysis were comparable, however just as with the adolescent sample, more trials were retained for the VEP analysis.

### 3.2.5 EEG analysis

Refer to Chapter 2: subsection 2.2.5

[1] A 10-item ID curiosity scale was also given to the participants used to measure epistemic curiosity in the participants in two dimensions: Interest and Deprivation (Litman & Silvia, 2006). This was not included in any analysis.

### 3.2.6 Procedure

Upon signing up for the study participants received a link to complete the surveys online in addition to participant information sheets and a consent sheet relating to taking part in the surveys. The surveys take approximately 15-20 minutes to complete, and participants were requested to complete the surveys prior to attending the experimental session. Upon arrival into the lab the participants were briefed on the experimental paradigm and were given additional information sheets which outline details pertaining to the EEG study, followed by consent sheets. First the participant's head circumference is measured in order to select a correctly fitting EEG cap. Then the EEG cap is placed on the head of the participants, and each electrode is individually gelled using blunt-needle syringe. This takes approximately 30 minutes dependent on hair texture and quality of EEG recording. After the EEG is set up, participants were given instructions to watch the videos presented on the screen and answer the questions when they come up by pressing the RIGHT or LEFT buttons on the keyboard. The end of each video can be taken as a break in case the participant needed to ask a question or do large movements like stretching otherwise they could press SPACE to continue playing the next video clip. After the videos are finished, the EEG cap is removed, and participants are directed to the washroom facilities within the lab if they choose to wash their hair. Participants were then given a debrief sheet and given an opportunity to ask additional questions if they had. The total length of the time participants watched the video was 25 minutes and the full EEG session including setting up and washing up afterwards is approximately 1 hour 30 minutes.

# 3.3 Results

## 3.3.1 Descriptives

Shapiro-Wilk tests showed scores on the BAPQ, ASRS, and WURS were not normally distributed (W (25) = .89, $p$ = .01, W (25) = .89, $p$ = .01, W (25) = .91, $p$ = .04 respectively), and scores on the AQ were normally distributed (W (25) = .92, $p$ = .07). Although Shapiro-Wilk tests indicated that the raw scores of the BAPQ, ASRS, and WURS deviated significantly from normality, we proceeded with parametric tests for the analyses. Due to the sample size being small, Shapiro-Wilk tests are less reliable with low power (Ruxton et al., 2015), for this reason visual inspection of frequency plots (presented in *Appendix D*) demonstrated that scores on these surveys demonstrated rough bell-shaped curves, without severe outliers and severe skews.

Descriptive statistics reveal that the range was varied across all the tests of individual differences (*Table 3.1*).

Scores from the two ASD surveys (AQ and BAPQ) and for the two ADHD surveys (Wender-Utah scale and ASRS) are combined to create a composite score for total ASD and total ADHD; this is then z-scored in order to normalise distribution of individual differences.

Data quality and cleaning shows that more channels were removed and extrapolated in the adult sample in comparison to the previous adolescent sample (7 in adults versus 3 in adolescent sample). This may be due to more hair and thicker scalps in the adult participants. The number of channels of interest that were removed were different (0.7 in adults and 0.4 in adolescents). This is presented, along with number of trails retained in analysis in *Table 3.2*.

Prior to the main analyses, we assessed for outliers on key neural measures. As mentioned in **Section 3.2.1,** one participant's data was not included due to values exceeding 3 standard deviations above the mean on neural measures of slope of modulation between theta and P1. No other participants exceeded the ± 3.29 z-score threshold for outliers (Field, 2018) on any of the neural measures (theta power in each video, P1 peak amplitude in each video, and overall slope of the theta/P1 modulation).

*Table 3.1*

*Descriptive statistics of individual differences measures across sample*

| | *Mean* | *Std. Dev* | *Minimum* | *Maximum* | *Range* |
|---|---|---|---|---|---|
| **Age** | 20.8 | 2.6 | 18 | 29 | 11 |
| **AQ** | 30.3 | 9.8 | 5 | 45 | 40 |
| **BAPQ** | 3.2 | .70 | 2.3 | 4.9 | 2.6 |
| **Wender-Utah** | 37.2 | 20.5 | 7 | 93 | 86 |
| **ASRS** | 8.4 | 3.5 | 4 | 18 | 14 |

*Table 3.2*

*Data quality and cleaning record for EEG data across sample*

| | *Mean* | *Std. Dev* |
|---|---|---|
| **Number of channels removed** | 7 | 6.7 |
| **Average number of channels of interest*** | 0.7 | 0.7 |
| **Retention of data following artifact rejection** | 94% | 8.7 |
| **Number of trials for VEP** | 460.8 | 44.1 |
| **Number of trials for frequency analysis** | 448.8 | 50.4 |

*channels of interest include the 9 channels from the fronto medial region (Fz, F3, FC1, FC2, F4, F1, FC3, FC4, F2) and the three channels of interest from parietal regions (POz, PO3, PO4)

### 3.3.2 Bi-variate correlations between key measures

Overall theta power and overall P1 peak amplitude were not correlated with each other, unlike in the previous chapter where there was medium strength association between overall P1 and overall theta ($r$ = .34, $p$ = .03). In this sample the direction of association is in the expected direction ($r$ (23) = .18, $p$ = .40) however it does not reach significance (this is also presented in *Table 3.3*). This may be attributed to smaller sample size in this study (N = 25), in comparison to the previous chapter (N = 39). A post-hoc power analysis using G power (Faul et al., 2009), indicates that with the adult sample of N = 25, the study is underpowered with a power of .39 in order to detect the effect size observed in the adolescent sample ($r$ = .34). To achieve the observed $r$ = .34 in the adult sample, with a power of 0.8, at $\alpha$ = .05, a sample of approximately 65 participants would be required.

There were no associations between age and overall P1 and overall theta, as was seen in the adolescent sample. These correlations are presented in *Table 3.3*. This likely represents a stabilising of scalp thickness in the adult sample, unlike in the adolescent sample, where younger participants (with thinner scalps) had stronger neural signals in both P1 and theta (Frodl et al., 2002; Hagemann et al., 2008).

There was a significant positive correlation between ASD z-scores and ADHD z-scores ($r$ (23) = .71, $p$ < .001). Individual differences are not significant associated with overall theta activity and overall P1 peak amplitude across the experiment, however both ASD scores and ADHD scores show positive significant associations with age. In other words, as age increases, scores in ASD and ADHD increase.

### 3.3.3 Change in Theta-band power with repeated videos

A repeated measures ANOVA was conducted to see changes in Theta power across the 11 repeated videos. Mauchly's Test of Sphericity revealed the assumption of sphericity to be violated and therefore Greenhouse-Geisser correction is used to reduce risk of Type 1 error. This showed that there was no significant change in theta power across the 11 repeated videos ($F$ (4.8, 115.3) = .93, $p$ = .46). Topographic representation of theta band activity, as well as broad spectrum change across the videos is presented in *Figure 3.1.* Overall change in theta band activity across the 11 repeated videos is presented in *Figure 3.1d.*

*Table 3.3*

*Correlation matrix of individual differences measures and overall average neural measures throughout the experiment*

| | | *Age* | *Overall P1* | *Overall Theta* | *Slope of P1* | *Slope of Theta* | *ASD* | *ADHD* |
|---|---|---|---|---|---|---|---|---|
| **Age** | ***r*** | **x** | | | | | | |
| | ***p*** | | | | | | | |
| **Overall P1** | ***r*** | .09 | **x** | | | | | |
| | ***p*** | .66 | | | | | | |
| **Overall Theta** | ***r*** | .38 | .18 | **x** | | | | |
| | ***P*** | .06 | .40 | | | | | |
| **Slope of P1** | ***r*** | .01 | .03 | .03 | x | | | |
| | ***p*** | .95 | .89 | .89 | | | | |
| **Slope of Theta** | ***r*** | .32 | .004 | -.13 | .07 | x | | |
| | ***p*** | .12 | .99 | .54 | .73 | | | |
| **ASD** | ***r*** | **.48** | .13 | .24 | .12 | .32 | **x** | |
| | ***P*** | **.02*** | .54 | .24 | .56 | .13 | | |
| **ADHD** | ***r*** | **.55** | .30 | .30 | -.15 | .31 | **.71** | **x** |
| | ***p*** | **.004**** | .14 | .14 | .47 | .13 | **.00**** | |

**** p<.001, *p<.05**

*3.3.3.1 Individual differences*

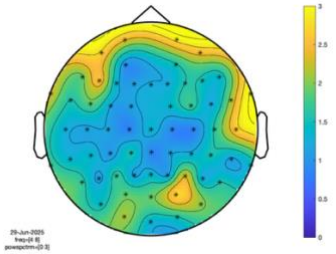
ASD and ADHD traits were added as covariates into a repeated measures ANCOVA to see the effects of individual differences on changes in Theta power across repeated videos. Firstly, using Greenhouse-Geisser corrections, the repeated measures ANCOVA revealed that when ASD and ADHD traits are controlled, there is still no main effect of change in theta band power across the repeated videos ($F$ (4.9, 107.8) = 1.00, $p$ = .42). There was also no significant interaction between repetitions and ASD ($F$ (4.9, 107.8) = 1.30, $p$ = .27) and while there was a trend for an effect of ADHD traits, the interaction between repetitions and ADHD did not reach significance ($F$ (4.9, 107.8) = 1.92, $p$ = .10) when corrected for sphericity. There was also no main effect of ASD scores on theta band activity ($F$ (1, 22) = .04, $p$ = .84), and no main effect of ADHD on theta band activity ($F$ (1, 22) = .84, $p$ = .37).

Individual scores of ASD and ADHD for this sample were also split into median splits for further analysis. Greenhouse-Geisser corrected repeated measures ANOVA with ASD median groupings still show no significant change in Theta ($F$ (4.9, 103.3) = 1.50, $p$ = .20). Additionally, it shows ASD traits did not significantly interact with change in theta ($F$ (4.9, 103.3) = 1.31, $p$ = .27). A trend for an interaction was found for ADHD traits ($F$ (4.9, 103.3) = 2.08, $p$ = .08), indicating a marginal association between change in theta activity and ADHD high/low groupings. There were no main effects of ASD median split grouping, nor ADHD median split grouping on theta band activity (($F$ (1, 21) = .51, $p$ = .48) for ASD, and ($F$ (1, 21) = .15, $p$ = .70) for ADHD respectively).

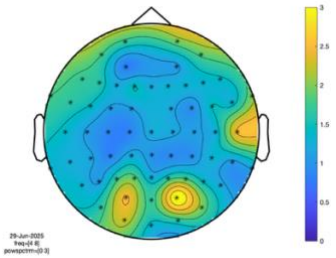
In this model where ASD and ADHD median split grouping groupings are entered as between subjects factors, the 3-way interaction between theta band across the repetitions, ASD, ADHD is significant ($F$ (4.9, 103.3) = 2.49, $p$ = .04) indicating an effect on theta change differed depending on the median split groupings for ASD and ADHD. In *Figure 3.1e* we demonstrate one way to visualise this using **Beta** values which depict the slope of change for theta band activity. We can see that when participants are low scoring in both trait measures, there is a more negative **Beta** (indicating a more negative slope), whereas when participants are scoring high in either ASD or ADHD or both, the average slopes are closer to 0 on the axis, indicating a shallow slope.

To further investigate this three-way interaction, we tested whether the Beta of low ASD + low ADHD participants was significantly different from 0. A one-sample $t$-test with 0 as the test variable reveals that participants who scored low on both measures were still not

significantly different to 0 ($t$ (7) = -2.29, $p$ = .06), although it is close to reaching significance. The other groupings, all revealed that the slope of theta was not significantly different to 0 (($t$ (3) = 1.24, $p$ = .30) for low-ADHD high-ASD, ($t$ (4) = 1.29, $p$ = .27) for high-ADHD low-ASD, and ($t$ (7) = .38, $p$ = .72) for high-ADHD high-ASD)).



*(a)*



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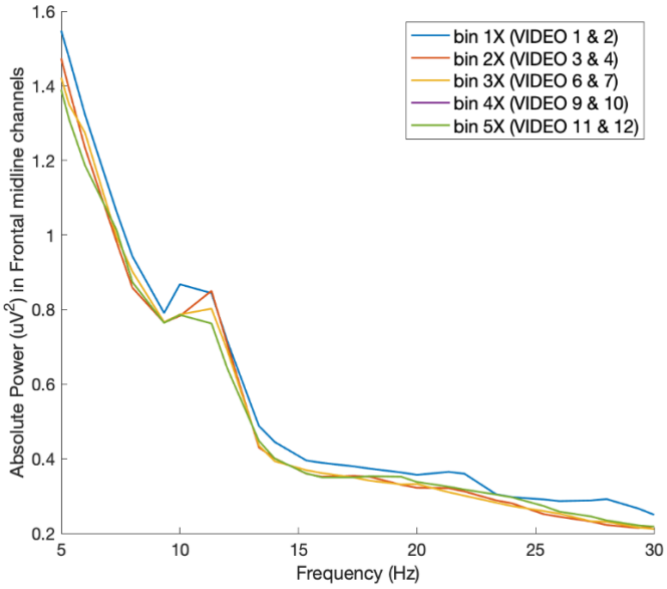


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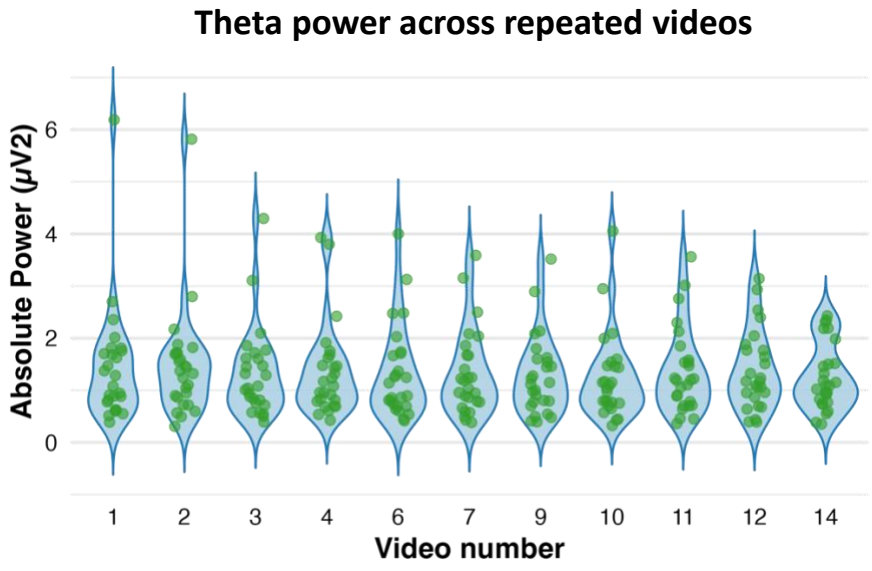


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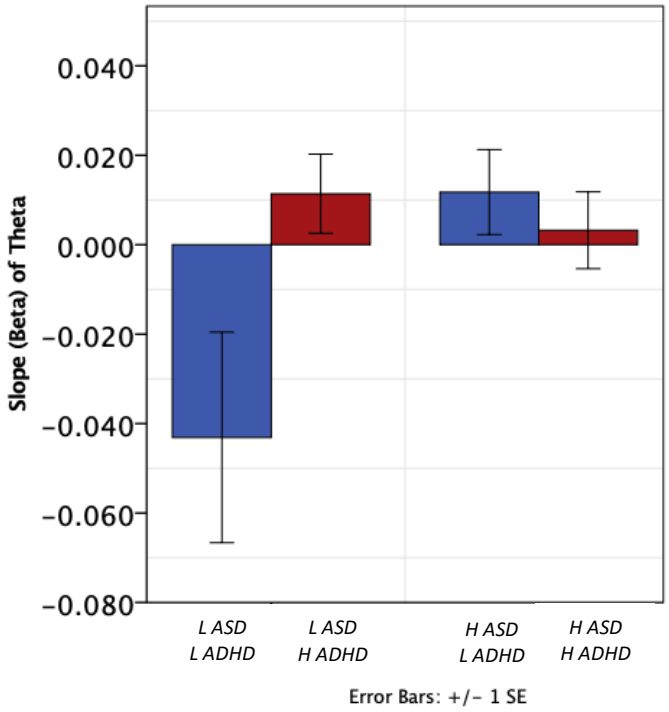



*(e)*

*Figure 3.1. (a)* Topographic representation of absolute theta band activity (4hz-8hz) across the whole scalp for combined time period of the first two repeated videos; video 1 and video 2. *(b)* Topographic representation for absolute theta band across the scalp for the two repeated videos appearing towards the end of sequence; video 11 and 12. *(c)* Broadband frequency signal from 4hz to 30hz in steps of 1 (i.e. 4.3, 5, 6.3, etc) in the frontal-medial channels of interest where the videos are combined into bins; each bin containing two of the repeated videos. *(d)* Violin plots showing change in theta band activity across repeated videos only. *(e)* Bar chart showing the **Betas** from the change across repeated videos for both theta band activity separated by ASD median split groupings and ADHD median split groupings.

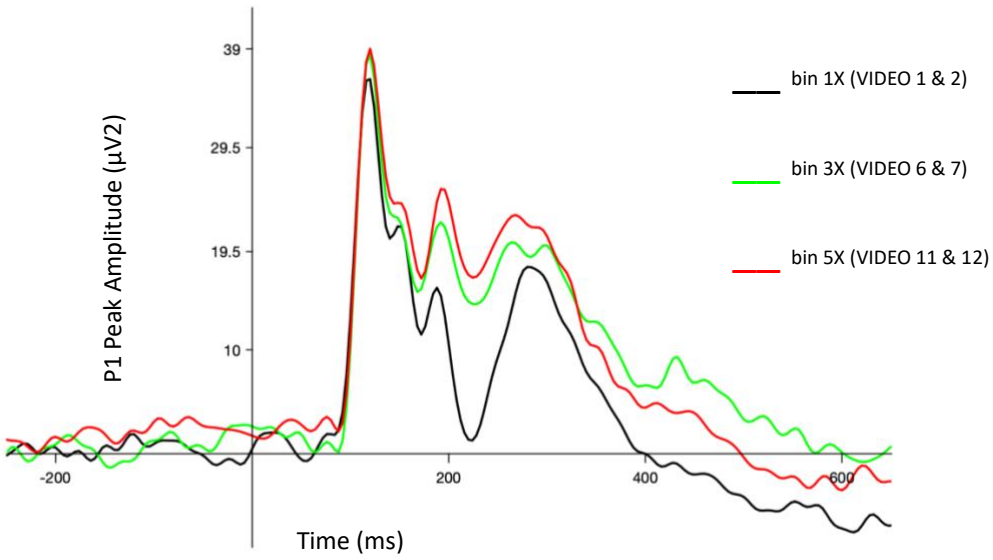



*Figure 3.2.* The ERP/VEP neural signal from the three parietal cluster (PO3, POz, and PO4) where videos are combined into three time-bins for where each time bin has 2 repeated videos for visualisation purposes.

### 3.3.4 Association between Theta-band change and P1-change

In the previous chapter P1 peak amplitude was analysed, however no notable findings were reported. We also have no specific hypotheses regarding change in P1 peak amplitude across the video repetitions, for this reason is has not been included in the analysis of this chapter. *Figure 3.2* is a representation of the average ERP waveform to confirm the presence of a robust peak P1 component which is used for further analysis below.

In a similar fashion to the previous chapter, **Beta** values (representing the slope of change) were extracted from regressions conducted on each participant's change in theta and P1 peak component. Unlike the previous adolescent sample however, the data does not show a significant association between participant's slope of theta and slope of P1 peak component ($r$ (23) = -.07, $p$ = .73).

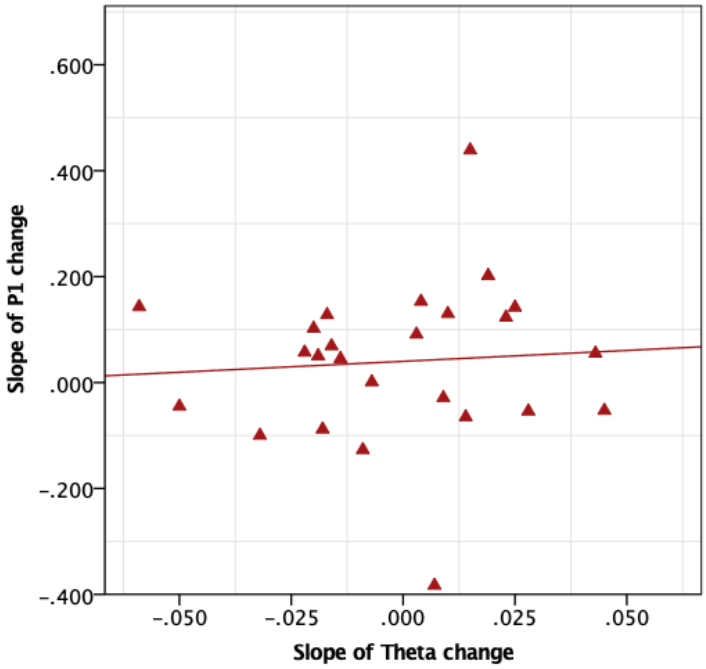



*Figure 3.3.* Represents the relationship between the slope of Theta change against the slope of P1 change. Negative scores here represent a negative change in theta/p1 (i.e. declining over time), numbers closer to 0 reflect a shallower slope whereas numbers further away from 0 reflect the slope becoming steeper. Here it seems like there is no relationship between slope of change in Theta and slope of P1.

### *3.5.1 Modulation of theta and P1 with individual differences*

Similarly to the adolescent sample, we computed the correlation between Theta power and P1 amplitude for each participant and then extracted the **Beta** of the correlation. This beta value is referred to as the Theta/P1 slope of modulation. A more negative beta indicates a clearer trade-off between attending to two different sources of information, and vice versa, a positive beta indicates less trade-off. Additionally, beta values closer to 0 indicate flatter slopes.

Finally, a regression model with the slope of modulation as the outcome and ASD and ADHD traits as the predictors reveals no significant effect overall ($F$ (3, 21) = 0.60, $p$ = .63). *Table 3.4* displays the Betas and significance values for each factor in the model.

*Table 3.4*

*Regression model with slope of modulation as an outcome and ASD and ADHD as predictors with an interaction element.*

| | *Estimate* | *Std. Error* | *T value* | *P* |
|---|---|---|---|---|
| **(Intercept)** | -.28 | 1.27 | -.21 | .83 |
| **ASD** | -.93 | 1.78 | -.52 | .61 |
| **ADHD** | -1.48 | 1.75 | -.85 | .41 |
| **ASD * ADHD** | 1.15 | .97 | 1.18 | .25 |

### 3.3.5 Novel video analysis

Novel videos appeared as the 5th, 8th, and 13th video in the order of sequence. First the average theta band activity in the novel video is computed (video 5, 8, and 13), followed by the average of theta band activity in the video preceding the novel video (video 4, 7, and 12), and then the average of theta band activity in the video proceeding the novel video (video 6, 9, and 14). The same was computed for P1 peak amplitude. Changes in theta power and P1 peak amplitude for novel videos in comparison to the videos preceding and proceeding it are presented in *Figure* 3.4.

A repeated measures ANOVA on the average power of signal for pre-novel, novel, and post-novel video reveals no significant change in Theta band activity ($F$ (2, 48) = 1.68, $p$ = .20), nor in peak P1 amplitude ($F$ (2, 48) = .07, $p$ = .93).

Theta power when controlling for ASD and ADHD traits also reveals no significant change across the three conditions ($F$ (2, 44) = 1.84, $p$ = .17). There were no main effects on theta band across the three presentations (pre-novel, novel, and post-novel) for ASD ($F$ (1, 22) = .02, $p$ = .91) nor ADHD traits ($F$ (1, 22) = 1.16, $p$ = .29). There were also no interactions between the three presentations and ASD traits ($F$ (2, 44) = .37, $p$ = .69), nor ADHD traits ($F$ (2, 44) = .48, $p$ = .62).

Similarly, no significant difference was seen in peak P1 amplitude when controlling for ASD and ADHD traits (($F$ (2, 44) = .12, $p$ = .89). There were no main effects on P1 peak amplitude across the three presentations (pre-novel, novel, and post-novel) for ASD ($F$ (1, 22) = .14, $p$ = .71) nor ADHD traits ($F$ (1, 22) = 2.21, $p$ = .15). There were also no interactions

between the three presentations and ASD traits ($F$ (2, 44) = 2.40, $p$ = .15), nor ADHD traits ($F$ (2, 44) = 2.09, $p$ = .14).

As discussed in the previous chapter, novel videos represent new information presented to the participants, however the neural signals are unaffected by novel information.

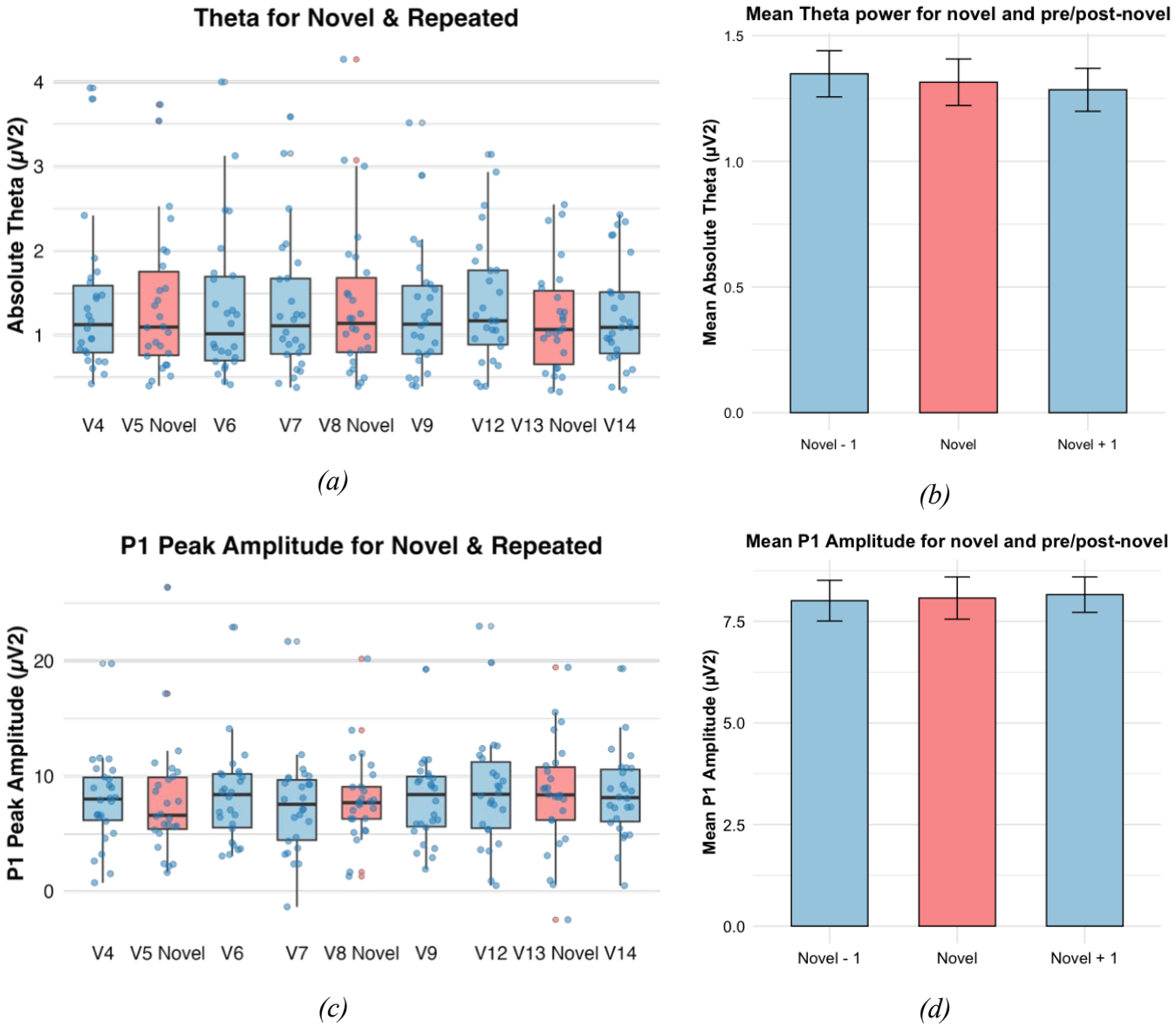



*Figure 3.4:* (a) and (c) Absolute theta and P1 peak amplitude across participants specifically for the three novel videos, and the videos preceding and proceeding the novel videos. Here video labelled 'V5 novel' 'V8 novel' and 'V13 novel' represent the novel videos. (b) and (d) total average theta and P1 peak amplitude for all three novel videos, and the videos preceding and videos proceeding it.

## 3.4 Discussion

The goal of this EEG study was to examine the role of individual differences in trait measurements of ASD and ADHD on neural signals that relate to engagement and disengagement with ongoing stimulation. This is a continuation from the previous chapter where we demonstrated that ADHD traits play a role in the modulation of theta/p1 slope and ASD likely playing a role in change in theta band activity over time. We hypothesised the same trends in this sample, although we measure subclinical traits in this sample instead of clinical measures used in the previous sample.

Contrary to the predictions, the findings from this study do not corroborate with the findings from the previous sample, instead of theta declining across the repeated videos, here we report that theta band activity remains stable across while the information from the videos is depleting. In the adolescent sample, this decline was also steeper in participants with lower ASD traits and flatter for participants with higher ASD traits, which is absent from our data. The results do however reveal a three-way interaction between median split groupings of ASD traits and ADHD traits have an impact on theta band activity across the repeated videos. Even in this small sample, from *Figure 3.1e* we see that participants who are characterised as low ASD and low ADHD, exhibit a steeper slope, representing a slope that we initially expected to see across the entire sample. Although the slope of the low ASD low ADHD group was not different from chance (one-sample t-test), there was a notable trend, approaching significance in the statistical test. Interestingly, this three-way interaction was not present in the adolescent data.

A key hypothesis of this study was that theta band activity is inversely correlated with P1 peak amplitude. This was present in the previous sample and not present in this sample. Additionally, there was no relationship between the theta/p1 modulation and ASD nor ADHD traits.

There may be several reasons why we do not see the expected results. Firstly, in comparison to the sample recruited in adolescents, this one is considerably smaller (39 in adolescents and 25 in adults). Additionally, a reason there might not be significant decline in theta band power – which indexes engagement – may be due to overall lack of engagement of the adult participants with video material. The short clips from *Fantasia* might be suited to infants (Piccardi et al., 2021), and in adolescents, however it may be the case that participants

never fully engaged with the videos and therefore progressive decline in engagement was not apparent in the neural signal.

It is also worth noting that in this sample we utilized subclinical measures of ASD and ADHD traits, and although we received a diverse range of responses, subclinical measures do not necessarily map onto clinical measures. This has been discussed with regards to ASD in recent work by Banker et al., (2025) where they used the BAPQ and the ADOS in two matched samples of participants and found that there was a significant discrepancy between scores in the samples reporting that self-report subclinical ASD. They found that surveys do not correlate with clinician-rated autistic traits and that these two measures may be capturing different aspects of ASD. Additionally, there has been research suggesting that poor sleep habits, and misalignment with social sleep-wake schedules may exacerbate symptoms of inattention, impulsivity and unstable temperaments – key traits measured in ADHD subclinical surveys (Ottoni et al., 2012; Park et al., 2015). Generally, university students – the sample we recruited – commonly experience sleep deprivation or poor sleep (Lund et al., 2010; Stores et al., 2023) which may contribute to higher scores on ADHD subclinical measures without necessarily reflecting underlying neurodevelopmental differences. The use of subclinical measures may explain why our adult sample results diverge from the adolescent study which utilised clinical measures such as ADOS-2 and Conners-4.

High co-occurrence of ADHD and ASD traits in adult non-referred sample has also been reported previously (Taylor, Charman & Ronald, 2015; Panagiotidi, Overton & Stafford, 2019). Panagiotidi, Overton, and Stafford (2019) also report that – unlike clinical samples where differences between ASD and ADHD traits are more apparent (Pehlivanidis et al., 2021) – there is a large overlap between ADHD and ASD traits; especially as it relates to attention and communication skills. There has also been large scale network analysis in young people (including adolescents) that show that ADHD and ASD to be distinct and segregated with little interconnectivity between symptomatology (Farhat et al., 2021). The use of self-report measures assessing traits that relate to autism and ADHD may be another limitation in this design. There is likely to be a response bias that could account for high levels of co-occurrence of ADHD and ASD traits. It can be argued and has been argued previously by other research (such as by Panagiotidi, Overton, & Stafford, 2019) that individuals who report abnormal behaviour in one modality are also likely to report abnormal behaviour in another. There is little that can be done in order to improve the limitations of self-report measures except for the use of more clinically appropriate behavioural measures

such as ADOS-2, although this is a more time-consuming administration, taking up to 1 hour to administer versus 10 minutes on a self-report survey measure. Nonetheless, it may be the case that in non-clinical adult populations, there is less separation between ASD and ADHD traits, and rather, these may be related to an overarching modality of neurodiversity.

Furthermore, mirroring the discussion point from the adolescent sample, where high-ASD group did not have a typical decline in theta, and this being a potential reason there was no effect of ASD traits on theta/p1 modulation, the same point can be made here for the entire sample. Where there is no typical decline in theta band activity across the sample, and the foundation for the theta/p1 modulation is that there should be a trade-off between theta and p1 activity. It is possible that in an alternative paradigm where a linear decline in theta is present, that the trade-off between this decline, and subsequent increase in P1 amplitude would also be present. In other words, if we are able to simulate engagement and subsequent disengagement from ongoing stimulation, then we may be able to see where individual differences may impact this engagement/disengagement relationship.

### 3.4.1 Limitations

Several limitations of this study should be acknowledged when interpreting the findings. The most apparent limitation is the sample size (N= 25) which is considerably smaller than the adolescent sample (N= 39), which has significantly reduced statistical power and increasing the risk of Type 2 error. This may be why some of the correlations we reported in the adolescent sample, were not found in this sample. Information on statistical power is reported in **subsection 3.2.3** in this chapter where it is noted that approximately 65 participants would be required to detect the effect sizes observed in the adolescent samples.

Secondly, the use subclinical self-report measures (AQ, BAPQ, ASRS, WURS) instead of clinically validated instruments is a significant difference between this study and the previous study, which likely contributed to the lack of individual differences effects on the data. Recent work by Banker et al., (2025) found that there was a significant discrepancy between clinical instruments and online survey scores. This suggests that these measures may be capturing different aspects of the participant's neurocognitive/neurodevelopmental profiles. The use of university student populations who may be presenting with poor sleep habits could exacerbate symptoms of inattention and impulsivity (Ottoni et al., 2012; Park et al., 2015) without having the underlying neurodevelopmental differences that associate with inattention and impulsivity.

Lastly, as mentioned above, the stimuli may have been developmentally inappropriate for adult participants. While these clips were effective in engaging infants (Piccardi et al., 2021) and adolescents, they may have failed to sustain adult attention, potentially explaining the absence of the expected linear decline in theta band power across repetitions. It is possible that adult participants never fully engaged with the video material, which would preclude observing the predicted engagement-disengagement trade-off.

### 3.4.2 Conclusion

This study explored how subclinical ASD and ADHD traits modulate neural markers of attentional engagement (theta) and disengagement (P1) in adults, building on prior findings in adolescents. Contrary to hypotheses, we observed stable theta power across video repetitions and no theta/P1 trade-off, diverging from the adolescent sample. The marginal three-way interaction between ASD, ADHD traits, and theta change suggests these traits may interactively shape engagement dynamics, potentially counterbalancing each other which aligns with proposals by Landry (2021). Although it is important to note that limitations such as the use of subclinical measures that may not reflect neurodevelopmental traits, low power, and stimuli potentially unsuitable for adults, caution against overinterpretation. Nonetheless, these results highlight that neural correlates of attention may manifest differently in subclinical adult populations compared to clinical adolescent groups. Moreover, these results emphasise the need for future work to use more robust clinical measures, engaging age-appropriate paradigms, and larger samples to disentangle trait-state confounds and developmental trajectories.

# CHAPTER 4:

## 4. Attentional disengagement in response to information gain in adolescents with Autism and ADHD traits

### Goal of chapter

*The overarching goal of this chapter is to investigate the fundamental attentional process of visual disengagement through the theoretical lens of information foraging. While the previous chapters employed neural signals to measure changes in engagement and information seeking, this study uses eye tracking to directly measure disengagement and disengagement costs associated with orienting away from stimuli of varying informational affordability. The core premise drawn from the works of Posner (1982), Gottlieb & Oudeyer (2018), and Manohar & Husain (2013), is that attention operates as a dynamic sampling system, continuously weighing the informational value of a current stimulus against the potential value of novel information elsewhere. Effective information sampling is therefore reliant on the attentional processes of disengagement and re-orientation.*

*We adapt and use the classic gap-overlap paradigm, a well-established tool for measuring disengagement costs. The critical adaptation we implement is the manipulation of informational value of the central stimulus from which the participants must disengage. By unpredictably presenting images (1 to 4 times), we simulate stimuli that is progressively declining in novelty and increasing in familiarity. The image that the participants must orient towards is always novel, ensuring that participants are orienting towards high-informational content, away from a content that is progressively declining in information.*

*We predicted that disengagement cost (the gap effect) in this experimental design would be highest when leaving novel stimulus and would decrease as central stimulus becomes familiar. Additionally, that this progressive decline would be modulated by individual differences in ASD and ADHD traits. We successfully executed this novel paradigm, and our findings here support our initial hypothesis of progressive decline in disengagement costs, however the role of individual differences is less clear. Our results show differences in disengagement for high scoring ASD participants, indicating persistence with familiarity. However, for the most part, the findings here point towards largely similar attentional disengagement in the face of changing informational value in adolescents with varying ASD and ADHD traits.*

# 4.1 Introduction

## 4.1.1 Background literature

Posner and Petersen's model of attention describes alerting, orienting, and executive control as three distinct but collaborative networks (Posner & Petersen, 1990; Petersen & Posner, 2012). As we and others (Manohar & Husain 2013; Gottlieb & Oudeyer 2018; Gottlieb et al., 2013; Bloomberg-Martin & Hikosaka 2009) have argued, the attentional system acts as an information sampling mechanism, permitting individuals to move between different objects in the environment to gain information about their locations, dangers, and affordances. The visual orienting system is placed at the centre of this account, shown to operate as an active decision-maker, whereby the brain selects specific regions to process and others to inhibit (Gottlieb & Oudeyer 2018). From infancy, humans use eye movements to explore their environment, and quite early develop the capacity to disengage attention and look away from salient stimuli (Elsabbagh et al., 2008; Johnson, Posner & Rothbart, 1991). Although it is not always the case (Beck & Lavie, 2005; Handen & Arntzen, 2015), humans do typically *look at* what they are *attending* to, and eye movements therefore, act as a reliable window into measuring orientation.

Attentional mechanisms such as orientation also provide a critical foundation for the emergence of higher-level cognitive skills (Keehn et al., 2012; Sacrey et al., 2014) and are thus heavily implicated in information foraging accounts. Posner (1982) outlined the three stages involved in orienting, (1) disengagement from the current stimulus; (2) movement to the new location; and (3) re-engagement with the target at the new location. According to this model, an individual must disengage first from the current stimuli in order to facilitate engagement with another. These processes involve multiple brain processes, each contributing to the overall processing time (Posner & Petersen 1990).

When processing our surroundings, unique aspects of a scene or certain types of motions or colours tend to attract an observer's attentional focus (Chastain, Cheal, & Kuskova, 2002; Franconeri & Simons, 2003). The ability to seamlessly disengage from one stimulus and orient towards another is critical for effective information seeking and determines how flexibly an individual can sample their environment for relevant information (Elsabbagh et al., 2008). The forging account of attention posits that individuals gather information from one source until a threshold is reached where the subsequent energy required to extract additional information outweighs the value of the information, and it is at

this point when individuals would search or re-orient elsewhere (Hills et al., 2015; Manohar & Husain 2013; Calhoun & Hayden, 2015). This account is also in-line with proposals by Gottlieb and Oudeyer (2018), and other works (Gottieb, 2023; Gottlieb et al., 2014) where attention is described as a form of information sampling in the environment; specifically, that the brain must make decisions regarding areas in scene to select and extract information relevant to the individual. Thus, it can be said that visual orienting – described as the ability to disengage from an existing point of focus, make a saccade to a new stimulus, and engage with the new stimulus (Posner 1982; Johnson et al., 1991) – serves as the behavioural mechanism through which individuals sample information in their environment.

Findings from previous research generally demonstrate support for this account. Tagu and Kristjánsson (2020) report visual orienting to be dynamic and context-dependent, also revealing the utility of using eye-movements to tap into different forms of visual orienting such as differences between feature-based foraging (frequent switching between target types) and conjunction-based foraging (exploiting one target before switching to another). It is also established that stimuli associated with uncertainty results in higher visual engagement (Yu & Dayan, 2005; Hogarth et al., 2008) and that humans and animals engage in information-seeking, even at the expense of reward (Bromberg-Martin & Hikosaka, 2009; Behrens et al., 2007). New information is considered valuable even when not immediately useful to the context (Gottlieb, 2012; Kidd & Hayden, 2015). In a majority of these experimental set ups, an individual is engaged with one source of information, which is followed by disengagement with the source of information, then transfer (or orientation) to another source of information.

In the previous chapters, we measured information seeking in a dynamic experimental paradigm where neural signals and modulations of neural signals indexed information seeking. In that account, participants split their attention between an ongoing-depleting source of information and an incoming-distractor. ADHD traits specifically played a role in modulating the relationship between engagement with ongoing stimulation, and disengagement with ongoing stimulation (and thus engagement with incoming stimulation). This informs us that the relationship between engagement and disengagement with a dynamic, and depleting source of information changes over time, and that it can be modulated by individual differences. This however does not inform us about the role of foundational attentional processes such as visual orientation and its role in information gain and attention switching.

Effective disengagement is the first stage of visual orientation. Both the speed and the timing of attentional disengagement can reveal insights into how individuals might be sampling their environment for information. Disengagement from one object or location that is too slow, limits the individual's ability to effectively scan the environment, update their information state, and is ultimately costly if there is potentially valuable information elsewhere. On the other hand, disengagement that is too fast, results in insufficient gathering of information from the original source. Indeed, slower disengagement in eye tracking tasks has been reported for individuals with anxiety (Fox et al., 2001; Mogg et al., 208), individuals with autism (Landry & Bryson, 2004 in children and Kawakubo et al., 2007 in adults), individuals with Alzheimer's (Parasuraman & Greenwood, 1998), and slow disengagement generally as a result of ageing (Polden, Wilcockson & Crawford, 2020).

Research by Brockmole and Boot (2009) used a novel search paradigm measuring oculomotor disengagement from central stimuli towards target and distracter stimuli surrounding the central stimulus. They found that saccadic latency to a peripheral stimulus was delayed when the centre stimulus was novel, i.e the disengagement cost was higher. Interestingly, in a follow up study Boot and Brockmole (2010) reported that attentional disengagement was also delayed when the centre stimulus was the same colour as the target. In other words, when there was no novelty or new information in the peripheral stimulus in comparison to the central stimulus, the disengagement cost is higher.

### 4.1.2 The proposed study

A paradigm that is often used to study attentional disengagement is the gap-overlap paradigm. This paradigm has been used in primates (Kano et al., 2011) and in human children and adults (Sacrey et al., 2014). Here participants experience two conditions; the gap condition, in which a central stimulus is presented for a period of time, typically proceeded by a momentary 'gap' where there is no stimulus on the screen, followed by presentation of a stimulus somewhere in the periphery. The second condition is the overlap condition, where the central stimulus is presented followed by a period where the central stimulus and peripheral stimulus are 'overlapped' in presentation. Instructions given to participants vary (Sacrey et al., 2014), but generally participants are asked to direct their gaze to the stimuli as soon as they are presented. From this paradigm, a *gap effect* is extracted which is the difference between mean saccadic reaction time (SRT) of the gap and overlap condition. The gap effect is taken to measure the disengagement part of the attention shift process, which is

present in the overlap trial but not in the gap trial, where participants shift gaze from a blank area on the screen to periphery, while accounting for differences in movement to the new location and re-engagement (identical in the gap and overlap conditions). Variations of different implementations of the gap-overlap paradigm are presented in *Figure 4.1* and *Figure 4.2*. The gap-overlap paradigm is also referred to as the pro-saccade paradigm, which is the opposite to the anti-saccade task, where participants are instructed *not* make a saccade towards certain targets or location and acts as a measure of inhibition.

This paradigm has been used frequently to assess the cost of visual disengagement and has been used in ASD and ADHD populations yielding inconsistent results. Some studies in ASD populations have demonstrated impairments in disengagement in children and adults with autism (Landry & Bryson, 2004; van der Geest et al., 2001; Elsabbagh et al., 2009), and this is also demonstrated in EEG studies (Kawakubo et al., 2007). These findings are in line with developmental accounts which suggest that problems in visual orienting are likely to be present very early in autism (Bryson et al., 2004). Impairments to attention related mechanisms are often considered to associate with ASD (Keehn et al., 2013). These attentional abnormalities are thought to underlie several core features of ASD, including inflexible behaviour (Landry & Bryson 2004; Casey et al., 1993), difficulty with joint attention (Schietecatte et al., 2012; Morales et al., 2000), as well as impairments in emotional regulation (Bryson et al., 2004). Studies conducted by Fischer et al., (2014) in toddlers, and Fischer et al., (2016) in children and adolescents, on the other hand, report unimpaired attentional disengagement. Studies that have used adolescent participants with autism of average intellectual ability also report no significant differences in attentional disengagement (Goldberg et al., 2002), and no differences in adults when comparing to IQ-matched controls (Kawakubo & Maekawa, 2005; Mosconi et al., 2009).

Wilson and Saldaña (2019) used the gap overlap paradigm in children, using both dynamic and static stimuli. They report that the ASD group had a significantly larger overall disengagement cost compared to the typically developing group however there were no differences relating to the stimuli type (i.e. whether the stimuli were moving or static, or whether they were low contrast or high contrast). An important note they make is that participants with ASD had slower SRT in the gap trials, but comparable SRT in the overlap trials and therefore the gap effect was inflated. They concluded that while visual attention mechanisms may differ significantly in ASD children, this is not explained by slower overall

disengagement, but rather due to fixation offset cues. Disengagement effects were also not predictive of cognitive abilities nor of sensory behaviours or severity of ASD symptoms.

Fischer et al. (2014; 2016) use a unique paradigm incorporating novel stimuli at every trial. The first study in 2016 was conducted in children aged between 5 to 12, where – without being given explicit instruction to do so – were orienting away from various non-social and social images that were presented in the central location. All the images used in this study were used once and not used again. The results of their study demonstrated that attentional disengagement and social orienting between ASD and non-ASD groups were strikingly similar. A follow up study in toddlers in 2014, using the same experimental set up also revealed the same results.

In young children aged 5-11 with and without ADHD, Matsuo et al., (2015) found that overall the SRTs for individuals with ADHD was slower, however the gap effect was attenuated. In other words, the disengagement cost was lower in individual with ADHD (smaller difference between gap and overlap trials), but that across all trials, their reaction time was slower overall. Another study which used both pro-saccade and anti-saccade conditions, reported that children with ADHD had matched SRT and matched gap effects to typically developing children in the pro-saccade condition (Hanisch et al., 2006).

Past research has used variations of the gap-overlap paradigm that are presented in *Figure 4.1* and *Figure 4.2*. The variation in testing conditions presents a challenge in interpreting and comparing findings across research as there is no standardised application of the gap-overlap paradigm. Especially with the case of 'gap' conditions, for example Fischer et al., (2014; 2016) and Mo et al., (2019), employ a version where there is no *gap* between the presentation of the central stimulus and the peripheral stimulus, instead they use a 'step' condition where the peripheral target is presented without any temporal gap. Another example of a discrepancy in approaches is with timings and durations of presentations. In Fischer et al., (2014) the duration of presentation for central image is randomised such that it is presented for 1 or 2 seconds in order to offset anticipatory saccades, in the study by Mo et al., (2019), the central image is presented for 1 second, and in Landry and Bryson (2004), 8 seconds is used. Moreover, Landry and Bryson (2004)'s paradigm consisted of three separate computer monitors wherein stimuli were presented and participants therefore, made relatively large distance eye movements in order to shift between central stimuli and peripheral stimuli. The images or stimuli used in studies also significantly vary, from dynamic engaging stimuli

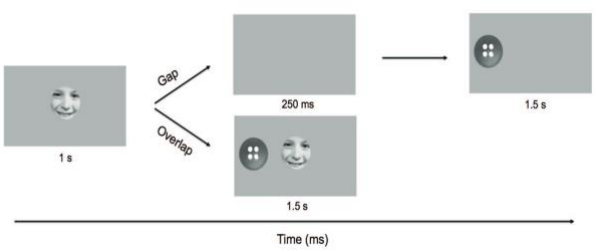
(Wilson & Saldaña, 2019) to novel images of objects and items (Fischer et al., 2014; 2016), to simple shapes (Hanisch et al., 2006).

Such methodological variation, also identified in a review by Sacrey et al. (2014) pose difficulties when comparing results across studies and therefore the underlying cognitive processes. This is particularly problematic when we look at this paradigm from the framework of attention as a mechanism of information sampling in the environment, where how long one had to sample information from the central stimulus and the amount of information available, may have an important impact on performance. To our knowledge, there has been no attempt to manipulate information gain in the gap-overlap paradigm. In our adaptation of the task, the central stimulus varies in novelty, while the peripheral stimulus is always novel and unique. Similarly to how we simulate information depletion in the previous EEG chapters by repeating stimuli, we use the same method by repeating images appearing in the central position of the paradigm 1 to 4 times. Images may be repeated twice, thrice, four times, or never repeated. This gives us stimuli that is progressively reducing in informational value; so participants are disengaging and making saccades – in overlap conditions – from images that they are familiar with and images they are not familiar with (images in the gap conditions have similar repeating and novelty patterns, however at the point of orientation, participants are technically disengaging from no-information). This design allows us to test whether disengagement costs differ based on the informational properties of the stimulus participants must leave behind for novelty. We design our paradigm incorporating relevant aspects of design from previous literature. For example, the use of a true 'gap' where there is a delay between the offset of central and subsequent onset of peripheral stimuli. Although disengagement from a 'step' condition is expected to be faster, having a 'gap' in presentation allows there to be a larger informational-value difference between the conditions at the point of disengagement. In addition, we use stimuli consisting of objects and common household items, making it more engaging than simple shapes on a screen. We use standardised 1 second as the presentation time throughout, with 300ms as a gap, and 300ms as the overlap time (see *Figure 4.3b* for illustration).

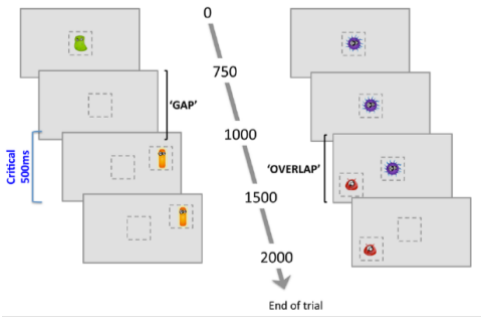
The number of times an image is repeated (0 to 3 repetitions, or 1 to 4 total presentations) was unpredictable. Each presentation of an image is labelled according to its presentation history: the first presentation (R1), the second (R2), the third (R3), and the fourth (R4). There are also images that are presented once, and never repeat which are labelled accordingly (U). The proportion of trials in each of these four states (U, R1, R2, R3,

R4) in the entire experiment is illustrated in *Figure 4.3c* in the **Methods** section of this chapter.

When humans shift their gaze from one location to another via saccade, competition occurs between two mutually exclusive activities: fixation at the original location, and making a saccade to an alternative location, and this is resolved using various perceptual and cognitive processes (Findlay & Walker, 1999). The gap-overlap paradigm measures the efficiency of disengaging from one location in order to orient towards another, specifically, disengaging from the central stimulus to orient towards a peripheral one. We aim to examine whether the information *value* of the central stimulus i.e. the informational value, modulates this process. If attention operates as an information-foraging system, disengagement from a novel central stimulus should be slower, as novelty sustains engagement (Brockmole & Boot, 2009). Conversely, disengagement from a familiar stimulus should be faster, as there is less information available to be acquired from an image that participants have viewed multiple times. By manipulating central stimulus novelty while keeping peripheral stimuli consistently novel, we can isolate the influence of information potential on disengagement costs.




*(a)*

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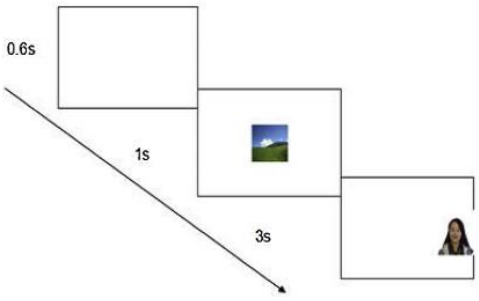
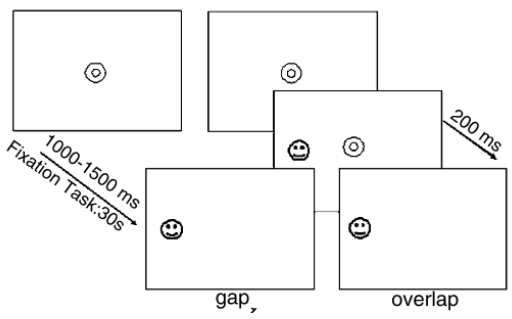



*(c)*




*(d)*

*Figure 4.1.* These figures are adapted from various experimental set ups in research using the gap overlap paradigms. *(a)* The paradigm used by McLaughlin et al., (2021) wherein there is a temporal gap between offset of central and onset of peripheral stimulus, additionally here, in the overlap condition, the peripheral stimulus does not persist beyond the overlap period. *(b)* This set up is from Wilson and Saldaña (2019), and here there is also a temporal gap in the gap condition, however in the overlap condition, the central image is displayed for 1500ms, which is 750ms longer than central image displayed in the gap condition. *(c)* This is the step-condition used in the study by Mo et al., (2019) where there is no temporal gap between the offset of the central stimulus and the onset of the peripheral stimulus. *(d)* This is adapted from Hanisch et al., (2006) and uses a paradigm similar to our design, however they used simple shapes and line drawings as stimuli.

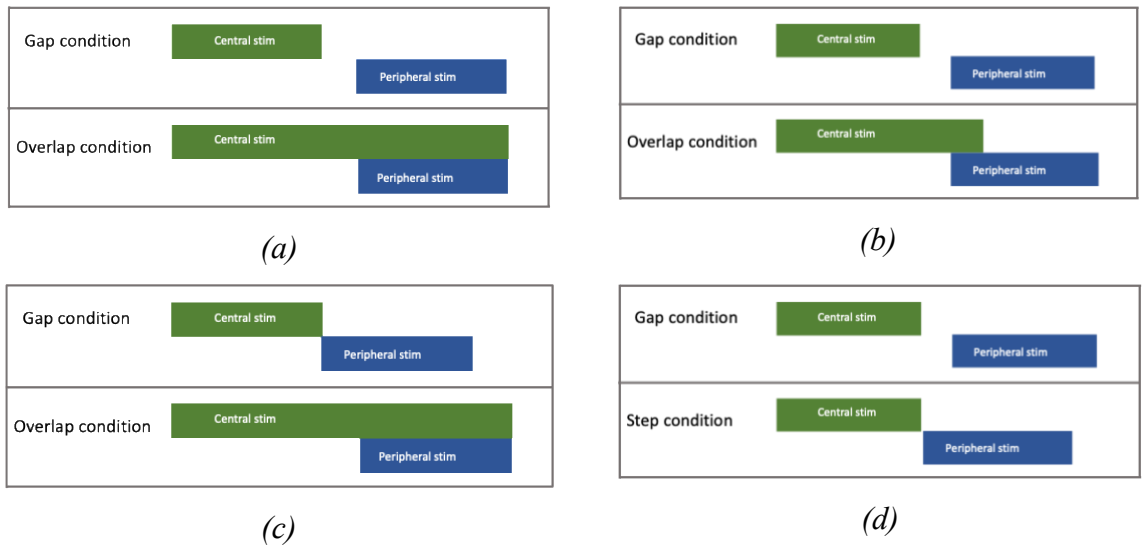



*Figure 4.2*. Here we have created visualisations of experimental paradigms mentioned above. (a) This represents the paradigm used by McLaughlin et al., (2021). (b) Represents the paradigm used by Wilson and Saldaña (2019). (c) Is the gap/step and overlap condition used by Mo et al., (2019). (d) These are the two conditions used by Matsuo et al.., (2015) where they compared between gap and step conditions in children with ADHD.

Attentional profiles – as explained in chapter 1 of this thesis – in Autism Spectrum Disorders (ASD) and ADHD present seemingly opposing characteristics that can be unified under a neural gain framework. In the work by Landry (2021), differences in attentional

profiles in ASD are characterized by hyper-focus, reduced distractibility, and resistance to disengaging from current stimulus or tasks. These traits align with high-neural gain profile, the opposite of low-neural gain profile described in detail by Hauser et al., (2016). According to this model, heightened gain amplifies signal differentiation, promoting stability in behaviour and choices. ADHD on the other hand, is described by Landry (2021) as having increased distractibility, frequent attentional shifts, and high behavioural variability, features consistent with a low-neural gain profile described by Hauser et al., (2016). Reduced neural gain has attenuated signal amplifications, impaired suppression of noise, and therefore variable responses and unstable goal allocation. These divergent profiles reflect fundamental differences in how the brain arbitrates between exploitation (stable selection), and exploration (variable selection), with ASD biased towards rigid, restrictive behaviours and ADHD towards excessive switching (Landry 2021).

The neural gain model offers testable predictions for attention disengagement in the gap-overlap paradigm where we manipulate the information gain of the stimuli participants disengage from (i.e.: the stimuli appearing in the central location). We hypothesise that for the first presentation of the image, the GE will be high, followed by reduction of GE across subsequent repetitions. This indicates higher disengagement costs for images that are novel, and subsequent decreasing of disengagement cost as familiarity (indexed by repetitions) to the image increases. We expect that this decline in GE will occur independent of any overall changs in SRT (i.e. reaction time) across a block of trials.

Furthermore, we hypothesise that changes in disengagement cost across repetitions will be modulated by individual differences in ASD and ADHD traits. We expect higher disengagement cost to persist until the later repetitions for participants that scored highly in their ASD traits, indicating either: higher threshold required to reach peak information gain, or preference for familiarity and thus difficulty orienting gaze away from the familiar image. On the other hand, we expect participants that scored highly in ADHD traits to have a steeper decline in disengagement cost after R1, indicating lower threshold required to reach peak information gain and therefore gaze is readily disengaged in pursuance of novelty in the peripheral image.

### 4.1.3 Preliminary testing

Prior to experimental testing, a preliminary version of the task was administered to a group of 14 participants made of colleagues at work to assess feasibility and confirm the

presence of a gap effect under the proposed experimental set up. This pilot employed the same gap-overlap paradigm, however the central stimulus repeating up to three times only; so experimental conditions were U, R1, R2, and R3. In addition, the preliminary paradigm had a selection of central images that do not repeat and a selection of images that repeat three times. In this preliminary pilot, every image that was a repeating image, repeated three times, and images that were not repeating did not repeat at any time. Half the participants were given an additional instruction where they were asked to press a button when images in the central position and peripheral position were matched.

While the results confirmed the expected gap effect – demonstrating slower disengagement in the overlap condition in comparison to the gap condition – the observed variation in disengagement costs across repetitions was less pronounced than expected. We also found that the gap effect for the 3rd repetition significantly increased. This may have been due to participants' knowledge of this image being the final presentation of the set (since all repeated images repeated 3 times). Finally, the results showed generally faster SRT when participants were given button press instructions.

To ensure the task captured a broader range of disengagement behaviour and better differentiate the effects of stimulus familiarity, a fourth repetition was added (R4) into the final version of the paradigm, and the central stimuli were organised to repeat unpredictably (so some repeated twice, and some other images repeated thrice, and some four times). This design ensured that participants could not anticipate the informational trajectory of a given stimulus at the time of presentation, preventing any impacts this could have on disengagement. Based on our pilot, we still expect that R4 will elicit the largest gap effect, due to being the final presentation – instead of relating to informational value or content – and for this reason it will be excluded from the analysis.

In summary, the preliminary testing phase validated the basic feasibility of the paradigm and underscored the potential importance of unpredictability in repetitions. The addition of a 4th repetition allows us to keep and use all three repetitions of the image without the R3 being artificially elevated simply due to participants recognising it as the final presentation. In the final paradigm therefore, we are able to preserve more trials (as we can retain all R3 trials and exclude R4 trials instead) and record a sufficiently long trajectory of change in disengagement, thus allowing for variation to emerge.

# 4.2 Methods

## 4.2.1 Participants

Forty adolescent participants took part in this Eye-tracking study (13 females, M age = 14.1, SD age = 1.05). Three participant's data were not included in the final analysis due to excessive data loss from the eye tracking recording, where either gap or overlap conditions had fewer than 20% of retained trials. The total number of participants included in this report are therefore Thirty-seven (12 females, M age = 14.1, SD age = 1.1).

See Chapter 2: **subsection 2.2.1** for remaining information about this adolescent population recruitment, we use the same sample of participants for this eye-tracking study.

## 4.2.2 Design

This study employed a repeated measures design and used individual differences measures of ASD and ADHD on continuous scales. The within-subject independent variables were condition (gap vs. overlap), and stimulus presentation (U, R1, R2, R3). Additionally, individual differences scores in ASD and ADHD traits were treated as covariates and between subject independent variables. The dependent variables were saccadic reaction time (SRT) – which is defined as the time it takes for the saccade to initiate from the central location towards the peripheral image from the time the peripheral image is presented – and gap effects (GE) – which the difference between SRT in the gap condition and the overlap condition.

The main manipulation here is the information gain from the central image; the image from which participants must disengage from. Participants were disengaging from one of 5 potential categories of images. First, unique image (U trials), which are images that are presented once and never appear again throughout the experiment. Secondly, from the first presentation of an image that will be repeated (R1 trials), which are images that are presented and will be repeated either two times, three times, or four times. Thirdly, from an image that is presented a second time, these are images that are from 1R trials, in their 2$^{nd}$ presentation (R2 trials). Fourthly, an image that is presented a third time (R3 trials). Finally, an image that is presented a fourth time (R4 trials). It is important to note that the repetitions of images are unpredictable, meaning that not all images are repeated an equal number of times; R1 trials are always repeated, but some are repeated only until R2 trials, and some until R3 trials, and some until R4 trials. This is to offset impacts of predictability, and knowledge about how

many times they will see the same image. This was the same in gap conditions and overlap conditions, and the stimuli that were used were only used according to their status of being U, R1, R2 etc, in other words, an image that had already been presented in as R4 was not used again for any other trials, and this was the same for gap and overlap conditions.

### 4.2.3 Materials

*4.2.3.1 Tests of individual differences*

See Chapter 2, section 2.2.3.1

*4.2.3.2 Stimuli*

The stimuli comprised 180 images freely available from the stimulus library of the Dynamic Perception Lab at Johns Hopkins University (https://dynamicperceptionlab.com/) as part of this experiment. Due to the number of trials, we supplemented with 14 additional freely available images, downloaded from Google. In total for this experiment, we used 194 non-social images of everyday objects and household items. The size of the images was 800px x 800px.

The presentation of stimuli in each 33-trial block was pseudo-randomized, specifically so that the stimuli appearing in the central location were appropriately interspersed within the block. The aim was to randomize the sequence such that any repetition of an image, did not occur too close (not occurring right after the previous presentation), or too far (no more than 7 trials away from the previous presentation) in the trial sequence and that the trials are equally interspersed with U trials. All the images appearing in the peripheral location were randomised and those images only presented once throughout the entire experimental session. A second sequence set was created where the repeated and novel images were swapped, participants were randomly allocated between the sequence sets and counterbalance orderings.

As for the number of trial types, in the entire experiment – across 4 blocks – there were 32 instances of U trials, 32 trials of R1 and 32 of R2 (because an image can only be categorised as R1 if it is going to be repeated at least once more). From these R2 trials, 24 images are repeated for a 3$^{rd}$ time as R3, and from that, 12 images are repeated for a 4$^{th}$ time as R4. This is 132 trials in total across the experiment. This is also illustrated in *Figure 4.3c*. Therefore, in each block of 32-trials, U, R1, and R2 trials each occur 8 times, and there are 6

trials which are R3, and 3 trials which are R4. Important to note that R4 is not used in analysis due to exhibiting large disengagement cost and due to few trial numbers.

*4.2.3.3 Apparatus*

The experiment is designed in SR Research Experiment Builder (SR Research Ltd. 2013) and eye-tracking data were collected using a desk-mounted infrared EyeLink 1000 Plus eye-tracker at 500hz. The stimuli were presented on a 17-inch LCD monitor (1280 x 1024 pixels 32 bits per colour) at a screen refresh rate of 60hz. The participants were seated at approximately 50cm from the monitor with their chin resting on a secured chinrest.

### 4.2.4 Eye-tracking analysis

To measure attentional disengagement costs, we measure SRTs, defined as the time between the onset of the peripheral stimulus and the first initiation of saccade from the central stimulus towards the peripheral stimulus that landed in the correct interest area of where the peripheral stimulus appeared.

We cleaned the saccade report generated by SR Research Data Viewer accordingly to several quality criteria. (1) We included trials where the first saccade started from the central fixation, towards the peripheral stimulus and landed within the bounds of the target image. (2) Trials with SRT lower than 80ms (representing a saccade that is too fast, and most likely an anticipatory eye movement) and SRT that is 2 standard deviations above the mean of the participant (representing disengagement that is abnormally slow) were excluded. Our decision to use 80ms as the anticipatory saccade cut off was informed by Matsuo et al (2015) where they used 70ms as the threshold for anticipatory saccades and Wilson and Saldaña (2019) where they used 100ms as the threshold; this range is the typical anticipatory saccade cut off in pro-saccade gap-overlap like paradigms across species and most ages (Pare & Munoz, 1996). (3) Lastly, we excluded participants with more than 80% of data loss due to cleaning per condition (in either gap or overlap conditions).

*Table 4.1*

*Overall data retained after removing invalid trials in ASD and non-ASD sample*

| | ASD n = 19, Female = 6 | | | Non-ASD n = 18, Female = 6 | | |
|---|---|---|---|---|---|---|
| | *% trials retained* | *No. trials* | *Std. Dev* | *% trials retained* | *No. trials* | *Std. Dev* |
| **Gap condition** | 43.5% | 28.7 *(out of 66)* | 15.7 | 40.7% | 26.9 *(out of 66)* | 5.4 |
| **Overlap condition** | 90.2% | 59.5 *(out of 66)* | 6.1 | 91.9% | 60.6 *(out of 66)* | 5.4 |
| **Total** | 66.9% | 88.2 *(out of 132)* | 12.3 | 66.3% | 87.6 *(out of 132)* | 9.9 |

Disengagement costs, which is the additional time participants took to orient to the peripheral stimulus during the overlap condition, were computed as mean SRT of valid overlap condition, minus the mean SRT from the valid gap conditions, this gave an overall gap effect for all participants. Additionally, for different trial types, the mean SRT of overlap for all U trials, minus mean SRT of gap condition for al U trials were extracted to give us a gap effect for Unique trials. The same was computed for 1R, 2R, 3R, and 4R trials.

### 4.2.5 Procedure

Participant recruitment and arrival is the same as Chapter 2 **section 2.4**; participants arrived, completed the EEG study, followed by surveys on the computer, and then took part in the eye tracking experiment.

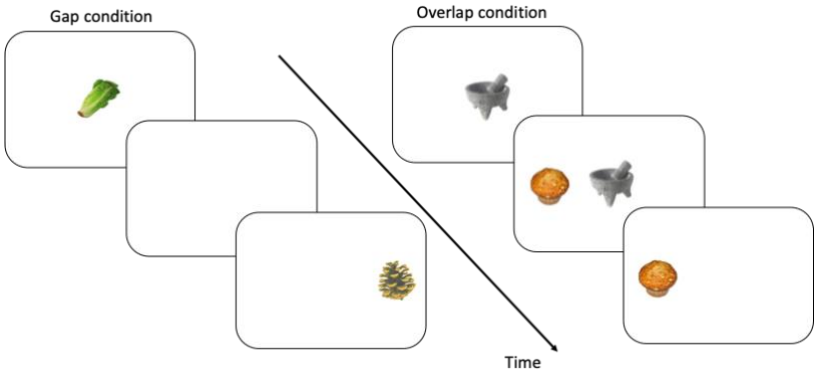
When participants arrive at the eye tracking room, they were briefed on how the eye tracker works, and about the experimental design. Participants were instructed with “images will be appearing on the screen either on the right or on the left of the central image, and they have to look towards them as soon as the images appear”. The same instruction was also typed on the screen before the experiment began.

Participants then took part in the gap-overlap paradigm where they viewed images presented in the centre of the screen, and are expected to shift their gaze towards another image appearing either on the right or left of the central image as soon as it appears. There are no cues prior to the presentation of the central or the peripheral image guiding their saccades. In the gap condition, participants view the central image for 1 second, followed by a gap

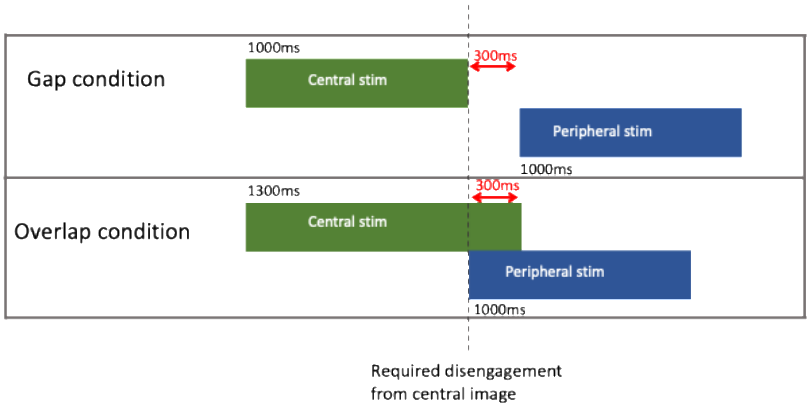
wherein there is nothing on the screen for 300ms and then a peripheral image on the right or left of the central image is presented. In the overlap condition, the peripheral image is presented on the right or left of the central image while the central image continues to be presented for 300ms. *Figure 4.3a & b* depicts the events occurring in all trials for gap conditions and overlap conditions. The peripheral image that the participants were orienting towards was always a novel image that never repeated. So, participants were orienting away from an image that was novel or repeated, towards an image that is always novel.

Left eye position of the participants was tracked. Before the experiment started, the left eye was calibrated to the screen using the automated 9-point calibration and validation procedure to ensure accurate tracking. Following this were 4 practice trials where coloured boxes were used in place of images. In total there were 132 trials, split into blocks of 33 trials, alternating between gap and overlap conditions, in between each block was a drift check.

Trial presentation is blocked so participants experience 32 trials from the gap condition, followed by 32 trials from the overlap condition, then 32 gap again, and finally 32 overlap. The order of the gap and overlap conditions are also counterbalanced across the sample in order to reduce order effects. Additionally, potential stimulus-biases are reduced by creating a version of the experiment where the selection of repeated images are swapped with the selection of non-repeated images, which is also counterbalanced. Participants are randomly allocated a 1 out of the 4 versions of the experiment (stimuli set 1 + G-O-G-O order, OR stimuli set 1 + O-G-O-G order, OR stimuli set 2 + G-O-G-O order, OR stimuli set 2 + O-G-O-G order).

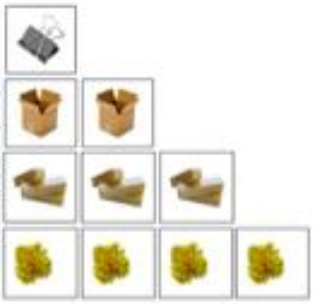



*(a)*




*(b)*

| Type | Number | % of experiment |
|---|---|---|
| U | 32 | 24% |
| R1 | 32 | 24% |
| R2 | 32 | 24% |
| R3 | 24 | 18% |
| R4 | 12 | 9% |
| | 132 | |



*(c)*

*Figure 4.3. (a)(b)* represent the proposed experimental design, using a true gap condition where participant must reorient towards peripheral image, and an overlap condition where there is an overlap of 300ms between the central and peripheral stimulus. Presentation time of the central image is 1300ms in the overlap condition. *(c)* The type of trial and the proportion experimental trials is represented, including gap and overlap trials, making a total of 132 trials.

# 4.3 Results

## 4.3.1 Descriptives

Distributions in the individual differences scores were visually inspected at first for this sample of 37 adolescent participants. Shapiro-Wilk tests showed scores on the ADOS-2 and combined parental and self-report Conners-4 to be normally distributed (W (37) = .97, $p$ = .50), W (37) = .98, $p$ = .62), respectively. Independent samples *t-test* shows a significant difference between ADOS-2 and Conners-4 scores between ASD sample and non-ASD sample ($t$ (35) = 3.60, $p < .001$ for ADOS-2 and $t$ (35) = 5.25, $p < .001$ for Conners-4) such that the ASD sample scored higher in their ADOS-2 scores and in the Conners-4 survey. This is presented in *Table 4.2*. Additionally, data quality checks (*Table 4.1)* shows that there was no significant difference between data loss between the ASD and the non-ASD group for total trials retained ($t$ (35) = .19, $p$ = .85). There was a significant positive correlation between ASD z-scores and ADHD composite z-scores ($r$ (36) = .34, $p$ = .04).

To test the difference between SRT in gap conditions (M = 237.82, SD = 26.84) and overlap condition (M = 274.00, SD = 42.42), we use a paired samples *t-test* and report that there was a significant difference between SRT in the gap conditions and SRT in the overlap condition ($t$ (36) = 6.80, $p < .001$). This shows that the paradigm worked as expected, and that there is a higher disengagement cost associated in the overlap condition versus the gap condition. Additionally, there was a significant difference between gap and overlap trials in both samples of participants; for the ASD group ($t$ (18) = 3.97, $p < .001$), and for the non-ASD group ($t$ (17) = 6.03, $p < .001$).

Average SRTs for all trial types is presented in *Figure 4.4a.* Visual inspection show SRT in the gap condition is reliably faster than the overlap condition across all trial types, and that SRT in the gap conditions are increasing slightly, whereas SRTs in the overlap conditions are decreasing slightly. For further analyses, U trials and R1 trials are combined into one group. This is because at the point of presentation, participants have no knowledge whether this is an image that will repeat; at the point of presentation, it is a new image they are viewing for the first time. Furthermore, R4 is removed from the analysis due to exhibiting an artificially high disengagement cost that is seen at the final presentation of an image, this was seen in preliminary piloting and is confirmed in this sample (*Figure 4.5b).* Gap effect (GE) was calculated for each trial type (U, R1, R2, R3) by subtracting the SRT in gap condition

from the SRT in overlap condition for each trial type. A total GE was also computed the same way which represents the average disengagement cost across the entire experiment.

*Table 4.2*

*Descriptives of individual differences measures between the ASD and non-ASD sample*

| | ASD n = 19, Female = 6 | | | | Non-ASD n = 18, Female = 6 | | | |
|---|---|---|---|---|---|---|---|---|
| | *Mean* | *Std. Dev* | *Minimum* | *Maximum* | *Mean* | *Std. Dev* | *Minimum* | *Maximum* |
| **Age** | 14.1 | 0.97 | 13 | 16 | 14.1 | 1.16 | 13 | 17 |
| **ADOS-2** | 24.6 | 10.3 | 10 | 44 | 13.2 | 8.8 | 1 | 27 |
| **Conners-4** | 24.5 | 6.9 | 11.5 | 34.5 | 12.1 | 6.3 | 1.5 | 23.5 |

### 3.2. The impact of repetitions and traits on SRTs

In order to test the relationship between gap and overlap conditions, across the different repetitions, a 2 (condition: gap, overlap) x 3 (presentation: first, second, third) repeated measures ANOVA is used. This allows us to examine the effects of condition and repetition on SRTs. Firstly, we report a significant effect of condition ($F$ (1, 35) = 32.57, $p$ <.001), and no significant effect of repetition ($F$ (2, 70) = .29, $p$ =.75). There is also no significant interaction between condition and repetition ($F$ (2, 70) = 2.74, $p$ = .07).

Next, two covariates were added into the model: z-scored ASD and ADHD traits. Firstly when ASD traits are added into the model without ADHD traits, the model reveals no interaction between condition and ASD traits ($F$ (1, 34) = 1.80, $p$ =.19), no interaction between repetition and ASD traits ($F$ (2, 68) = .28, $p$ = .75), and no three-way interaction between condition, repetition and ASD traits ($F$ (2, 68) = .86, $p$ = .43). There is also no main effect of ASD traits ($F$ (1, 34) = 1.83, $p$ =.19). Then ADHD traits are added into the model without ASD traits. The model reveals no interaction between condition and ADHD traits ($F$ (1, 34) = .11, $p$ = .74), no interaction between repetition and ADHD traits ($F$ (2, 68) = 1.80, $p$ = .17), and no three-way interaction between condition, repetition and ADHD traits ($F$ (2, 68) = .24, $p$ = .79). There was also no main effect of ADHD traits ($F$ (1, 34) = .01, $p$ = .91),

Finally, in a model where ASD and ADHD are both added as covariates, the ANCOVA still reveals a significant main effect of condition ($F$ (1, 33) = 32.53, $p$ <.001), however there is no significant main effect of repetition ($F$ (2, 66) = .28, $p$ = .76). Notably, there is a significant interaction between condition and repetition with linear contrast ($F$ (1,

33) = 5.05, $p$ = .03) (but falls short of reaching significance without the linear contrast ($F$ (2, 66) = 2.69, $p$ = .08)). This indicates that changes along the different repetitions are a result of changing SRTs in both gap and overlap conditions, not overlap conditions alone.

between ASD and ADHD traits on the relationship between condition and repetition show no significant outcomes (($F$ (2, 66) = 1.22, $p$ = .30) for ASD and ($F$ (2, 66) = .61, $p$ = .55) for ADHD). There are also no main effects of ASD traits in this model ($F$ (1, 33) = 1.89, $p$ = .18), nor main effects for ADHD traits ($F$ (1, 33) = .12, $p$ =.73). Interactions Graphs with median split groupings of the individual differences' measures are presented in *Figure 4.5*.

### 3.2 The impact of repetitions and traits on Gap effects

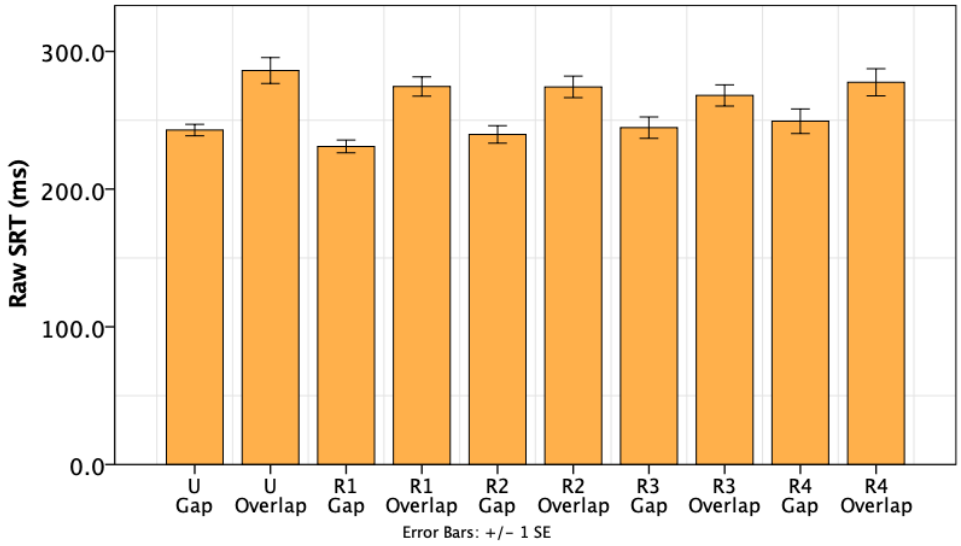
A repeated measures ANOVA was conducted to establish changes in GE across the three repetitions of the image. This showed that there was a significant linear decline in GE across the three repetitions of the image ($F$ (1, 35) = 5.35, $p$ = .03). Beta values – representing the slope of change in GE from presentation 1 until presentation 3 – were extracted from regressions conducted for each participant. Using a one-sample *t-test*, we report that across the sample, participants exhibited slopes that were significantly different to 0 ($t$ (36) = -2.49, $p$ = .02) and the $t$ test value reveals that this the slopes were significantly negative. This shows that generally, participants exhibited a decrease in GE across the presentations.

Z-scored ASD and ADHD traits were added as covariates into a repeated measures ANCOVA to see any effects of their scores on changes in GE across the three presentations. The repeated measures ANCOVA retained a significant change in GE in the linear contrast when controlling for ASD and ADHD traits ($F$ (1, 33) = 5.05, $p$ = .03). There was no main effect of ASD traits on overall GE ($F$ (1, 33) = 2.36, $p$ = .13), and no main effect of ADHD on overall GE ($F$ (1, 33) = .69, $p$ = .41). In addition, there were no notable interactions between individual differences scores and change in GE (($F$ (2, 66) = 1.22, $p$ = .30) for ASD and ($F$ (2, 66) = .61, $p$ = .55) for ADHD).

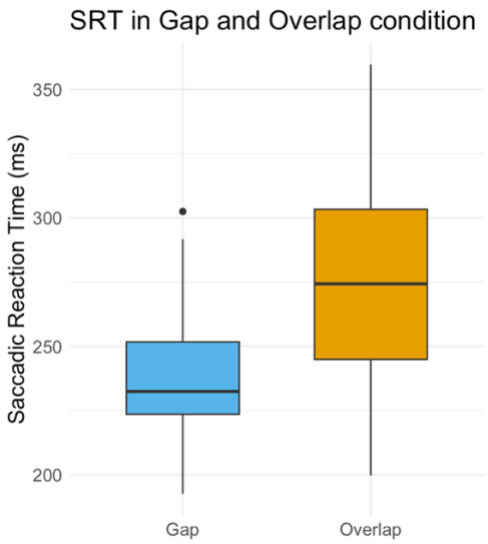
Participant samples were also median split by their individual differences scores into high/low groups. A repeated measures ANOVA only on the low scoring ASD group reveals that the significant decline in GE in the linear contrast is still apparent ($F$ (1, 18) = 5.95, p = .03). Interestingly however, this decline is not significant in the high ASD scoring group ($F$ (1, 16) = .98, p = .34). This is represented in *Figure 4.5a* where there is a more apparent linear decline in the low scoring which is not present in the high scoring group. The high scoring group in fact has comparable GE at presentation 1 and presentation 2, and GE

declines at presentation 3. The same test was conducted for high versus low scoring ADHD groups, where we report high scoring ADHD group had a significant linear decline ($F$ (1, 17) = 4.34, p = .05) whereas low scoring ADHD group did not have a significant decline ($F$ (1, 16) = 2.04, p = .17)., although in the *Figure 4.5* it is apparent that both groups declined in a comparable way.

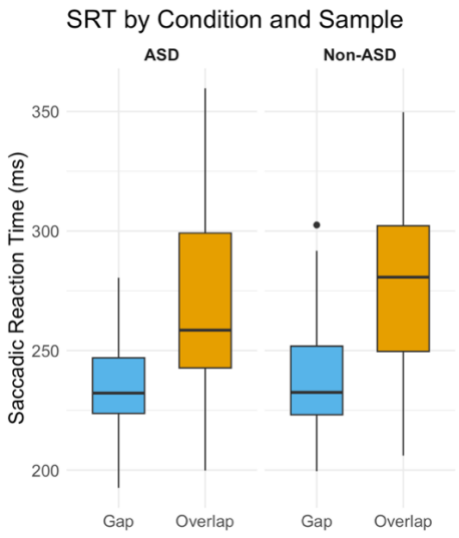
Trials were pseudo-randomised to ensure variation in stimulus order, so that images in their different states (i.e. U, R1, R2, R3) were presented in an unpredictable matter. While this was the intention, it is still the case that R1 trials and R2 trials on average would occur slightly earlier in the trial sequence in comparison to R3 and R4 trials. In order to check that this repetition effect was due to informational states of the stimuli and not due to changing of SRT over time, SRT for each repetition in both gap and overlap condition is averaged for every participant. Repeated measures ANOVA shows that there is no significant change in SRT across the three repetitions when gap and overlap conditions are combined $F$ (2, 72) = .23, $p$ =.80). This shows that any significant change in GE was due to the difference between gap and overlap conditions and their informational states and not due to any overall SRT changes across a block of trials.




*(a)*

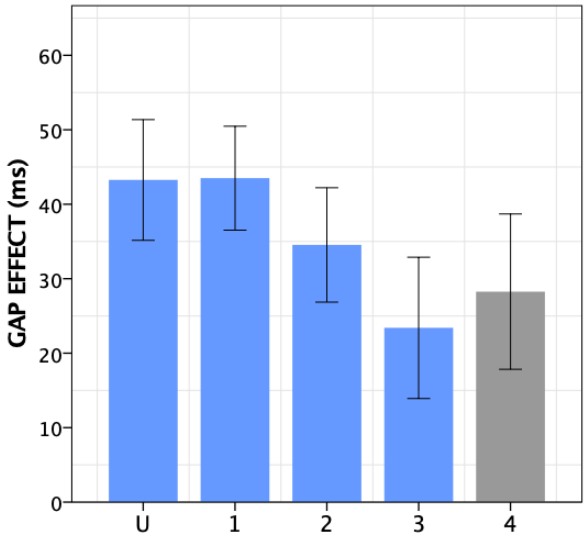



*(b)*

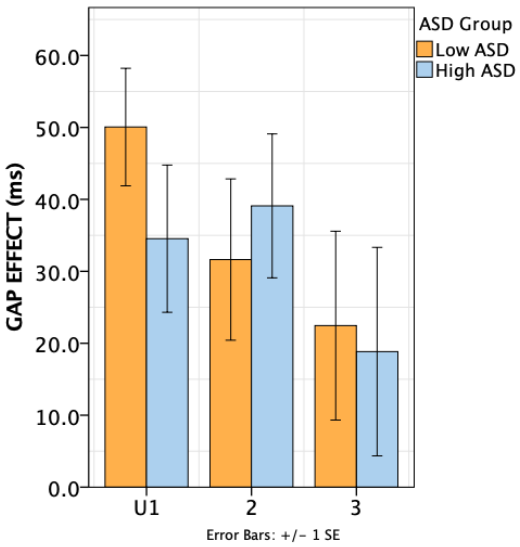



*(c)*

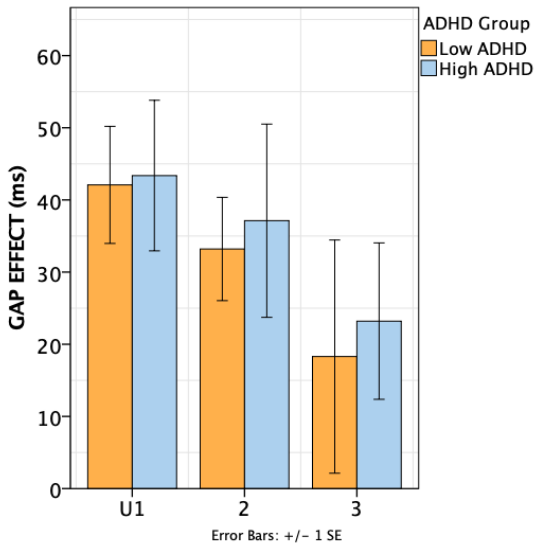
*Figure 4.4 (a)* Raw saccadic reaction times (SRT) for each trial type and each condition. In later analysis U trials and R1 trials are combined and R4 trials are excluded. *(b)* Average SRT for gap and overlap conditions shows that there is more variation within the overlap condition, however SRT in the overlap condition is reliably higher compared to gap. *(c)* Average SRT for gap and overlap conditions between the ASD and non-ASD samples reveal identical trends among both groups.




*(a)*




*(b)*




*(c)*

*Figure 4.5. (a)* The gap effect (GE) for all trial types. The graph shows comparable GE for U and R1 trials, and the trend of reduced disengagement cost is apparent. Additionally, R4 which is the final presentation shows an increase in the GE which was recorded in preliminary testing. *(b)* First, second, and third presentation for ASD median split groupings is presented. Low ASD shows a stronger linear decline in comparison to high ASD groups, although this is not significant. *(c)* First, second, and third presentation for ADHD median split groupings generally shows comparable decline in GE between the groups.

## 4.4 Discussion

The ability to disengage attention from one location and orient elsewhere is essential for gathering information about our surroundings, and to safely navigate complex and dynamic environments. This study aimed to investigate attentional disengagement using an adapted gap-overlap paradigm where we manipulated the informational value of the stimuli participants are expected to disengage from. The first hypothesis was that disengagement cost – quantified as the gap effect (GE) – would decrease as the central stimuli was repeated, or as the information gain declined. The second hypothesis was related to how this change in disengagement cost relates to their individual differences in ASD and ADHD traits. Specifically, that the change in disengagement cost across the repetitions will be smaller for individuals with higher ASD traits, and larger changes for individuals with higher ADHD traits. We report novel findings demonstrating that disengagement is indeed dependent on value of information provided by the stimuli, but in our sample of participants, the relationship with individual differences is not as strong as what was predicted.

Firstly, the presence of a GE in our study indicates that our execution of the gap-overlap paradigm was successful in capturing a disengagement cost that ensues when individuals actively disengage their gaze from stimuli on the screen in order to look at another coexisting stimulus. Crucially, we observed a significant linear decline in GE scores across stimulus repetitions, which aligns with our predictions based on information gain and information foraging theories (Gottlieb & Oudeyer, 2018; Hills et al., 2015; Manohar & Husain, 2013). As the central image becomes familiar, its informational value depletes, which in turn facilitates faster disengagement towards novel peripheral stimuli. When participants must disengage from an image which has similar informational value to the image they are orienting towards, competition between the two stimuli increases, as both provide equal information. However, when there is a discrepancy between informational value between the central and peripheral stimuli, this alters the dynamic of the competition, such that it becomes *easier* to disengage in favour of the novel stimuli. This supports the view that attention operates as a dynamic sampling mechanism, continuously weighing information utility against exploration costs (Brockmole & Boot, 2009; Manohar & Husain, 2013). Our use of the unrepeated trials, randomly interspersed with the repeated trials, helps rule out that the GE change with repetition is simply due to more practice in the task (R3 trials will appear later than R1 trials). Performance in U trials was identical with R1 trials, as expected from their information value.

Previous research had found mixed findings in relation to global disengagement cost differences between adults and children with and without ASD (Fischer et al., 2014; Fischer et al., 2016; Mo et al., 2019; Sacrey et al., 2014; McLaughlin et al., 2021; Matsuo et al., 2015; Kawakubo & Maekawa, 2015; Kawakubo et al., 2007). For example, research by Kawakubo et al., (2007), Matsuo et al., (2015), and McLaughlin et al., (2021), show differential results in visual disengagement in individuals who present with ADHD or ASD. On the other land, research by Wilson and Saldaña (2019), Hanisch et al., 2006, and Fischer et al., (2014; 2016), report no differential results in visual disengagement in individuals who present with ADHD or ASD. Our findings correspond with the line of research that reports no differences in GE between typically developing and ASD groups as well as in relation to ADHD traits. Previous research which have found slowed disengagement in ASD samples has also been attributed to global developmental delay rather than autism specifically (Chawarska et al., 2010), and it is the case that many children with a autism do experience several developmental delays (Shan et al., 2022) which may have been driving the differences in disengagement speeds in previous research.

Contrary to predictions derived from the neural gain model and attentional models of ASD and ADHD (Hauser et al., 2016; Landry 2021), individual differences in traits did not modulate disengagement patterns. This suggests disengagement costs associated with information gain is not affected by individual differences in this attention switching paradigm. Instead, our results from our sample reveal that stimulus-driven factors (i.e informational value of a stimuli) affects disengagement similarly across all participants and that potential differences in attentional systems do not present as having different attentional disengagement patterns. The neural gain model may better explain higher-level cognitive processes such as decision-making and task-switching than the relatively low-level oculomotor disengagement measured here (Hauser et al., 2016). Second, the paradigm may lack sufficient sensitivity to detect subtle trait-related differences in disengagement, particularly given the restricted range of ADHD traits in our sample. Third, as Fischer et al. (2014) noted, it is possible difficulties may emerge in real-world conditions and not apparent in more controlled laboratory settings. The same can be said with regards to our findings where there are no different patterns of disengagement relating to differences in ASD and ADHD traits; as it may be the case that this presents in real-world settings, or in paradigms that better mimic attentional disengagement from real-life settings.

An interesting finding in this work is the lack of significant decline in high scoring ASD participants. From the data and the visual representation from *Figure 4.5b* is that individuals with higher ASD traits are choosing to persist longer with the repeating images. These findings seem to indicate that individuals with higher ASD traits reach the threshold after which disengagement becomes *easier* later than individuals with lower ASD traits. This is in line with our predictions; however, this trend is only apparent and significant when participants are median split according to their ASD traits, weakening the finding. This is also comparable to the finding reported in the EEG study in adolescents where theta band activity (relating to engagement) did not have a linear decline in the high-scoring ASD group as it did in the low-scoring ASD group.

Core processes from the information foraging models (Calhoun & Hayden, 2015; Hills et al., 2015) are that individuals engage with a source of information until a threshold is reached where the amount of effort required to continue extracting information from this source, outweighs the relative utility of that information. Our proposal based on the works of Gottlieb and Oudeyer (2023), Manohar and Husain (2013), and Bromberg-Martin and Monosov (2020) is that information foraging relies on attentional process of disengagement, transfer, and engagement (as outlined by Posner, 1982) to effectively sample for information in the environment. Additionally, research has shown that this system is susceptible to individual differences, that individuals with particular attentional profiles, will be sampling their environment for information differently (Landry 2021).

There are several matters relating to the sample that must be noted, and it is possible that this may have played a role in the observed results. Firstly, the small sample size, which is smaller than what has been recruited in most, but not all previous research; for example, Fischer et al., (2016) recruited 84 participants, and Matsu et al., (2015) recruited 125 participants, where 35 were of the ADHD group. For reference, Wilson and Saldaña (2019) recruited 34 participants which is similar to our sample. This sample size was sufficiently sensitive to look at effects of repetitions of the images and see broad changes that can be reliably traced to informational gain; however it is possible that smaller interaction effects between traits and disengagement patterns may be obscured.

Our experimental paradigm addressed several critical limitations from previous research, for example using a version of a task with a true 300ms gap condition, in order to simulate disengagement with *no information* and disengagement *with information* between

the gap and overlap conditions respectively. We also standardised the time participants looked at the stimuli to be 1 second, even in the overlap condition where central image presentation was an additional 300ms, the peripheral image was presented at the 1 second mark, forcing participants to initiate the disengagement process at that exact moment. In our experiment we used everyday object images which was expected to increase engagement in comparison to simple shapes (Hanisch et al., 2006). Our aim was to incorporate information gain and therefore we used repeated images; in order to offset anticipatory strategies or anticipatory information gain, the images were repeated in unpredictable patterns.

A notable feature of the data is the difference between retention rates between conditions, with the gap condition retaining approximately 42% of trials compared to 91% in the overlap condition (presented in *Table 4.1*). This disparity can be primarily explained by the design of the gap condition itself. During the 300ms gap interval, the screen is entirely blank, providing no visual anchor to maintain gaze in the central location. Our inclusion criteria required the participant's saccade to originate from the central location (not from anywhere else on the screen, to minimise longer than expected or shorter than expected travel distance of a saccade), and land in the correct peripheral position of the image presentation. This naturally resulted in large number of trials being rejected as participant's gaze likely drifted away from the central position to elsewhere on the screen during the 300ms interval. One potential remedy for future implementations of this experiment could be to include a subtle fixation cross at the centre of the location during the gap condition. This would introduce a small visual anchor for gaze that would result in higher number of trails retained, however it does also introduce a small amount of visual information in a condition where we intend participants to disengage from no information.

### 4.4.1 Limitations

Some methodological and sample-related limitations should be considered when interpreting the findings of this study. Firstly, the use of non-social, everyday objects as stimuli – although it may have allowed greater experimental control – may have limited the ecological validity of the paradigm. As noted in Fischer et al., (2014), trait-related differences in attentional disengagement may be more apparent when stimuli are socially or emotionally salient. Our participants, particularly those who scored higher in their ASD traits, may present different disengagement patterns if their initial engagement with the stimuli was higher. Future work could incorporate stimuli that are significantly more salient and interesting, yet

still non-social to examine disengagement patterns when engagement is expected to be higher initially. Second, as discussed above, the high exclusion rate in the gap condition represents a potential issue with the design of the experiment. It is important that future studies consider using a subtle fixation cross during the gap interval in order to anchor gaze in the central position.

With respect to ADHD traits, it is important to acknowledge, that while the ADHD-index scores – used as the ADHD traits – in this research were varied, we had very few participants with a clinical diagnosis of ADHD, and we did not recruit a comparable ADHD-diagnosed group. This does limit our sample in addressing our research questions fully, as we had very few participants with high ADHD traits in the absence of ASD traits.

### 4.4.2 Conclusion

The task we used appropriately and successfully measured disengagement changes as a response to changing informational value of the stimuli. Our results demonstrates that attentional disengagement is affected by the informational value of the stimuli, such that disengagement cost is higher when disengaging from stimuli with high value (novel information), and disengagement cost progressively lowers in response to increasing familiarity of the stimuli (stimuli depleted in information). Our novel version of a classically used paradigm also addressed several design limitations of previous research but despite a large range of ASD and ADHD scoring participants, information gain did not modulate changes in disengagement in entirely in accordance to our predictions. We do report that disengagement costs in high-scoring ASD groups reflect a persistence of familiarity and a late reaching of sufficient information gain. Disengagement mechanisms of neurodiverse and typically developing adolescents seem to be both be sensitive to information depletion, and are not modulated by ADHD trait scores. The process of disengagement from sources of information with depleting returns operates independently of ASD and ADHD related traits. This challenges previous notions of altered attentional disengagement (Landry 2021), and the idea proposed previously in this work relating to differences in information sampling in Autism and ADHD.

# CHAPTER 5:

## 5. Attentional disengagement in response to information gain in typical adults with Autistic traits and ADHD traits

### Goal of chapter

*This chapter uses the same eye tracking paradigm from the previous chapter, in which we modulate informational value through stimulus repetition, but this time in a larger sample of young adult participants, adopting the neurodiversity approach. The primary goal was to extend our adolescent findings into adulthood to investigate whether a more mature system (Lune at al., 2004) and the use of sensitive self-report trait measures would reveal the hypothesized modulatory role of ASD and ADHD traits on information-based disengagement, as predicted by neural gain models.*

*This chapter presents 3 experiments, in which we modulate the information gain available. The first experiment uses an identical gap-overlap paradigm as that presented in the previous chapter, in which participants actively disengage and orient their gaze from a central image towards an image appearing in the periphery. In the second experiment overall information gain potential of the experiment is decreased, by reducing stimulus presentation times from 1000ms to 500ms. In the third experiment overall information gain potential is increased, by blocking presentations of the 3 repeated images.*

*Across these experiments, we found variation in the findings. In Experiment 1 we report no main effect of repetitions on the disengagement costs, however we report a significant non-linear (quadratic) pattern of disengagement associated with higher ADHD traits. In Experiment 2, the low information gain manipulation, which was achieved through limiting presentation and overlap times, diminished the gap effect. Finally, in experiment 3, the high information gain manipulation resulted in a successful gap effect, and we report a significant non-linear trend (quadratic) between information extraction and disengagement.*

*Together these findings show that the relationship between informational value and attentional disengagement is nuanced in adults and that experimental paradigms such as the gap overlap paradigm is sensitive to methodological designs.*

# 5.1 Introduction

In the previous chapter we adapted a well-known disengagement paradigm by using repetitions in the stimuli to modulate informational value and therefore informational gain from stimuli. The paradigm features two conditions, the gap and overlap condition, and the difference in RT between these two conditions yields the Gap Effect (GE) which is the key dependent variable. Participants must disengage their gaze from the central image towards images appearing in the periphery.

This paradigm has been widely used to investigate potential disengagement differences in ASD and ADHD, and the literature reveals mixed findings. Some studies showed slower disengagement in ASD (Landry & Bryson, 2004; Kawakubo et al., 2007; Elsabbagh et al., 2009), while others report no such differences (Fischer et al., 2014; 2016; Goldberg et al., 2002). Although fewer studies are conducted in this paradigm in ADHD, the findings are also mixed, some have reported attenuated gap effects overall (Matsuo et al., 2015) or no differences to typically developing groups (Hanisch et al., 2006). A comprehensive review by Sacrey et al., (2014) summarised various methodological variations such as differences in stimuli, timing parameters, and sample characteristics as potential limitations in previous research. In the previous chapter we suggested that, rather than being limitations, these factors are key variables, influencing RTs by modulating information gain from stimuli.

In this paradigm we manipulate the central image, such that it is either offering novel information that participants are viewing for the first time and thus informational value is high, or offering information that has been viewed once before, and thus informational value is somewhat depleted, or offering information that has been viewed twice before, where informational value is the lowest. In the previous sample of adolescent participants, we found that disengagement speed reduced in a linear trend, corresponding with a linear decline in informational value of the central image – the image participants are actively disengaging from. However, we did not find a role played by individual differences in Attention Deficit Hyperactivity Disorder (ADHD) traits and Autism Spectrum Disorders (ASD) entirely in the way that we predicted, contrary to our hypotheses based on neural gain models proposed by Hauser et al., (2016) and Piccardi and Gliga (2022). However, some trends in the data point towards higher ASD traits associating with persistence with the repeating image for longer than those with lower ASD traits.

The present chapter seeks to extend the findings of the previous chapter in several crucial ways, aligning with the approach we took in our EEG studies. First, we administer the same novel paradigm to a larger adult sample, adopting a neurodiversity and multi-dimensional approach where we examine ASD and ADHD as traits along a continuous spectrum in all participants. Attentional systems, particularly prefrontal and striatal dopaminergic pathways, undergo significant maturation from adolescence into adulthood (Luna et al., 2004; Crone, 2009). It is therefore unknown whether the relationship between informational value and disengagement cost is similar across the lifespan or whether there are changes that emerge between adolescents into adulthood due to a more mature system (Lune et al., 2004; Crone, 2009; Paus, 2005). Secondly, by moving to an adult sample, we are able to employ self-report measures of subclinical traits, and which are efficient and sensitive to the natural variations present in the general populations (Happé & Frith, 2020). This approach also aligns with the multidimensional and neurodiversity framework successfully applied in Chapter 3. This approach also allows us to recruit larger samples and gives us better power to detect more subtle modulations that may have been obscured in the smaller adolescent sample.

It should be noted that while Experiment 1 successfully achieved this larger sample size (n=98), subsequent experiments (Experiments 2 and 3) were conducted with smaller samples. This was partly due to practical constraints of participant recruitment, but most of all due to the aims of the follow-up experimentation, which were to address the questions from the findings of Experiment 1 rapidly, and not necessarily to expand sample size.

Based on the original neural gain framework (Hauser et al., 2016; Piccardi & Gliga, 2022; Landry, 2021), which theorise opposing attentional profiles in ASD (high gain, stable goal allocation) and ADHD (low gain, unstable goal allocation) we put forth the same hypotheses as described in the adolescent sample. Firstly, we predict that overall, across all participants, disengagement will be facilitated as the informational value of the stimuli decline. Secondly, we hypothesise that this pattern of disengagement across repetitions will be modulated by individual differences in ASD and ADHD traits. Specifically, that higher ADHD traits will be associated with a steeper decline in disengagement costs, reflecting a greater sensitivity to information depletion and a stronger preference for orienting towards novel images presented in the periphery. Conversely, we expect higher ASD traits to be associated with more attenuated change in disengagement cost across the repetitions, demonstrating a preference/persistence of engagement with familiar stimuli, and a higher

threshold for stimulus to be sufficiently depleted of information. In the previous chapter we found that these predictions were not supported, we expect that in a larger sample of adults with sensitive ASD and ADHD measures, these differences may emerge.

There are three experiments described and conducted as part of this chapter. The first one is an identical paradigm to the one conducted previously in adolescents, the second and third paradigm are adjusted for information gain such that one decreases the information gain at each presentation by shortening the presentation times, and one increases the information gain at each presentation by blocking repetitions of the stimuli, and are motivated by findings from the first experiment.

# 5.2 Experiment 1

## 5.2.1 Methods

### 5.2.1.1 Participants

From a sample of 104 undergraduate Psychology students, who were recruited through SONA in return for course credit, six were removed, due to bad recording or low retention of trials. Final sample was of 98 participants aged between 18 – 31 years old (76 women, M age = 20.4, SD age = 2.1). In this sample there was one person who self-reported to be diagnosed with ASD, one person reported ADHD, and one person reported ASD, ADHD, and anxiety.

All participants reported normal or corrected-to-normal vision and procedure was approved by the Research Ethical Committee of the University of East Anglia, Norfolk, United Kingdom.

### 5.2.1.2 Design

The design of this experimental paradigm is identical to the previous chapter conducted in an adolescent sample. The key difference in methodological approach between the adolescent sample and the adult sample is measures used to compute individual differences in ASD and ADHD traits. In the previous adolescent sample, Conners-4 rating scale (Conners, 2023) and ADOS-2 were used (Lord et al., 2012), whereas in this sample, we use the Autism quotient (Baron-Cohen et al., 2001), Broad autism phenotype questionnaire (Hurley et al., 2007), Wender Utah scale (Ward, Wender, & Reimherr., 1993), and the Adult ADHD self-report scale (Kessler et al., 2005).

The study employed a repeated measures design and used individual differences measures of ASD and ADHD related traits on continuous scales. The within-subject independent variables were condition (gap vs. overlap), and stimulus presentation (U, R1, R2, R3). Additionally, individual differences scores in ASD and ADHD traits are treated as covariates and between subject independent variables. The dependent variables was saccadic reaction time (SRT) – which is defined as the time it takes for the saccade to initiate from the central location towards the peripheral image from the time the peripheral image is presented – and gap effects (GE) – which the difference between SRT in the gap condition and the overlap condition.

Participant's SRT are measured for each trial, and a GE is calculated from the difference between gap conditions and overlap conditions. The image participants are disengaging from is manipulated for information gain potential, being either a completely novel image, or an image they are viewing for a second time, or an image they are viewing for the third time. Condition randomisation, counter balancing, and stimuli swapping is conducted similarly to the adolescent sample.

#### 5.2.1.3 Material

*5.2.1.3.1 Tests of individual differences*

*See Chapter 3, subsection 3.2.3.1*

*5.2.1.3.2 Stimuli*

*See Chapter 4, subsection 4.2.3.2*

*5.2.1.3.3 Apparatus*

*See Chapter 4, subsection 4.2.3.3*

#### 5.2.1.4 Eye-tracking analysis

*See Chapter 4, subsection 4.2.4*

#### 5.2.1.5 Procedure

Upon signing up to the study, participants received a link to complete the surveys in addition to participant information sheets and corresponding consent sheets. The surveys take approximately 15-20 minutes to complete and should be completed before attending the experimental session. Participants were also encouraged to sleep sufficiently the night before the scheduled lab experiment and are asked to avoid heavy make-up in order to facilitate better eye tracking.

Upon arrival participants are briefed on the experimental paradigm, given the chance to read the participant information sheets once more, followed by signing consent sheets.

Participants are instructed with the following: "images will be appearing on the screen either on the right or on the left of the central image, and they have to look towards them as soon as the images appear". This instruction was also presented to them on the screen as part of the experiment and was kept standardised across all adult and adolescent sample testing.

Left eye position was tracked, except in the situation where participants had lazy-eye where the eye would drift, in these cases, the right eye position was tracked. The eye was calibrated to the screen using the automated 9-point calibration and validation procedure. Participants completed 4 practice trials and then started the experiment.

## 5.2.2 Results

### 5.2.2.1 Descriptives of traits measures

We visually inspected distribution in the individual differences scores for this sample of 98 adult participants. Shapiro-Wilk tests showed scores on the ASRS, WURS, and AQ violate normality (W (98) = .95, $p < .01$), W (98) = .88, $p < .01$), W (98) = .90, $p < .01$ respectively), whereas scores on the BAPQ were normally distributed (W (98) = .99, $p = .65$). Although Shapiro-Wilk tests indicated that the raw scores of the ASRS, WURS, and AQ deviated significantly from normality, we proceeded with parametric tests for the primary analyses. This decision is supported by the well-established robustness of the F-test to violations of normality, particularly with moderate-to-large sample sizes (n = 98; Blanca et al., 2017).

Descriptive statistics reveal that the range was varied across all the tests of individual differences (*Table 5.1.1*). In this sample of participants, raw scores from the surveys were correlated, and when z-scored, there was a strong significant correlation between ASD z-scores and ADHD composite z-scores (r (96) = .45, $p < .001$). Data retention is also presented in *Table 5.1.2* showing comparable trial retention between gap and overlap conditions.

*Table 5.1.1*

*Participant characteristics and descriptives from individual differences surveys*

| | *Mean* | *Std. Dev* | *Minimum* | *Maximum* | *Range* |
|---|---|---|---|---|---|
| **Age** | 20.4 | 2.1 | 18 | 31 | 13 |
| **AQ** | 20.3 | 16.0 | 5 | 41 | 36 |
| **BAPQ** | 3.1 | .70 | 1.6 | 4.7 | 3.2 |
| **WURS** | 23.2 | 19.7 | 0 | 84 | 84 |
| **ASRS** | 7.7 | 4.7 | 0 | 18 | 18 |

*Table 5.1.2*

*Overall data retained after removing invalid trials*

| | *% trials retained* | *No. trials* | *Std. Dev* |
|---|---|---|---|
| **Gap condition** | 76.1% | 50.3 *(out of 66)* | 8.3 |
| **Overlap condition** | 85.8% | 56.6 *(out of 66)* | 5.6 |
| **Total** | 81.0% | 106.9 *(out of 132)* | 6.9 |

### 5.2.2.2 The impact of repetitions and traits on RTs

To test the difference between SRT in gap conditions (M = 228.87, SD = 21.74) and overlap conditions (M = 256.70, SD = 35.40), a paired samples *t*-test was used, and we report a significant difference between SRT in both conditions ($t$ (97) = 10.37, $p < .001$). This confirms the paradigm worked as expected, and that there is a higher disengagement cost associated with the overlap condition as opposed to the gap condition.

To test the relationship between gap and overlap conditions, across the three repetitions, a 2 (condition: gap, overlap) x 3 (presentation: first, second, third) repeated measures ANOVA. This revealed a significant main effect of condition ($F$ (1, 97) = 89.73, $p <$ .001), confirming the presence of a gap effect. There was no significant effect of repetition ($F$ (2, 194) = .93, $p$ = .40), and no significant interaction between condition and repetition ($F$ (2, 194) = .43, $p$ = .65).

The two individual differences measures (z-scored ASD and ADHD traits) were then added as covariates individually into the model. Firstly, ASD traits were added, and the model shows that there is no significant interaction between condition and ASD traits ($F$ (1, 96) = .35, $p$ = .56), no significant interaction between repetition and ASD traits ($F$ (2, 192) = 2.68, $p$ = .07), and no significant three-way interaction between condition, repetition and ASD traits ($F$ (2, 192) = 2.13, $p$ = .12). There was also no main effect of ASD traits ($F$ (1, 96) = .23, $p$ = .63). Next, ADHD traits were added into the ANCOVA without ASD traits. This revealed no significant interaction between condition and ADHD ($F$ (1, 96) = .12, $p$ = .73), there was however, a significant interaction between repetition and ADHD traits ($F$ (2, 192) = 5.62, $p < .01$), and no significant three-way interaction between condition, repetition and

ADHD traits ($F$ (2, 192) = .41, $p$ = .67). There was also no main effect of ADHD traits ($F$ (1, 96) = .03, $p$ = .85).

Finally, an ANCOVA was implemented with both the individual differences measures added as covariates. This revealed that when ASD and ADHD traits are controlled for, there is still a significant difference between conditions ($F$ (1, 95) = 88.63, $p$ < .001) and still no significant effect of repetitions ($F$ (2, 190) = .98, $p$ = .38). In addition, there is no main effect of either ASD ($F$ (1, 95) = .39, $p$ =.53) nor ADHD ($F$ (1, 95) = .20, $p$ =.66). In this model, there is however there is an interaction between repetition and ADHD traits ($F$ (2, 190) = 4.01, $p$ = .02), that is also significant in the quadratic contrast ($F$ (1, 95) = 13.86, $p$ < .01). There was no interaction between repetitions and ASD ($F$ (2, 190) = 1.15, $p$ = .32). This shows that SRT across the three repetitions changes as a function of ADHD traits such that individuals with higher ADHD traits show a V-shaped changed across repetitions in comparison to those with lower ADHD traits. This is presented in *Figure 5.1.1c* where participant's gap and overlap SRTs are averaged for each presentation to visualise the effect of repetitions alone across the two conditions. No other interactions in this model were significant.

In order to check whether there were any overall repetition effects – that may be attributed to improved reaction times – SRT for each repetition in both gap and overlap condition is averaged for every participant. Repeated measures ANOVA shows that there is no significant change in SRT across the three presentations of the stimuli ($F$ (2, 194) = .93, $p$ = .40). This shows that in this sample overall reaction times of participants stayed stable across a block of trials.

#### 5.2.2.3 The impact of repetitions and trait on Gap effects

Gap effect (GE) was calculated for each trial type (U, R1, R2, R3, and R4) by subtracting the SRT in gap condition from the SRT in overlap condition for each trial type. As previously explained, due to final presentation having fewer trials and exhibiting an inflated disengagement cost, R4 is removed from analysis, this was apparent from the preliminary testing, as well as in the results of Chapter 4, and is also apparent in *Figure 5.1.2a* in this sample. In addition, U trials and R1 trials are combined into one condition (U1) for analysis due to its state as the first presentation.

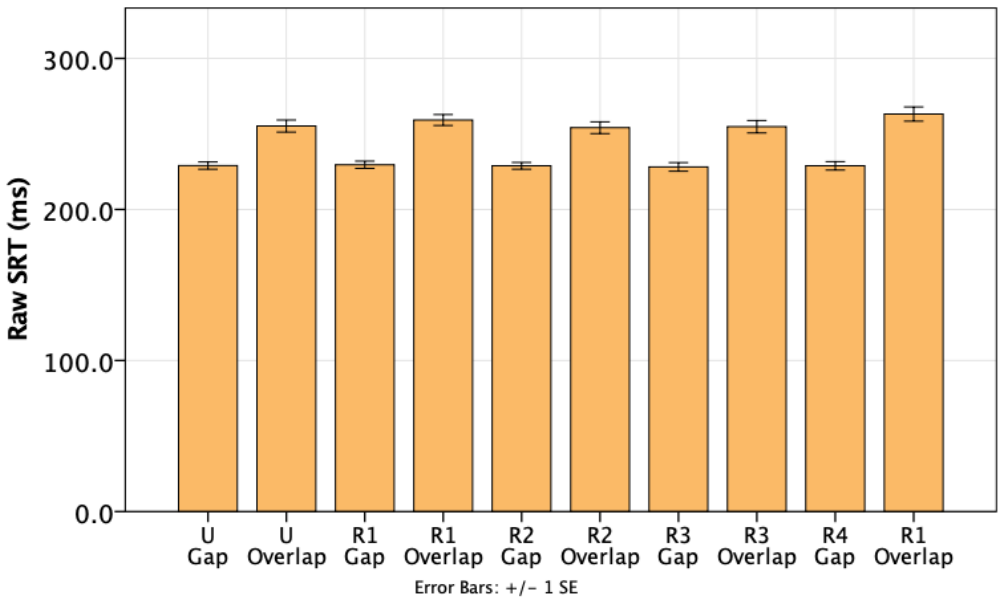
A repeated measures ANOVA was conducted to test changes in GE across the three repetitions of the image. Contrary to the findings of the previous sample, we report no

significant change in GE across the three repetitions of the image ($F$ (2, 194) = .43, $p$ = .65). Beta values which represent the slope of change in GE from presentation 1 until presentation 3 were extracted for each participant. Using a one-sample $t$-test, we first report that across the sample, the slope of change was not significantly different to test statistic 0 ($t$ (97) = -.40, $p$ = .69). This tells us that there was no systematic change across the repetitions.

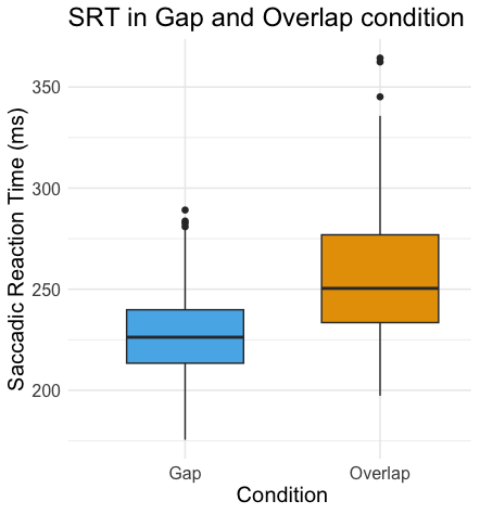
Z-scored ASD and ADHD traits were added as covariates into a repeated measures ANCOVA to see any effects of their scores on changes in GE across the repetitions. The ANCOVA revealed no significant main effect of repetitions when ASD and ADHD traits are controlled for $F$ (2, 190) = .43, $p$ = .65). In addition, there was no main effect of ASD traits ($F$ (1, 95) = .69, $p$ = .41) nor ADHD traits $F$ (1, 95) = .47, $p$ = .50) on overall GE in this model. There were also no interactions between ASD and repetitions ($F$ (2, 190) = 1.876, $p$ = .16) nor between repetitions and ADHD traits ($F$ (2, 190) = .17, $p$ = .84).

The quadratic trend that emerged from the 2 x 3 repeated measures ANCOVA in the raw SRT in relation to ADHD traits, was not apparent when looking at GE ($F$ (1, 95) = .02, $p$ = .89). The quadratic trend that was present as a function of ADHD traits, in this statistical test is significant with ASD traits ($F$ (1, 95) = 8.44, $p$ = .01). In *Figure 5.1.2b & c* there is a trend in high scoring ADHD and high scoring ASD that appears more quadratic in comparison to the low scoring ADHD and ASD group. This, along with the strong correlation between ASD and ADHD traits suggests that the quadratic trend that emerges from this paradigm is a modulation that arises from a general neurodiversity domain, instead of ASD or ADHD specifically.

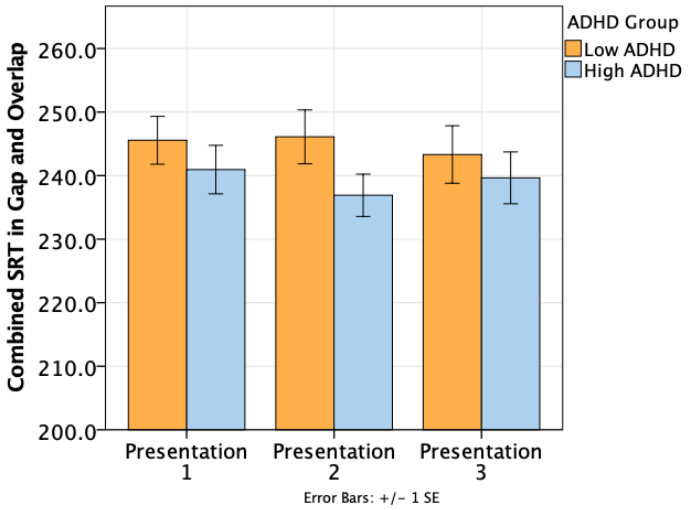
A total GE was also calculated representing the overall average disengagement cost across the experiment. In this adult sample of participants, overall GE was not correlated with z-scored ASD traits ($r$ (97) = .05, $p$ = .62) and not correlated with z-scored ADHD scores ($r$ (97) = -.02, $p$ = .88). This corroborates with findings from the adolescent sample finding no overall disengagement deficits or differences associated with ASD and ADHD traits.


Raw SRT (ms)
300.0
200.0
100.0
0.0
U Gap
U Overlap
R1 Gap
R1 Overlap
R2 Gap
R2 Overlap
R3 Gap
R3 Overlap
R4 Gap
R1 Overlap
Error Bars: +/- 1 SE

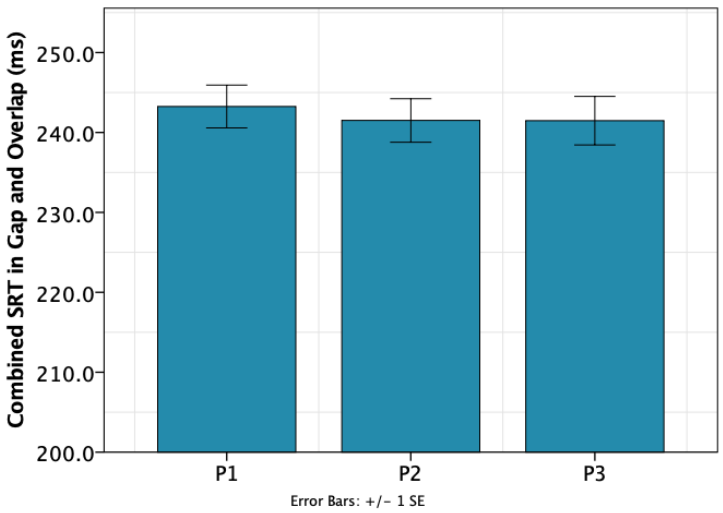
*(a)*


SRT in Gap and Overlap condition
Saccadic Reaction Time (ms)
350
300
250
200
Gap
Overlap
Condition

*(b)*

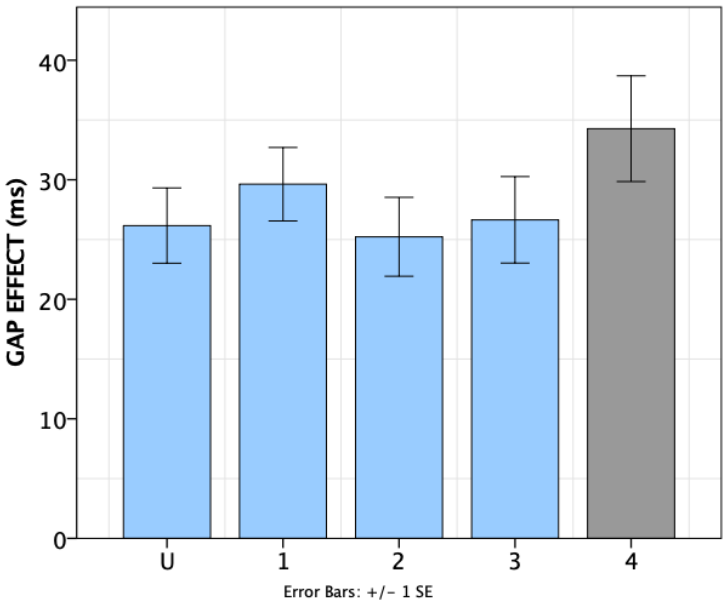

ADHD Group
Low ADHD
High ADHD
Combined SRT in Gap and Overlap
260.0
250.0
240.0
230.0
220.0
210.0
200.0
Presentation 1
Presentation 2
Presentation 3
Error Bars: +/- 1 SE

*(c)*

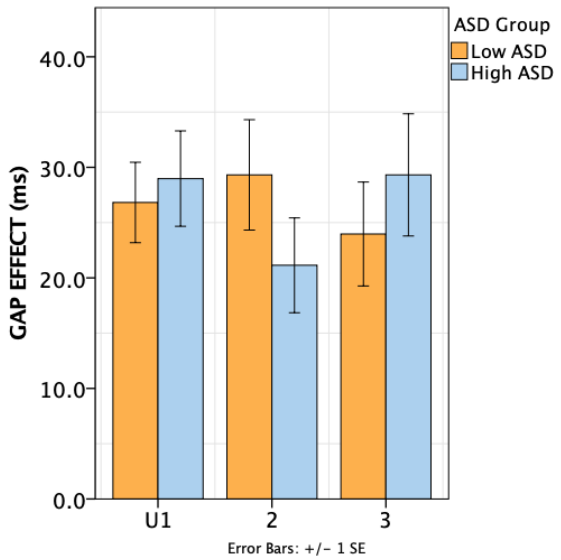

Combined SRT in Gap and Overlap (ms)
250.0
240.0
230.0
220.0
210.0
200.0
P1
P2
P3
Error Bars: +/- 1 SE

*(d)*

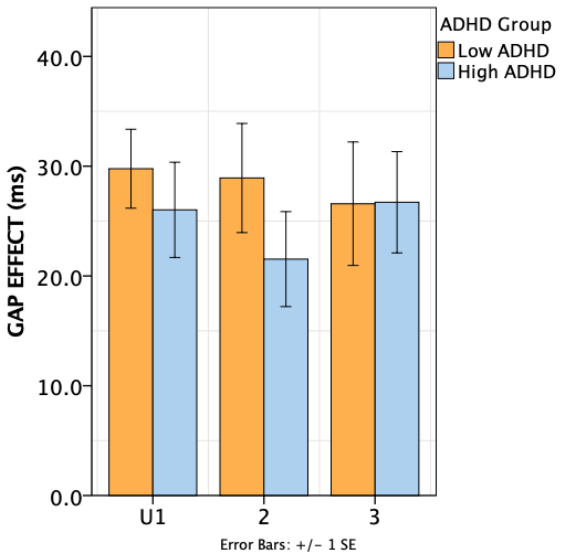
*Figure 5.1.1 (a)* Raw saccadic reaction times (SRT) for each trial type and each condition. In later analysis U trials and R1 trials are combined and R4 trials are excluded. *(b)* Average SRT for gap and overlap conditions shows that there is more variation within the overlap condition, however SRT in the overlap condition is reliably higher compared to gap. *(c)* Average SRT for combined gap and overlap conditions for each presentation type is illustrated to see the broad effect of repetition on reaction time. Participants are median split into high versus low scoring ADHD groupings for visualisation although the repeated measures ANCOVA had ADHD traits as a continuous variable. *(d)* Total SRT for combined gap and overlap conditions shows no significant change over time. Note that *y*-axis starts from 200ms, this is to see clearer the variation within the data.




*(a)*




*(b)*




*(c)*

*Figure 5.1.2 (a)* The gap effect (GE) for all trial types. The graph shows comparable GE for U, R1, R2, and R3 trials, and that there is no overarching trend of decreasing GE. Additionally, R4 which is the final presentation shows an increase in the GE which was recorded in preliminary testing. In all analysis, U and R1 is combined into U1 and R4 is removed. *(b)* First, second, and third presentation for ASD median split groupings is presented. High ASD shows a stronger quadratic change across repetitions in comparison to low ASD group, although this is not significant. *(c)* First, second, and third presentation for ADHD median split groupings generally shows similar trends to ASD groups, such that high ADHD display a more quadratic change in comparison to low ADHD.

## 5.2.3 Discussion

This study aimed to extend the findings from our previous adolescent sample by investigating attentional disengagement within an information foraging framework in a larger adult sample. We employed our novel, adapted gap-overlap paradigm designed to manipulate informational value of the central stimulus through repetition. In the adolescent sample, this paradigm successfully captured faster disengagement as stimuli were depleting in information, although no trends relating to individual differences emerged.

As with the adolescent sample, the gap-overlap paradigm was executed successfully, and a gap effect – which is the main dependent variable and affirms the presence of a disengagement cost – did emerge. In this current study, our first hypothesis, predicting the same decrease in disengagement cost (GE) with decreasing information gain potential, was not supported. Instead, our data shows that disengagement patterns overall are not affected by the novelty or familiarity of the stimuli. Additionally, no expected trends relating to ASD and ADHD traits emerged in this sample. We report an unexpected finding where the pattern of disengagement across the stimulus repetitions was modulated by individual differences in ADHD traits.

The lack of global correlation between overall GE and ASD and ADHD traits in adults corroborates with the findings of our adolescent sample and is in line with research suggesting that basic disengagement function is intact in neurodiverse populations when measured in similar lab-based tasks (Fischer et al., 2016; Goldberg et al., 2002; Hanisch et al., 2006).

Contrary to the predictions and the results from the adolescent sample, we did not observe a significant linear decline in GE across the stimuli repetitions, representing easier disengagement from stimuli that the participant has become familiar with. This could be due to several reasons, it could be that the relationship between information potential and disengagement is more complex in adults than in adolescents, such that adults – who have fully matured attentional systems – are less affected by small changes in informational value when disengaging and orienting elsewhere. Indeed, there are overall differences between average SRT of adolescents and average SRT of adults in this sample such that adults were disengaging faster than adolescents on average (M adolescents = 255.90, SD = 34.63; M adults = 242.78, SD = 28.57) which may be attributed to a more matured system. Luna et al., (2015) outlines that a matured cognitive control system directly relates to better signal-to-

noise ratio, better connectivity between dopaminergic and GABAergic systems, which may lead to faster information gathering system. It may be the case that at the first presentation of an image adult participants acquire maximum amount of information and disengaging already as fast as possible from the first presentation. Alternatively, as is the case with university student samples, is that the adult participants may be less engaged and therefore are not aware of the 2nd, 3rd, and 4th presentations as they often occur with a gap of up to 7 trials. This may result in participants experiencing most presentations as novel presentations with less variation between trial types, particularly if occurring with larger gaps. These two issues are directly addressed and manipulated in *experiment 2* and *experiment 3* in following sections of this chapter.

One interesting finding here is the significant interaction between ADHD traits and SRT in the gap and overlap condition across the three presentations. It is important to note that this pattern did not reach significance when looking at the main dependent variable of this paradigm (i.e. the GE which is truer measurement of disengagement speed in this paradigm), but rather significance was reached when gap and overlap conditions were entered as separate conditions into a repeated measures ANOVA. In any case, the V-shaped change (represented with a quadratic contrast) observed in individuals with higher ADHD traits is an indication that disengagement is slower with the first presentation of an image (novel presentation), then disengagement quickens at the second presentation, followed by slowing down again at the third presentation. This pattern is not exactly what we initially hypothesised, which was that disengagement would show a steeper decline across three presentations; however, the trend that emerged from the data reflects an alternative pattern. This pattern reflects a more variable disengagement pattern that does not modulate in a linear pattern in response to linear decline in informational value of the stimuli. It can be argued that this in a way still represents the neural gain model proposed by Hauser et al., (2016) such that, due to the weak differentiation between signal and noise in ADHD, their disengagement speeds across the repetitions changes in a more unstable pattern. This challenges the initial view that disengagement in ADHD would be characterised by faster disengagement, instead it represents a more dysregulated pattern where disengagement is slower at both ends of the spectrum in relation to information gain. This is also aligned with models describing ADHD as condition of cognitive dysregulation and variability (Castellanos et al., 2005).

*Conclusion and follow-up study*

In summary, this study used a novel gap-overlap paradigm where information gain from the central stimuli were modulated in a large adult sample. While a gap effect was observed, unlike the previous adolescent sample, we did not replicate a linear influence on disengagement due changes in informational content from the stimuli. We propose that this may be due to either too-much-gain in the first instance of presentation or due to too-little-gain across the presentations of the images. To test these competing explanations, we designed two follow-up experiments. In Experiment 2 we reduce the potential for information gain by decreasing the stimulus display time from 1000ms to 500ms, forcing a 50% reduction in information acquisition time from the central stimulus. Conversely, in Experiment 3 we increase the information gain potential by blocking presentation of the repeating images such that every repetition occurs immediately following previous presentation. This design will enhance the salience of stimulus repetition, thereby increasing the rate of information gain.

# 5.3 Experiment 2

## 5.3.1 Methods

### 5.3.1.1 Participants

A sample of 41 undergraduate Psychology students were recruited through SONA in return for course credit, aged between 18 – 32 years old (30 females, M age = 20.1, SD age = 3.0). In this sample, one participant self-reported as diagnosed with ASD, one participant with ADHD, and one participant with ASD, ADHD, OCD and anxiety. All participants reported normal or corrected-to-normal vision and procedure was approved by the Research Ethical Committee of the University of East Anglia, Norfolk, United Kingdom.

Here we acknowledge that this sample is less than half the size of Experiment 1, which limits the statistical power to detect subtle effects and represents a limitation in interpreting null findings. This reduction in sample size was due to time constraints in the academic calendar and the exploratory nature of the follow-up experiments.

### 5.3.1.2 Design

The design of this experimental paradigm was similar to the previous chapter with condition (gap and overlap) and presentation (U, R1, R2, R3) as the independent variables, and the SRT and GE as dependent variables. The key manipulation here were the timings of stimuli presentations are shortened. So instead of participants viewing all images for 1000ms, this has been shortened to 500ms. The 'gap' has been shortened to 200ms and the 'overlap' also occurs for 200ms. This is presented in *Figure 5.2.1*. This version of the task serves as the *low-information-gain* adaptation of the originally paradigm. The variations of the paradigm are described in *Appendix E*.

In this sample, we use the Autism quotient (Baron-Cohen et al., 2001), Broad autism phenotype questionnaire (Hurley et al., 2007), Wender Utah scale (Ward, Wender, & Reimherr., 1993), and the Adult self-report scale (Kessler et al., 2005).

Participant's saccadic reaction times (SRT) are measured for each trial, and a gap effect (GE) is calculated from the difference between gap conditions and overlap conditions. Blocking of trials, counter balancing, and stimuli swapping is conducted similarly to previous version of the task.

#### 5.3.1.3 Material

*See Chapter 4, subsection 4.2.3*

#### 5.3.1.4 Eye-tracking analysis

*See Chapter 4, subsection 4.2.4*

#### 5.3.1.5 Procedure

*See Chapter 5, subsection 5.2.1.5*

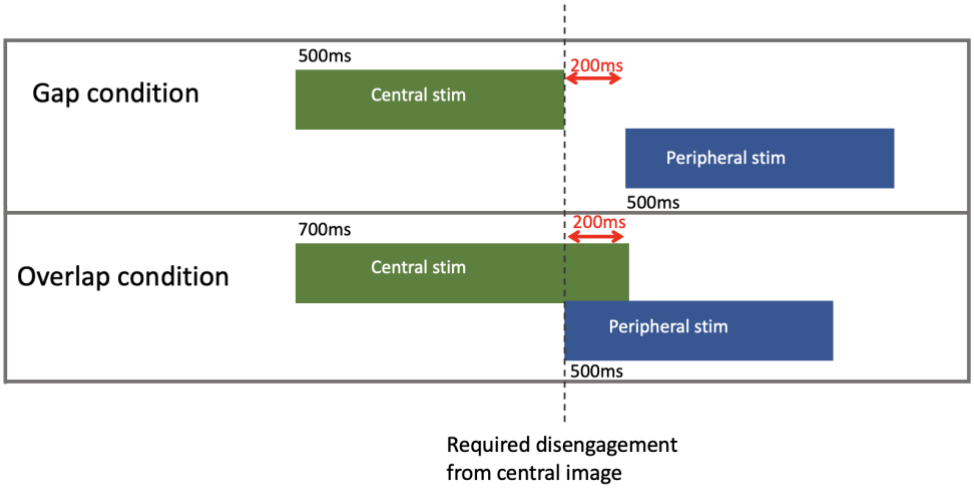



*Figure 5.2.1* Represents the proposed experimental design, here in the gap condition the central stimulus is presented for 500ms, followed by a 200ms temporal gap, then the peripheral image is presented for 500ms. In the overlap condition there is an overlap of 200ms between the central and peripheral stimulus. Presentation time of the central image is 700ms in the overlap condition however participants are expected to disengage at 500ms as soon as the peripheral image is presented.

## 5.3.2 Results

### 5.3.2.1 Descriptives of traits measures

Spreads in the individual differences scores were looked at first for this sample of 41 adult participants. Shapiro-Wilk tests showed raw scores on the BAPQ, AQ, ASRS, and WURS, to be normally distributed (W (41) = .98, $p$ = .78, W (41) = .96, $p$ = .15, W (41) = .98, $p$ = .48, W (41) = .97, $p$ = .25 respectively). Descriptive statistics reveal that the range was varied across all the tests of individual differences (*Table 5.2.1*).

Data retention table is presented in *Table 5.2.2* which shows comparable trial retention between gap and overlap conditions.

*Table 5.2.1*

*Participant characteristics and descriptives from individual differences surveys*

| | *Mean* | *Std. Dev* | *Minimum* | *Maximum* | *Range* |
|---|---|---|---|---|---|
| **Age** | 20.2 | 3.0 | 18 | 33 | 15 |
| **AQ** | 20.7 | 6.3 | 6 | 31 | 25 |
| **BAPQ** | 3.1 | .59 | 1.8 | 4.7 | 2.9 |
| **WURS** | 38.8 | 19.9 | 5 | 91 | 86 |
| **ASRS** | 8.8 | 4.5 | 0 | 18 | 18 |

*Table 5.2.2*

*Overall data retained after removing invalid trials*

| | *% trials retained* | *No. trials* | *Std. Dev* |
|---|---|---|---|
| **Gap condition** | 80.7% | 53.3 *(out of 66)* | 5.7 |
| **Overlap condition** | 87.5% | 57.8 *(out of 66)* | 4.1 |
| **Total** | 84.2% | 111.1 *(out of 132)* | 4.8 |

In this sample, total z-scored ASD traits and ADHD traits are strongly correlated with one another ($r$ (40) = .53, $p$ < .001). This is the same finding as the adolescent sample and in the adult sample from experiment 1.

To test the difference between SRT in gap conditions (M = 222.05, SD = 15.95) and overlap conditions (M = 223.51, SD = 24.3), a paired samples *t-test* was used. Interestingly, in this experimental set up we report that there was no significant difference between SRT in the two conditions ($t$ (40) = 1.06, $p$ = .30).

Although the trend was in the expected direction, with longer RTs for the overlap than the gap trials, this indicates that the paradigm did not work as expected, and that the disengagement cost in the overlap condition was not significantly different to the disengagement cost in the gap condition. To look at this further, paired samples *t*-test was conducted on each trial type (U, R1, R2, R3, and R4), revealed that the trends in the data were in the expected direction, however there were no significant differences between the gap and overlap conditions in any of the trial types.

The paradigm did not produce the expected disengagement cost difference, nevertheless, to ensure consistency in reporting across experiments in this chapter, we conduct the same analysis of the main dependent variable.

#### 5.3.2.2 The impact of repetitions and traits on SRTs

Firstly, a 2 (condition: gap, overlap) x 3 (presentation: first, second, third) repeated measures ANOVA is used. This revealed no significant effect of condition ($F$ (1, 40) = .98, $p$ = .33) but interestingly, there is a significant effect of repetitions ($F$ (2, 80) = 6.12, $p$ < .01). This effect is also apparent with a linear contrast $F$ (1, 40) = 9.49, $p$ < .01). Overall SRT between the three presentations when gap and overlap conditions are combined is presented in *Figure 5.2.3* and it is apparent that reaction time quickens across the trials across the three presentations.

The two individual differences measures (z-scored ASD and ADHD traits) were then added as covariates into the model. First, ASD traits are added which reveals that there is a significant interaction between condition and ASD traits ($F$ (1, 39) = 5.43, $p$ = .03). This shows that the differences between gap and overlap conditions are modulated by ASD scores. This can be visualised in *Figure 5.2.4a* where it is apparent that the gap effect is more apparent in participants with higher scoring versus lower scoring ASD. There was no

interaction between ASD and repetitions ($F$ (2, 78) = .97, $p$ = .39), and no significant interaction between condition, repetition and ASD traits ($F$ (2, 78) = 2.54, $p$ = .09). There was also no main effect of ASD ($F$ (1, 39) = 2.49, $p$ = .12). ADHD traits are then added into the model without ASD traits revealing no significant interaction between condition and ADHD traits ($F$ (1, 39) = .03, $p$ = .86), and no significant interaction between repetition and ADHD traits ($F$ (2, 78) = 1.24, $p$ = .30). There is a significant three-way interaction between condition, repetition and ADHD traits ($F$ (2, 78) = 4.30, $p$ = .02), This is visualised in *Figure 5.2.4b* where we can see lower scoring ADHD group to have a stronger gap effect across presentations, whereas for higher scoring ADHD, the gap effect differs greatly between the presentations. There was also no significant main effect of ADHD traits in this model ($F$ (1, 39) = 3.27, $p$ = .08).

Lastly, both ASD and ADHD traits are added together into the ANCOVA. The model confirms that there is still no significant main effect of condition, ($F$ (1, 38) = 1.15, $p$ = .29) but there is a significant effect of repetitions when ASD and ADHD traits are controlled for ($F$ (2, 76) = 6.04, $p$ < .01). This effect is also apparent with a linear contrast $F$ (1, 38) = 9.45, $p$ < .01). There were no main effects of ASD ($F$ (1, 38) = .57, $p$ = .46) nor ADHD in this model ($F$ (1, 38) = 1.30, $p$ = .26). However, an interaction emerged between condition and ASD traits ($F$ (1, 38) = 8.52, $p$ = .01). This is presented in *Figure 5.2.2c* where participants are split by high/low median split groupings based on their ASD traits in order to visualise the difference of condition (gap versus overlap). This indicates that individuals with high ASD traits exhibited a larger difference between the gap and overlap conditions. To check this further, a paired-sample *t-test* on only the low-scoring ASD group reveals no significant difference between gap and overlap condition ($t$ (20) = 1.24, $p$ = .23), whereas the paired-sample *t-test* on only the high-scoring ASD group reveals a significant difference between the gap and overlap condition ($t$ (19) = 2.59, $p$ = .02). This indicates that there was a gap effect present in the high-scoring ASD group within our sample, even in the case that it is not apparent overall across the sample.

In order to check whether there were any overall repetition effects – that may be attributed to improved reaction times – SRT for each repetition in both gap and overlap condition is averaged for every participant. Repeated measures ANOVA shows that there is a significant change in SRT across the three presentations of the stimuli ($F$ (2, 80) = 6.12, $p$ < .01). This shows that in this sample overall reaction times of participants increased across an

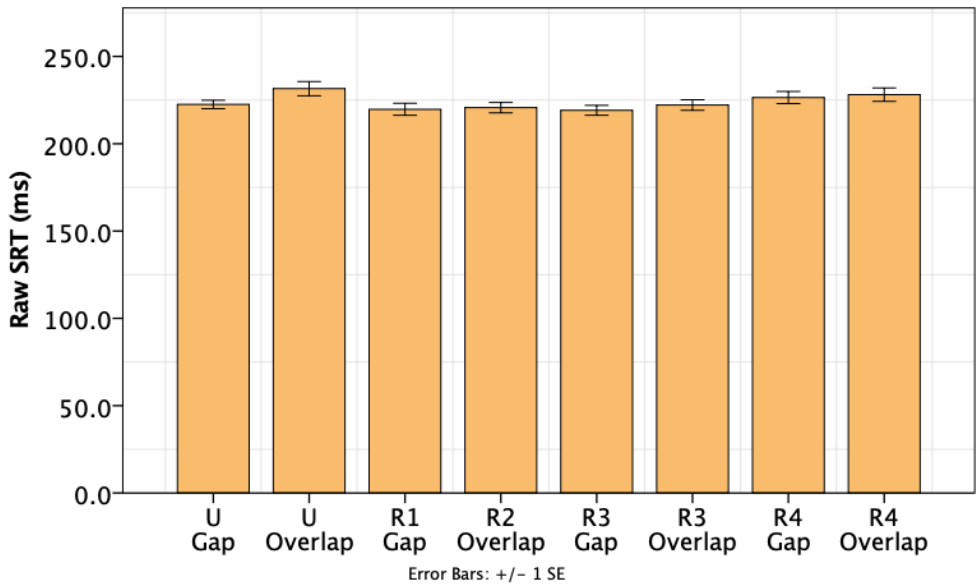
experimental block. This is also apparent and significant in the linear contrast ($F$ (1, 40) = 9.49, $p$ < .01) and is presented in *Figure 5.2.3*.

#### 5.3.2.3 The impact of repetitions and traits on Gap effects

Beta values which represent the slope of change in GE from presentation 1 until presentation 3 were extracted for each participant. Using a one-sample *t-test,* we report that across the sample, the slope of change was not significant different to test statistic 0 ($t$ (40) = .30, $p$ = .79). This tells us that there was no systematic change across the repetitions. The slope of change was also not significantly correlated with ASD ($r$ (40) = -.27, p = .09), nor ADHD traits ($r$ (40) = -38, p = .08), however from the $p$ values of the correlation, there does seem to be a trend for both ASD and ADHD traits. Specifically, that more negative slopes were associated with higher scores on ASD and ADHD traits.

ASD traits and ADHD traits were added as covariates into a repeated measures ANCOVA conducted on the GE from the three repetitions of the image. The results showed no significant changes in GE across the three repetitions ($F$ (2, 76) = .47, $p$ = .63). There was a main effect of ASD traits in this model ($F$ (1, 38) = 8.53, $p$ <.01) which indicates that there was a significant difference in overall GE as it relates to ASD traits. This corresponds with the previous finding where high scoring ASD participants showed a significant difference between gap and overlap trials, therefore presenting a more robust GE whereas lower scoring ASD group did not present a robust GE. As for ADHD, there was no main effect in relation to overall GE ($F$ (1, 38) = 2.87, $p$ = .10). There was no interaction between GE across the repetitions and ASD ($F$ (2, 62) = .53, $p$ = .58) nor ADHD traits ($F$ (2, 62) = 2.2, $p$ = .12).

Overall GE was not correlated with ADHD traits ($r$ (40) = .05, p = .80) however a positive correlation emerged for ASD traits ($r$ (40) = .39, p = .01) showing that higher ASD traits were correlated with a larger GE.

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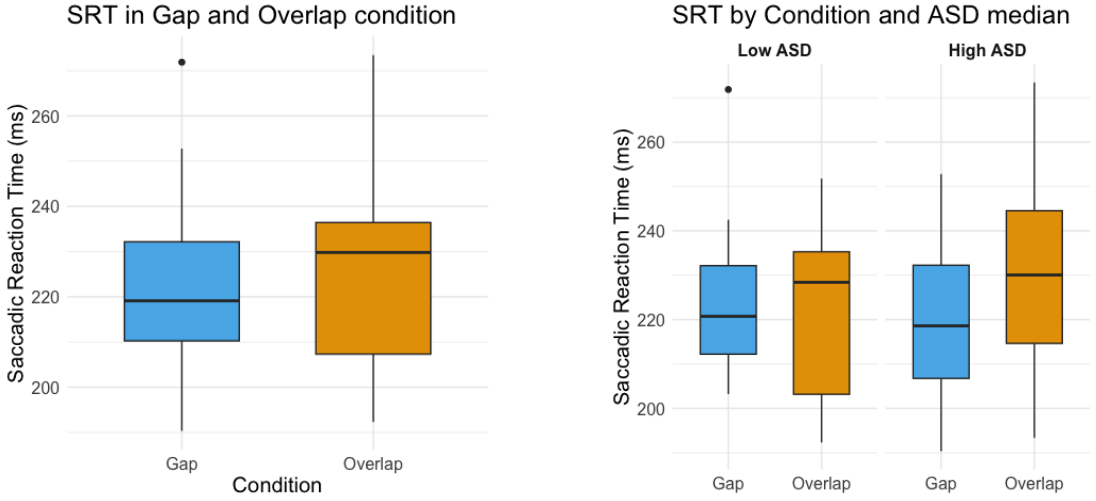



*(b)* *(c)*

*Figure 5.2.2 (a)* Raw saccadic reaction times (SRT) for each trial type and each condition. In later analysis U trials and R1 trials are combined and R4 trials are excluded. *(b)* Average SRT for gap and overlap conditions shows that there is more variation within the overlap condition, however SRT both conditions is comparable and therefore no gap effect emerged. *(c)* Average SRT for gap and overlap conditions between high and low scoring ASD groupings in order to illustrate the presence of a gap effect in the high-scoring ASD group, which is not present in the low-scoring ASD group. Participants are median split into high versus low-scoring ASD groupings for visualisation although the repeated measures ANCOVA had ASD traits as a continuous variable.

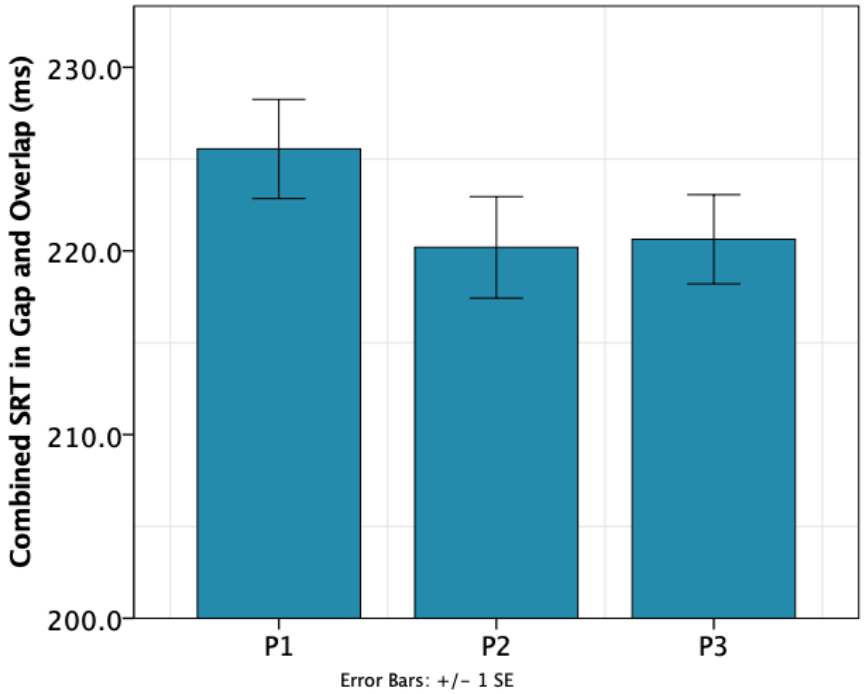
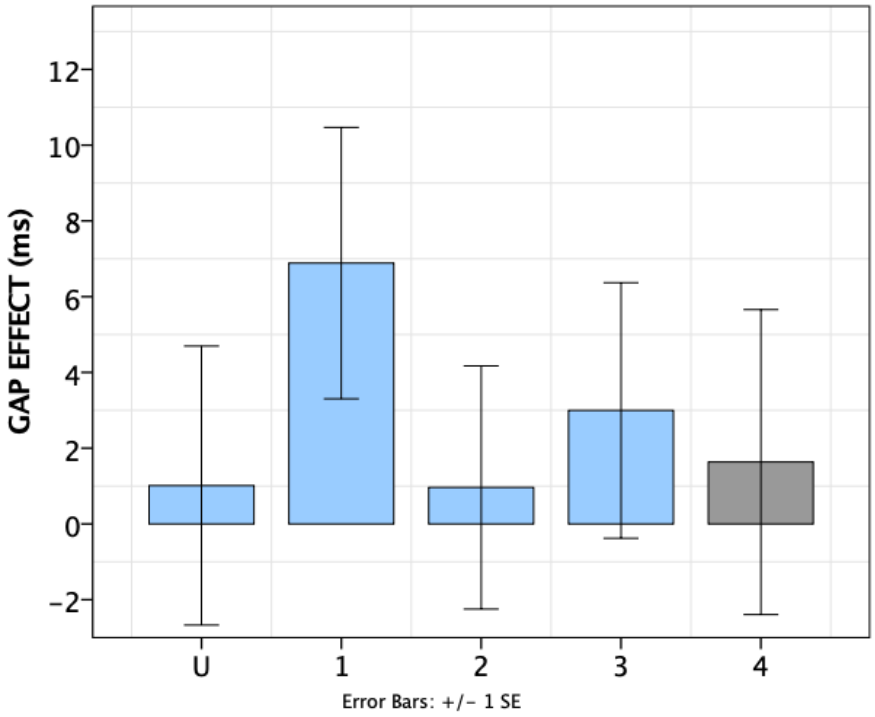



*Figure 5.2.3* Average SRT for combined gap and overlap conditions for each presentation type is illustrated to see broad effect on reaction time. Note that the *y*-axis starts from 200ms, this is to see clearer the variation in the data showing a significant linear change.

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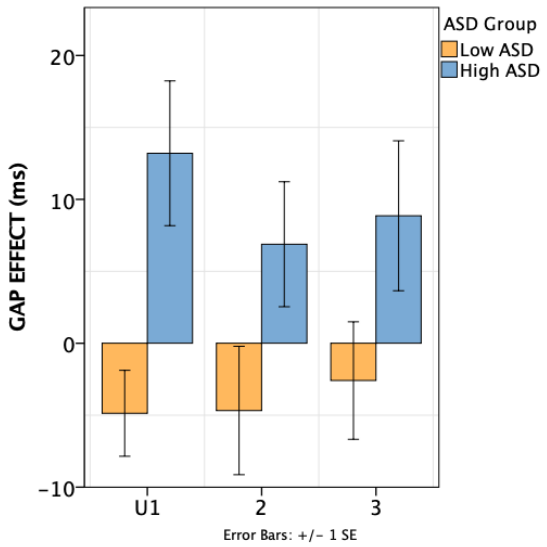


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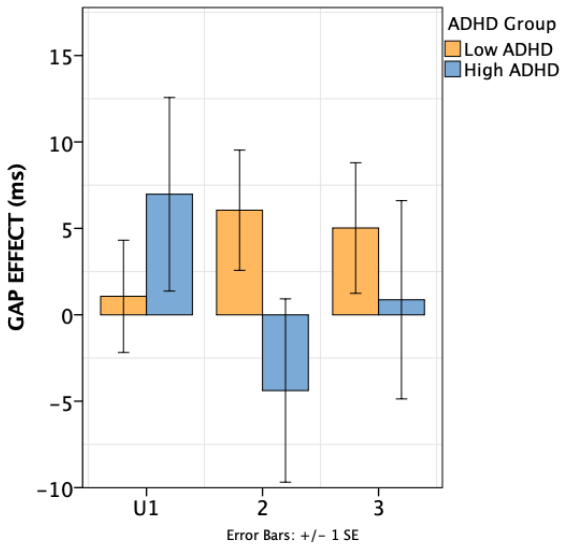



*(c)*

*Figure 5.2.4* (a) The gap effect (GE) for all trial types. The graph shows comparable GE for U, R1, R2, and R3 trials, and that there is no overarching trend of decreasing GE. (b) First, second, and third presentation for ASD median split groupings is presented. High ASD shows the presence of a GE across the three presentations, whereas the same trend is not there for low ASD group. (c) First, second, and third presentation for ADHD median split groupings shows variable trends in both high and low scoring ADHD.

### 5.3.3 Discussion

The primary objective of experiment 2 was to test whether reducing the information gain potential by shortening the stimulus presentation time from 1000ms to 500ms would alter the pattern of attentional disengagement and its relationship with ASD and ADHD traits. We reasoned that adults may have depleted all information at the first presentation of the stimuli, preventing us from seeing a linear decline with repetition. Contrary to the successful execution of the paradigm in the adolescent sample and in experiment 1 of this chapter, the key finding here is that that the main dependent variable in this paradigm – that is the gap effect – did not emerge. There was no statistically significant difference in disengagement cost between the gap condition and the overlap condition when looking at the participants overall. This indicates that the manipulation of shortening the presentation times has fundamentally altered the perceptual experience of the participants and the cognitive processes that are otherwise robust in such paradigms (ie: that in overlap condition, disengagement cost is higher).

The absence of a significant gap effect suggests that the combination of shorter central stimulus presentation (500ms) in addition to a shorter overlap period (200ms) may have fundamentally altered the dynamic of the paradigm such that it attenuated the competitive nature that presents in the overlap trials. In the adolescent sample as well as in experiment 1 of this chapter, average SRTs hovered around 200ms-300ms, we report similar SRTs in this sample of participants, except without variation between conditions and without variation between the repetitions of images. The previous paradigm – with 300ms as the overlap period – ensured that the competition between central stimulus and peripheral stimulus remained for a longer duration enough to elicit a measurable disengagement cost. Reducing the overlap period to 200ms likely affected this critical window of competition, resulting in similar SRT responses between gap and overlap conditions. It is possible that in many trials, or among many participants, the central stimulus disappeared before the disengagement process started. This manipulation was designed with the intention to reduce information gain, however, may have inadvertently reduced competitive dynamic required to produce a robust gap effect. Indeed, in the work by Fischer et al., (2014; 2016), they used 1000ms or 2000ms for presentation for central image, and 3000ms as the overlap period. Wilson and Saldaña (2019) had a 1500ms overlap period, McLaughlin et al., (2021) had a 1500ms overlap period, and Kawakubo et al., (2007) had a 2000ms overlap period.

There were several notable findings from the results of this sample, for example the main effect of repetition emerges when controlling for ASD and ADHD traits in a model that has the gap and overlap conditions entered separately. It is more likely that the linear changes in repetitions from this model reflect the speed of orienting to the peripheral image increasing over time in the experiment, not necessarily due to a change in disengagement cost in relation to the informational content of the central repeated images due to these changes not translating into a significant modulation of the gap effect.

Another finding key finding here relating to individual differences was that there was a significant difference between gap and overlap conditions in individuals who scored higher in their ASD traits. Subsequent analysis on median split groupings of high versus low scoring ASD, revealed that GE was present and robust in participants who scored higher in their ASD traits. In addition, there was also a significant positive correlation between overall GE and ASD traits, which was no observed in any other experiment. This finding is not what we had predicted, however this finding of longer disengagement with higher ASD traits has been the key prediction of research before ours and has also been reported in several works (Sacrey et al., 2014; Fischer et al., 2014; 2016; Wilson & Saldaña, 2019; McLaughlin et al., 2021). Taken together it indicates that adults with elevated ASD traits were presenting with a higher disengagement cost in the overlap condition, which may demonstrate higher engagement or persistence with the central image, that is not present in the lower scoring ASD participants. Another key note is that SRTs in this sample hovered around 200ms-250ms, whereas the overlap period (where competition occurs and causes a disengagement latency) was 200ms. This likely suggests that participants with higher ASD traits who demonstrated more robust GE, exhibited a disengagement latency even in the absence of a sufficient competitive window. In other words, the delayed disengagement likely occurred even when participants were disengaging from the central location without the presence of a stimulus. This could be a sign of generally high engagement with the experiment, that was not present in the participants who had lower ASD scores, or it may be linked "high gain" states in ASD which leads to more stable engagement with current stimuli (Hauser et al., 2016; Piccardi & Gliga, 2022). Within the context of this experiment, where there was no overall successful gap effect across the sample and a significant linear change in reaction times (*Figure 5.2.3*) this finding should be interpreted with caution.

The gap-overlap paradigm is designed to measure the specific cognitive and neural process involved in attentional disengagement by requiring participants to shift their gaze

from a central location to a peripheral location (Saslow, 1967). The main strength of this paradigm lies in its within-subjects design, where the gap condition acts as the control baseline for saccadic reaction times, and the overlap condition introduces competition and the need to actively disengage in order to re-orient to the peripheral stimulus. The key dependent variable here is the saccadic reaction time difference between these two conditions, which provides a measure of disengagement that is internally controlled for each participant and accounts for individual variability in reaction time speeds or oculomotor speeds. Due to not seeing an overall gap effect in this study, any other trends in the data, whether significant or otherwise are discounted due to the internal validity of the paradigm being compromised, the findings may reflect noise or type 1 errors.

In summary, experiment 2 demonstrates that attenuating information gain potential by changing the viewing time disrupts the disengagement patterns that are typical in the gap overlap paradigm. It is possible that in this sample traits relating to neurodiversity are playing a role in some of the significant findings from this study however this is overshadowed by the lack of gap effect, and therefore interpretations of these findings are less likely to be reliable. In experiment 3 we implement an opposite approach where we increase the information gain potential of the experiment by blocking presentation of the repeating stimuli.

# 5.4 Experiment 3

## 5.4.1 Methodology

### 5.4.1.1 Participants

A sample of 34 undergraduate Psychology students were recruited through SONA in return for course credit, aged between 18 – 33 years old (27 females, M age = 19.4, SD age = 2.5). In this sample one person reported to have ADHD and obsessive-compulsive disorder, and one participant reported having dyslexia. All participants reported normal or corrected-to-normal vision and procedure was approved by the Research Ethical Committee of the University of East Anglia, Norfolk, United Kingdom.

### 5.4.1.2 Design

The design of this experimental paradigm used the original viewing timings of 1000ms for central and peripheral images. The condition (gap and overlap) and presentation (U, R1, R2, R3) remained the independent variables, and the SRT and GE as dependent variables. The key manipulation here is the blocking of repetitions, such that any repetitions of the images are appearing immediately after each other without any randomisation of trials. This version of the task serves as the *high-information-gain* adaptation of the originally paradigm. Here, the repeated images are presented closer to each other temporally and therefore it is expected that more information is extracted at each repetition of the image when there are no alternative images presented in the sequence order.

In this sample, we use the Autism quotient (Baron-Cohen et al., 2001), Broad autism phenotype questionnaire (Hurley et al., 2007), Wender Utah scale (Ward, Wender, & Reimherr., 1993), and the Adult self-report scale (Kessler et al., 2005).

Participant's saccadic reaction times (SRT) are measured for each trial, and a gap effect (GE) is calculated from the difference between gap conditions and overlap conditions. Blocking of trials, counter balancing, and stimuli swapping is conducted similarly to previous version of the task.

### 5.4.1.3 Material

*See Chapter 4, subsection 4.2.3*

#### 5.4.1.4 Eye-tracking analysis

*See Chapter 4, subsection 4.2.4*

#### 5.4.1.5 Procedure

*See Chapter 5, subsection 5.2.1.5*

## 5.4.2 Results

### 5.4.2.1 Descriptives of trait measures

Spreads in the individual differences scores were looked at first for this sample of 34 adult participants. Shapiro-Wilk tests showed raw scores on the AQ, to violate normality (W (34) = .94, $p$ = .05, and scores on the BAPQ, WURS, and ASRS to be normally distributed (W (34) = .95, $p$ = .11, W (34) = .96, $p$ = .25, W (34) = .96, $p$ = .31 respectively). Data were not transformed here given that the violation of normality in the case of the AQ was borderline and mild. Descriptive statistics reveal that the range was varied across all the tests of individual differences (*Table 5.3.1*) and comparable to the previous sample from experiment 2. Data retention (*Table 5.3.2*) across the two conditions shows that similar number of trials were rejected in gap and overlap conditions.

*Table 5.3.1*

*Participant characteristics and descriptives from individual differences surveys*

| | *Mean* | *Std. Dev* | *Minimum* | *Maximum* | *Range* |
|---|---|---|---|---|---|
| **Age** | 19.4 | 2.5 | 18 | 33 | 15 |
| **AQ** | 20.7 | 8.2 | 6 | 43 | 37 |
| **BAPQ** | 3.2 | .67 | 1.9 | 5.2 | 3.3 |
| **WURS** | 37.7 | 20.1 | 2 | 73 | 71 |
| **ASRS** | 8.6 | 4.7 | 0 | 17 | 17 |

*Table 5.3.2*

*Overall data retained after removing invalid trials*

| | *% trials retained* | *No. trials* | *Std. Dev* |
|---|---|---|---|
| **Gap condition** | 88.3% | 58.3 *(out of 66)* | 5.3 |
| **Overlap condition** | 91% | 60.1 *(out of 66)* | 4.6 |
| **Total** | 89.7% | 118.4 *(out of 132)* | 5.0 |

In this sample, there was a significant correlation between z-scored total ASD and ADHD traits ($r$ (33) = .37, p = .03).

To test the difference between SRT in gap conditions (M = 229.15, SD = 26.84) and overlap conditions (M = 260.43, SD = 38.83), a paired samples *t-test* was used, and we report a significant difference between SRT in the two conditions ($t$ (33) = 5.74, $p < .001$). This confirms the paradigm worked as expected, and that there is a higher disengagement cost associated with the overlap condition as opposed to the gap condition.

#### 5.4.2.2 The impact of repetitions and traits on RTs

In order to test the relationship between gap and overlap conditions, across the three repetitions, a 2 (condition: gap, overlap) x 3 (presentation: first, second, third) repeated measures ANOVA is used. This revealed a significant effect of condition ($F$ (1, 33) = 30.85, $p < .001$) and a significant effect of repetition ($F$ (2, 66) = 5.79, $p < .01$). There was no significant interaction between condition and repetition ($F$ (2, 66) = 2.6, $p = .08$).

Z-scored ASD traits were then added into the model as a covariate, revealing no significant interaction between ASD and condition ($F$ (1, 32) = .19, $p = .66$), no significant interaction between repetition and ASD, ($F$ (2, 64) = .77, $p = .47$), and finally, no three-way interaction between condition, repetition and ASD traits ($F$ (2, 64) = .22, $p = .81$). There was also no main effect of ASD ($F$ (1, 32) = .93, $p = .34$). ADHD traits were then added next into the model without ASD traits. Similar to the findings in with ASD traits, there was no significant interaction between condition and ADHD ($F$ (1, 32) = 1.81, $p = .19$), no significant interaction between repetition and ADHD, ($F$ (2, 64) = .58, $p = .56$), and finally, no three-way interaction between condition, repetition and ASD traits ($F$ (2, 64) = 2.15, $p = .13$). There was also no main effect of ADHD ($F$ (1, 32) = .92, $p = .34$).

The two individual differences measures (z-scored ASD and ADHD traits) were then added together as covariates into the model. As expected, the model showed that when individual differences were controlled, a significant effect of condition still emerged, ($F$ (1, 31) = 30.62, $p < .001$), indicating that SRT differed significantly between the gap and overlap conditions. In this model, there is also a significant main effect of repetition ($F$ (2, 62) = 5.80, $p = .01$), indicating that disengagement did differ across the three repetitions. Notably the change in the three repetitions is also significant with a quadratic contrast ($F$ (1, 31) = 8.69, $p < .01$). This indicates there is an overall change in the SRTs across the block of experiment that likely reflects changes in reaction times. This is presented in *Figure 5.3.1c.*

There was no significant main effect of ASD in this model ($F$ (1, 31) = .28, $p$ =.60) and no main effect of ADHD ($F$ (1, 31) = 1.17, $p$ =.29). There were also no significant interactions between conditions nor repetitions and the covariates entered into the model.

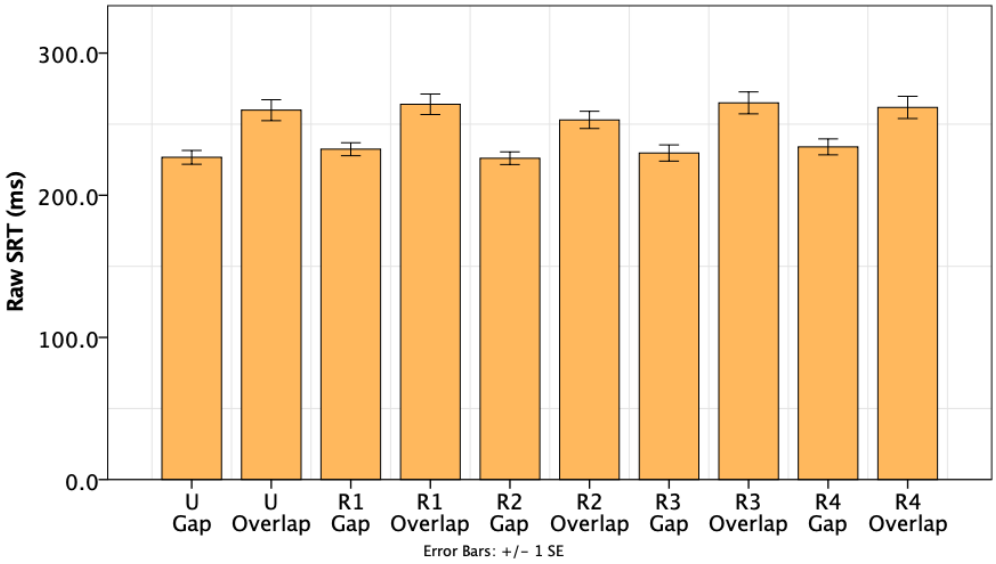
#### 5.4.2.3 The impact of repetitions and traits on Gap effects

Gap effect (GE) was calculated for each trial type (U, R1, R2, R3, and R4) by subtracting the SRT in gap condition from the SRT in overlap condition for each trial type. A total GE was also calculated representing the overall average disengagement cost across the experiment. In this adult sample of participants, overall GE was not correlated with z-scored ASD traits ($r$ (33) = .04, p = .84) and not correlated with z-scored ADHD scores ($r$ (33) = .20, p = .26). This corroborates with findings from the adolescent sample finding no overall disengagement deficits or differences associated with ASD and ADHD traits.

Beta values which represent the slope of change in GE from presentation 1 until presentation 3 were extracted for each participant. Using a one-sample $t$-test, we report that across the sample, the slope of change was not significant different to test statistic 0 ($t$ (33) = .88, $p$ = .39). This tells us that there was no systematic change across the repetitions. The slope of change was also not correlated with z-scored ASD traits ($r$ (33) = .11, p = .52) nor with ADHD traits ($r$ (33) = -.09, p = .62).

A repeated measures ANCOVA was conducted to test changes in GE across the three repetitions of the image. Z-scored ASD and ADHD traits were also added as covariates into the model. Interestingly, there were no significant changes in GE across the three repetitions ($F$ (2, 62) = 2.64, $p$ = .08) when ASD and ADHD traits are controlled for. The quadratic contrast for changes in GE across the repetitions also shows a strong trend, however, falls short of reaching significance statistically ($F$ (1, 31) = 3.96, $p$ = .06) and the linear contrast does not show any notable relationship ($F$ (1, 31) = .75, $p$ = .39).

There were no main effect of ASD ($F$ (1, 31) = .003, $p$ =.96), nor ADHD ($F$ (1, 31) = 1.57, $p$ =.22). There is also no significant interaction between GE across repetitions and ASD traits ($F$ (2, 62) = .43, $p$ = .66) nor ADHD traits ($F$ (2, 62) = 2.31, $p$ = .11) in this model. An interesting trend however is the quadratic contrast in the interaction between repetitions and ADHD traits which – although not significant – echoes a similar trend as experiment 1 of this chapter ($F$(1, 31) = 3.46, $p$ = .07), however in this case, it is lower scoring ADHD traits that exhibit a more quadratic change across the three repetitions whereas higher scoring ADHD participants exhibit a more stable change across repetitions.

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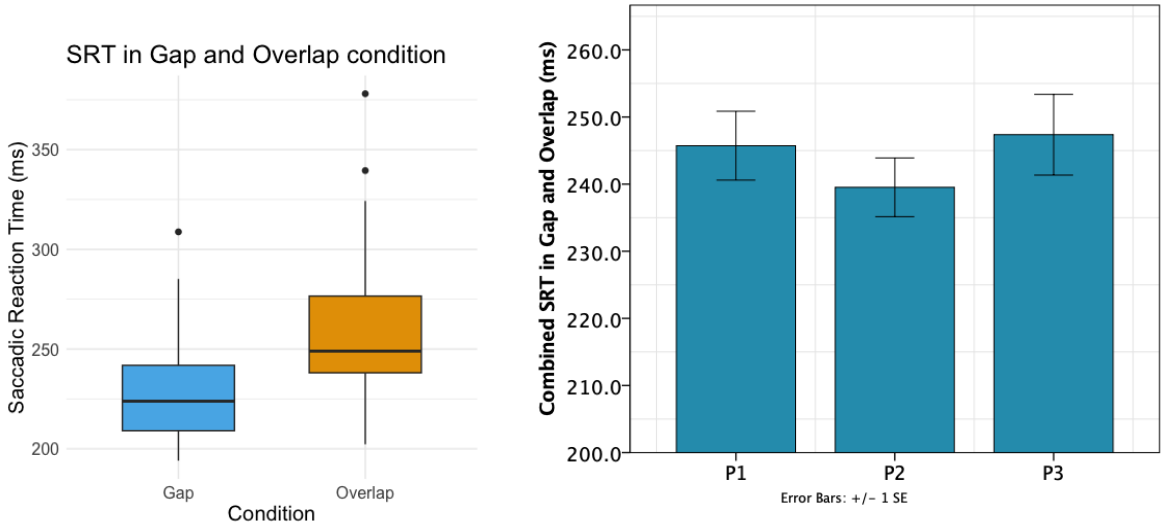
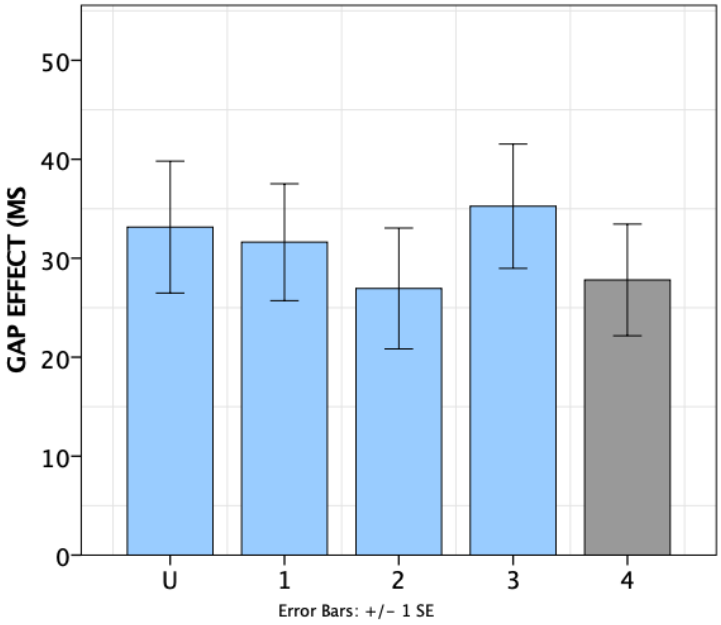



*(b)* *(c)*

*Figure 5.3.1 (a)* Raw saccadic reaction times (SRT) for each trial type and each condition. From this illustration, all the gap trials and overlap trials seem to be stable across different trial types. In later analysis U trials and R1 trials are combined and R4 trials are excluded. *(b)* Average SRT for gap and overlap conditions shows that there is more variation within the overlap condition and that SRT in the overlap condition is reliably higher compared to gap. *(c)* Average SRT for combined gap and overlap conditions for each presentation type is illustrated to see the broad effect of repetition on reaction time. Note that *y*-axis starts from 200ms, this is to see clearer the variation within the data.

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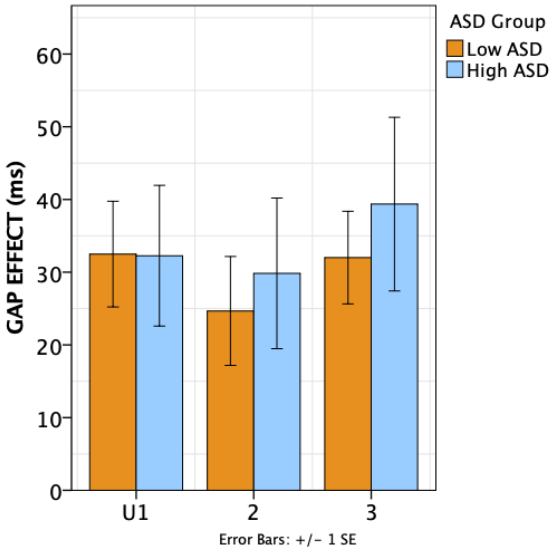


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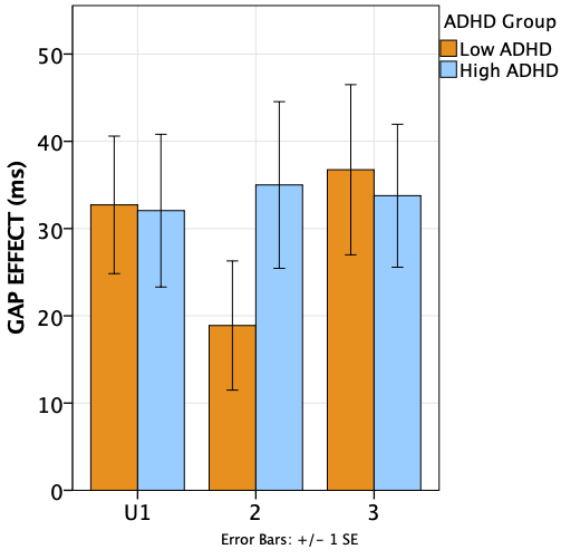


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*Figure 5.3.2. (a)* The gap effect (GE) for all trial types. The graph shows comparable GE for U and R1, followed by a decline in disengagement cost in R2 and then an increase in R3. There seems to be an overarching quadratic trend in GE. *(b)* First, second, and third presentation for ASD median split groupings is illustrated. High ASD and low ASD show similar quadratic trends. *(c)* First, second, and third presentation for ADHD median split groupings shows that low scoring ADHD group have a quadratic trend of change across the three presentations however high scoring ADHD have a more stable GE across the presentations.

## 5.4.3 Discussion

We reasoned that the lack of a repetition effect in Experiment 1 may have resulted from the delayed presentation of the 2nd and 3rd repetitions of images. While know that adults integrate information across time (Guillory & Kaldy, 2019), the delays between trials in our study may have been longer than ideal, resulting in too much forgetting between presentations. Experiment 3 was designed to increase information gain potential by blocking the presentations of repeated images, thereby allowing participants to accumulate the information extracted from each stimulus at each trial, with minimal forgetting. This manipulation successfully elicited a robust gap effect, confirming the paradigm worked as expected. The key results from this study point towards a more complex, non-linear relationship between information gain and disengagement cost due to the manipulations implemented in this design.

The most compelling finding was the significant main effect of repetition along with a significant quadratic contrast when analysing SRT across conditions. This indicates that disengagement speed did not change in a simple linear fashion but rather, it followed a V shaped pattern across the three presentations. This V shaped pattern was also present when looking at the GE changes in study, however it fell short of reaching significance. This is in contrast with the linear decline found in the adolescents sample and suggests that adults with a more matured attentional system (Luna et al., 2004), have a more complex relationship between information value and disengagement. It is possible that this blocked design allows for more extensive processing, or exhaustive encoding of the image by the second presentation, and therefore disengagement speed quickens as habituation occurs. By the third presentation, it is possible that the stimulus has become overly familiar, potentially triggering different cognitive processes that relate to deeper processing (Friston, 2005). This non-linear pattern reflects a more dynamic information foraging strategy than a simple linear depletion model.

From the analysis on the GE, the effect of repetitions did not reach significance, however the quadratic trend that was present in the SRT analysis is also present here which suggests an interesting phenomenon. This pattern is similar to the V-shaped effect of ADHD traits found in experiment 1 with a larger sample size. A similar trend emerged here however in experiment 1 higher ADHD traits associated with a stronger quadratic curve, whereas in this sample, lower ADHD traits associated with a stronger quadratic curve. Results from

experiment 1 therefore indicate that ADHD is characterised by a more variable and unstable disengagement pattern, whereas in this sample the data shows a more stable disengagement in ADHD.

Consistent with the adolescent sample and adult Experiment 1, we again found no overall correlation between GE and ASD or ADHD traits. This reaffirms the core conclusion that basic disengagement mechanisms, as measured by the overall gap effect, are intact in adults with varying levels of neurodiverse traits. The effects of neurodiversity are not necessarily in the overall cost of disengagement, but in the pattern of how disengagement costs change over time as information is gathered.

In conclusion, experiment 3 reveals that enhancing information extraction through a blocked design unveils a non-linear, quadratic pattern of disengagement in adults. This suggests that the information seeking processes involve a more complex calculation than a linear response to linear information depletion. It is likely that varying stages of encoding and processes are involved when processing information in a high information gain set up.

## 5.5 General discussion: Eye-tracking, adult sample

This chapter extended the findings from our adolescent sample into adulthood, using a larger sample size in the original replication of the study and following up with smaller samples to test variations of the experiment. Across these three experiments, we attempted to systematically manipulate information gain potential within a classic gap-overlap paradigm to explore how neurodiverse traits modulate patterns of attentional disengagement.

Experiment 1 (n=98) replicated the basic gap-overlap effect but did not replicate the linear decline in disengagement cost across repetitions observed in adolescents. Instead, we found a significant non-linear (quadratic) pattern of disengagement associated with higher ADHD traits. Experiment 2 (n=41), which reduced information gain through shorter presentation times, failed to produce a significant gap effect overall, although individuals with higher ASD traits showed a more robust gap effect. Experiment 3 (n=34), which enhanced information gain through blocked presentations, produced a successful gap effect and revealed a quadratic trend in disengagement across repetitions. Taken together, these findings demonstrate that the relationship between information value and attentional disengagement in adults is more complex than in adolescents, and our exploratory testing highlights the sensitivity of the gap-overlap paradigm to methodological variations.

One pattern that appeared across experiments is the quadratic (V-shaped) across disengagement. Rather than the simple linear decline in response to lower information gain across repetitions, we saw on several occasions that disengagement speed first increased, then decreased, and then increased again. This pattern may reflect processes that we did not initially anticipate, for example it is possible that the first presentation provides novel information, resulting in slower disengagement, followed by the second presentation which is offering less novel information resulting in faster disengagement, however by the third presentation, the stimulus has become overly familiar, potentially triggering deeper processing or memory retrieval. Alternatively, from a Bayesian perspective (Parr & Friston, 2017), the third presentation may itself become surprising because participants do not expect the image to appear again, leading to renewed engagement and slower disengagement.

Across all three experiments, there was no consistent evidence of an overall disengagement deficit associated with either ASD or ADHD traits. Overall GE was not correlated with trait scores. This supports the conclusion that basic attentional disengagement mechanisms are intact in neurodiverse populations, aligning with studies reporting no group

differences in gap-overlap paradigms (Fischer et al., 2014, 2016; Goldberg et al., 2002; Hanisch et al., 2006). However, the pattern of disengagement across repetitions did show associations with neurodiverse traits. In experiment 1, a significant quadratic interaction emerged between ADHD traits and SRT across presentations. This suggests that neurodiversity may influence the dynamics of information processing rather than the overall speed or cost of disengagement. Individuals with higher ADHD traits showed more variable and non-linear patterns, consistent with models characterising ADHD as involving cognitive dysregulation and variability (Castellanos et al., 2005; Hauser et al., 2016).

The three experiments provided important insights into the methodological sensitivity of the gap-overlap paradigm. The lack of an overall gap effect in experiment 2 demonstrates the importance of the duration of the overlap period in creating significant competition between the stimuli which is necessary to measure an active disengagement process. When the overlap period is too short (200ms in experiment 2), the central stimulus may disappear before the disengagement process begins, effectively turning overlap trials into gap trials. This is consistent with previous work using longer overlap periods (1500-3000ms; Fischer et al., 2014; 2016). The blocked design in experiment 3 proved more effective in enhancing the salience of the repetition manipulation, likely because participants could track the repetition more easily and experienced a more pronounced change in informational value. This did, however, result in a quadratic V-shaped pattern of disengagement across the repetitions.

The absence of a linear repetition effect in adults similar to what appeared in the adolescent sample, suggests developmental changes in how information value influences disengagement. Adults showed slightly faster overall SRTs (M = 242.78ms) compared to adolescents (M = 255.90ms), consistent with maturation of prefrontal and dopaminergic systems supporting efficient cognitive control (Luna et al., 2004). This matured system may allow adults to extract information more rapidly, reducing sensitivity to the repetition manipulation. In general, in both the adult samples and the adolescent sample, the lack of an overall disengagement deficit in ASD and ADHD traits supports the view that basic attentional mechanisms are intact, and that differences emerge in the regulation of attention rather than in the fundamental ability to disengage. This is consistent with the neural gain model (Hauser et al., 2016), which posits that differences in ASD and ADHD relate to gain parameters (higher gain in ASD, lower gain in ADHD) influencing the stability and variability of attention, rather than deficits in basic attentional orienting.

Another notable difference between the results in this chapter and the adolescent chapter is the rate of trial rejection specifically in the gap condition. In both age groups, the gap trials lacked a fixation cross during the gap period where there were no stimuli presented on the screen. Here there was no anchor to maintain fixation in the central location. This is important because the condition for an accepted trial is that the participants saccade must start from the central location, in the direction of the peripheral image that appears either on the right or the left of the screen. In the adolescent sample, a large portion of the gap trials were rejected due to participant's gaze moving away from the central location and therefore their saccade towards the peripheral image would be started from locations other than the central location (approximately 40% retention in gap condition, and 90% in overlap condition). In the adult sample however, trial retention was comparable to the overlap conditions (roughly between ~80% - 90% retention in both gap and overlap conditions). This suggests that adults with more mature oculomotor control were better able to maintain central fixation voluntarily, even without an explicit fixation cue. This interpretation is supported by research conducted by Luna et al., (2015), Luna and Sweeney (2004), and Kramer et al., (2005) where in general, for adolescents, they describe a cognitive profile that has reached adult-like maturity in some aspects, however several key top-down control functions continue to develop and improve into adulthood.

A final important note for this chapter is the inconsistency in sample sizes across experiments. While experiment 1 achieved a large sample, experiments 2 and 3 were substantially smaller. A post-hoc power analysis using G Power (Faul et al., 2007) for a repeated measures ANCOVA interaction, assuming a medium effect size ($f = 0.25$), $\alpha = 0.05$, and power = 0.80, suggests approximately 52 participants would be needed. Experiments 2 and 3 therefore had limited power to detect subtle interaction effects, particularly relating to individual differences. In experiment 1 however, the sample was sufficiently large, yet we report an absence of a main effect of repetitions. This suggests that lack of changes in visual disengagement in response to changing informational value, is a genuine null effect reflecting developmental differences rather than insufficient statistical power. Adults did not show the linear decline in disengagement cost observed in adolescents, consistent with maturation of attentional systems (Luna et al., 2004).

### 5.5.1 Limitations

A key limitation of this chapter is the inconsistency in sample sizes across experiments. Our smaller samples therefore had limited power to detect robust effects. As mentioned previously throughout this thesis, there are limitations associated with using an undergraduate sample in research. This population commonly experiences sleep deprivation (Lund et al., 2010), which can exacerbate ADHD-related symptoms (Ottoni et al., 2012), meaning elevated trait scores that do not necessarily reflect presence of underlying neurodevelopmental conditions. Self-report measures also have significant limitations; recent evidence suggests subclinical ASD surveys may not correlate with clinician-rated autistic traits (Banker et al., 2025). Additionally, ASD and ADHD traits show high co-occurrence in non-referred adults (Taylor, Charman & Ronald, 2015; Panagiotidi, Overton & Stafford, 2019), possibly reflecting a general neurodiversity factor or response bias (Panagiotidi et al., 2019). This complicates our interpretation of trait-specific effects and the testing of our hypothesis relating to opposing attentional profiles between ASD and ADHD traits. Finally, the static images used may not have been sufficiently engaging for adults, mirroring concerns from Chapter 3 about stimuli suitability (Piccardi et al., 2021), which may explain why the linear decline observed in adolescents was not replicated.

### 5.5.2 Conclusion

In conclusion, this chapter demonstrates that attentional disengagement in adulthood is influenced by neurodiverse traits in a more complex, non-linear manner, with a consistent quadratic (v-shaped) pattern emerging across repetitions rather than the linear decline seen previously. There were no overall disengagement deficits were associated with ASD or ADHD traits, supporting the view that basic orienting mechanisms remain intact in neurodiverse populations. The presence of quadric trend in the data indicates that attentional disengagement may not be stable across stimuli with varying information gain potential. The methodological sensitivity of the gap-overlap paradigm was also confirmed, with overlap duration and presentation format significantly affecting the presence of a gap effect. Finally, developmental comparisons revealed that adults exhibited faster reaction times and better fixation control compared to adolescents, which we think strongly reflects maturational improvements in cognitive control.

# CHAPTER 6:

## 6. General discussion

## Goal of chapter:

*The aims of this thesis were to explore attentional mechanisms of engagement and disengagement through the lens of information foraging. In addition, we aimed to test theoretical accounts of ASD and ADHD in their attention and information seeking behaviours based on literature that describe ASD and ADHD to present as opposing sides of a continuum in relation to their attentional system. We first discuss and summarise key findings from our experimental paradigms conducted in two samples (adolescents and adults), followed by reviewing our findings in relation to the literature that was outlined in Chapter 1. Specifically, how our findings fit in with the neural gain model, and attentional processes of engagement and disengagement. We also mention that the results from our adolescent sample were more robust, and closer aligned with our theoretical paradigm, more so than the adult sample. Finally, we discuss limitations of our design and give directions for future research building on the findings reported here.*

## 6.1 Summary and key findings

The overarching aim of this thesis was to investigate the principles of information foraging in attentional engagement and disengagement, and to test the hypothesis that individual differences in neurodiverse traits – specifically associated with Autism Spectrum Disorders (ASD) and Attention-deficit/hyperactivity disorder (ADHD) – modulate these processes in accordance with models of neural gain (Hauser et al., 2016; Piccardi & Gliga 2022) and dopaminergic function (Paval 2017; Hills, 2006). To achieve this, a multi-method approach was employed, using EEG and eye tracking experiment across adolescent and adult populations. Both experiments had stimuli designed to manipulate information gain though repetition, thereby simulating depletion of information or of a resource "patch" as outlined in the foraging theory (Stephens & Krebs, 1986; Charnov, 1976).

Based on the works by Landry (2021), Hauser et al., (2016), Paval (2017), and Hills et al., (2015), our hypotheses in relation to individual differences in ASD and ADHD traits were such that the attentional system of these conditions should present at opposite ends of the spectrum. In Landry (2021)'s work, attention switching, task switching, novelty preference, and delay aversion are some examples of areas where research has shown ASD and ADHD to present behavioural patterns at opposing ends of the same spectrum. Landry (2021) relates some of this to the dopaminergic system, however Paval (2017) makes the case that key behaviours in ASD such as repetitive, restrictive behaviours/interests and difficulties in social communication can be traced directly to altered dopaminergic pathways. Further to this, Hauser at al., (2016) explains the neural gain model which implicates the catecholaminergic system – neurotransmitter systems involving dopamine and noradrenaline – as the key system involved. In this neural gain model, ADHD is described as low gain and associated attenuated signal amplifications, impaired suppression of noise which presents as variable and unstable goal allocation. High gain in this model is described as the opposite, where signal differentiation is amplified, and there is more stability in behaviour and choices.

The findings from our experimental works present a more nuanced picture. In Chapter 2, with our adolescent sample, the adapted EEG paradigm (Piccardi et al., 2020) successfully captured the proposed trade-off between engagement with a depleting information source (indexed by declining frontal theta power) subsequent processing of a novel stimulus (indexed by P1 amplitude). This trade-off reflects the core foraging process of exploiting a current course versus exploring a new one (Cohen, McClure, & Yu, 2007; Daw, O'Doherty et

al., 2006). Crucially, the slope of this trade-off was significantly modulated by ADHD traits, but no ASD traits. Individuals with lower ADHD traits exhibited a steeper, more negative slope, indicating a clearer neural distinction between states of engagement and disengagement. This aligns with part of the neural gain model, suggesting that higher ADHD traits are associated with lower gain states, with more unstable goal-directed allocation of resources and shallower disengagement from depleted sources. Although in this sample we did not find ASD to present neural traces that are in line with the proposed low gain states, we do find that lower ADHD traits were associated with higher gain states (Hauser et al., 2016; Piccardi & Gliga, 2022).

In addition, the finding that ASD traits were associated with a flatter decline in theta power indicates persistent engagement with repetitive stimuli suggests an altogether distinct attentional profile in autism, characterised by an altered baseline of engagement not necessarily by the trade-off between engagement and disengagement. This is in support of research showing altered theta activity in ASD (Gliga et al., 2018; Heaton & Freeth, 2016), and can also be linked to differences in prediction error signalling (Gomot & Wicker, 2012; Lawson, Rees, & Friston, 2014). In other words, brain resources continued to be allocated cognitive resources to continue processing a repeating video even as it became highly predictable and offered diminishing information.

Extending these findings, in Chapter 3 we used a subclinical adult sample, and we report results that were significantly different to the ones from the previous adolescent sample. The key theta/P1 modulation was absent, and theta remained stable across video repetitions. Overall, this indicates that the paradigm's ability to elicit the core attentional phenomena that was elicited in the previous sample, may be developmentally sensitive as it was apparent in 10-month-old infants (Piccardi et al., 2020) and also in our adolescent sample, but not in the adult sample of this chapter. It may be the case that maturation of attentional and executive networks alters how information foraging strategies are deployed Luna et al., 2004; Crone, 2009). The notable three-way interaction between change in theta band with ASD and ADHD median split groupings in adults also points towards a complex, potentially counterbalancing interplay between these traits.

In Chapter 4 we use a more direct behavioural measure of attentional disengagement: the gap overlap paradigm, adapted with unpredictable repetitions of stimuli in order to tap into depleting informational value. We predicted that disengagement cost should decrease as

central stimulus becomes familiar and the expected information gain (EIG) diminishes (Gottlib, 2023). This hypothesis was strongly supported in this adolescent sample, and a significant linear decline in disengagement cost emerged across the stimulus repetitions. However, contrary to predictions derived from the neural gain and dopaminergic models, this basic stimulus driven mechanism was not modulated by ASD or ADHD traits in this sample. We took this to indicate that we successfully implemented this novel paradigm but that general disengagement from rich and poor information sources remains intact in neurodiverse young people. We also did not report any overall deficits in disengagement abilities in ASD nor ADHD participants, challenging previous findings and predictions (Fischer et al., 2014 Elsabbagh et al., 2009; McLaughlin et al., 2021; Landry & Bryson, 2004). There was a trend relating to participants with higher ASD traits, that the change in disengagement cost across repetitions was not as steep as participants with lower ASD traits, echoing the sustained engagement seen in the EEG data and potentially reflecting maintained interest in familiar stimuli (Awh, Belopolsky, & Theeuwes, 2012).

Finaly, Chapter 6 investigated disengagement costs in adults across three experiments; one using an identical paradigm to the adolescent eye tracking study, and two variants of the same program with modified information gain. In experiment 1, the linear decline in disengagement cost was not replicated, potentially due to matured adult attentional system which acquires information more efficiently (Luna et al., 2004) or potentially due to less engagement with stimuli, resulting in loosing track of repetitions. In order to test this, we then modified experiment 2 and experiment 3 in information gain. In experiment 1 there were non-linear patterns of disengagement in relation to ADHD, which indicates a more variable dysregulated disengagement system, consistent with models of cognitive variability in ADHD (Castellanos et al., 2005). In experiment 2 and 3 we found that changing information gain potential of the paradigm did not result in similar findings to the adolescent sample. Instead, we found that increasing information gain potential by way to blocking repetitions in experiment 3 resulted in similar results to experiment 1, and lowering information gain potential by way of reducing stimuli presentation and overlap timings in experiment 2 obstructed the presence of the Gap Effect (GE), which was the dependent variable indexing disengagement costs. Interestingly in experiment 3, individuals with higher ASD traits maintained the presence of a GE across all repetitions in this study whereas individuals with lower ASD traits did not. Collectively, the results from our experimental works make several key contributions to the theoretical models of attention and neurodiversity.

Overall, both experimental paradigms were successful in capturing the core processes they were designed to measure in the adolescent populations. In some instances, the paradigms also revealed expected and notable findings as they relate to individual differences in ASD and ADHD traits in accordance with our predicted models.

The adapted EEG paradigm was highly successful in the adolescent sample, our results replicated and extended the key findings from Piccardi et al., (2020), demonstrating a significant inverse relationship between frontal theta and P1 peak amplitude. Furthermore, the emergence of modulation by ADHD traits, as well as the emergence of unique ASD profile of engagement suggests that this paradigm is sensitive enough to capture different engagement profiles associated with ASD and ADHD. Interestingly, through our adaptation and inclusion of novel videos within the sequence of clips, we were able to demonstrate the role of theta-band activity as an index for engagement instead of information seeking as previous thought (Begus, Southgate, & Gliga., 2015, 2016; Piccardi et al, 2021; Piccardi & Gliga, 2022). In the adolescent sample, we successfully simulated a depleting source of information from which participants were progressively

## 6.2 Theoretical implications

### *6.2.1 Neural gain and dopaminergic models*

Across the experiments there has been variable support for the neural gain model proposed by Hauser et al., (2016). Support for this model is clearest in the EEG paradigm, where the modulation of theta/P1 trade-off associated with ADHD traits in adolescents. Low-gain states in this paradigm were predicted to have a shallower slope of modulation, reflecting unstable goal allocation and impaired suppression of noise. We found that ADHD traits perfectly fit into this model, such that individuals with higher ADHD traits had theta/P1 modulation that was not significantly different from 0 (indicating a flat slope) whereas individuals with lower ADHD traits had slopes significantly different to 0 and were significantly negative. This reflected more stability in behaviour, and heightened signal differentiation. In the eye tracking experiments, ADHD continued to be associated with variable responses, eliciting a quadratic change in disengagement cost across the repetitions. Although this was only the case in the adolescent sample from both studies, it lends clear, although partial support for the neural gain model proposed by Hauser et al. (2016) and Piccardi & Gliga (2022).

Extending the model put forth by Hauser et al (2016) with works by Landry (2021), Hills (2006), and Paval (2017), we suggested that ASD may be working as the attentional opposite of ADHD. This is expounded upon with various literature in the review by Landry (2021). However, our proposal that ASD was a simple "opposite" (or high-gain state) of ADHD was not supported. In our experimental paradigms, ASD did not present as behaviours opposite of the ADHD but oftentimes presented as its own unique set of behaviours. For example, where theta/P1 modulation had a negative correlation with ADHD, it was not the case that ASD traits were positively associated with a theta/P1 modulation. Instead of showing a steeper slope, ASD profile was marked by sustained engagement in which was indexed by baseline theta band activity.

This can further be linked to the eye tracking study, where – although there were no strong evidences for individual differences associations – when looking at it as a whole, ADHD tended to show more unstable patterns of disengagement (based on data from experiment 1) which aligns with the neural gain model, however ASD traits did not present as more stable patterns of disengagement in relation to information gain. Instead, ASD (in experiment 2) uniquely associated with presentation of a robust GE, even when the mechanics of the study design did not provide sufficient competitive difference between the gap and overlap conditions. Specifically, with the design of gap-overlap paradigm, it was the case that the overlap period (where competing images are presented at the same time) was too short to elicit a long enough period of competition, creating a larger latency to disengage from central location and orient towards the peripheral location. However, as individuals with higher ASD traits still presented this disengagement latency, it indicates that they may have been engaged in the central location after the disappearance of the image in that location. This pattern of findings suggests that a critical refinement is needed to our original hypotheses relating to opposing attentional profiles. While ADHD is characterised by dysregulated and unstable disengagement choices in the face of depleting sources of information, ASD may be better defined by an altered baseline valuation of stimuli leading to more sustained engagement with specific content regardless of its informational state.

This disassociation implies that the neurobiological mechanisms underpinning these profiles may be distinct rather than diametrically opposed on the single scale. The ADHD profile of unstable engagement is consistent with its association with noradrenergic dysfunction and low neural gain impacting cognitive control and salience detection (Hauser et al., 2016; Cremer et al., 2023). In contrast, the ASD profile of sustained engagement may

align with a global “high gain” state (if it can be argued to include an unstable persistence), however better associates with models proposing atypical dopaminergic function in prediction error signalling. Specifically, it supports theories that repetitive behaviours and interests in ASD may stem from disrupted reward system where predictable, familiar stimuli retain their subjective value and salience (Paval, 2017; Kohls et al., 2013), or that prediction errors are attenuated which slows the process of leaving a stimulus that is no longer informative (Gomot & Wicker, 2012; Lawson, Rees, & Friston, 2014).

Finally, one reason why we may not see neural-gain model related results from the eye-tracking experiment could be due to the model being better suited in explaining higher-level cognitive processes such as decision-making and task-switching than the relatively low-level oculomotor disengagement measured with the eye-tracking study (Hauser et al., 2016).

### *6.2.2 Distinguishing engagement and disengagement*

A critical observation from the findings here is the necessity to disentangle the mechanisms of sustained engagement (reliant on alerting and executive networks described by Posner & Petersen 1990; Esterman & Rothlein, 2019) from those of more active disengagement (a function of the orienting network as described by Posner, 1980). From our experimental paradigms, in adolescents, it appears that individual differences in ASD and ADHD are more reliably associated with neural markers of sustained attention (as measured by the EEG paradigm) in comparison to disengagement of attention (as measured by the eye tracking paradigm).

When comparing between the results from our adolescent sample, the EEG paradigm, which tracked the gradual changes in neural signal directed towards a depleting information source over time, proved to be sensitive to ADHD traits in the modulation between theta/P1 and proved to be sensitive to ASD traits in relation to sustained theta engagement. In contrast, the eye-tracking gap-overlap task, a more direct measure of disengagement and orienting – although revealed strong effects of stimulus repetition – showed far less robust modulation by individual traits. Overall these findings suggest that the low-level, mechanistic ability to disengage attention – a function of the dorsal and ventral frontoparietal networks (Corbetta & Shulman, 2002) – may be largely intact in our neurodiverse sample. When this is mapped onto attentional foraging accounts (Calhoun & Hayden, 2015; Hills 2006), it posits that ASD and ADHD may have a stronger influence on the thresholds for sufficient information gain and how an individual engages with a ‘patch’ they decide to process, rather than the

mechanisms required to execute disengagement saccade once a decision to orient elsewhere has been made.

*6.2.3 The role of development*

From our experiments, we report a stark difference between adolescent and adult samples. Specifically in relation to paradigm functioning, we reported that across the adolescent sample, the EEG paradigm found both: the expected decline in theta band power and the expected theta/P1 modulation. Both of these measures were not reliably present within the adult sample. In addition, in the adolescent sample in the eye-tracking study we report a significant decline in disengagement cost across the repetitions whereas the same was not found in any of the 3 experiments conducted in adults. Additionally, another demonstration of the developmental differences from the eye-tracking study is the contrast between trial retention in adolescents versus adults. In both age groups, the gap condition lacked a fixation cross during the gap period, meaning there was no anchor to maintain central fixation. Adolescent samples showed substantially lower retention in gap trials (~40%) compared to adults (~80-90%), suggesting more mature oculomotor control and voluntary fixation in adults (Luna et al., 2004; Kramer et al., 2005). This improved data quality in adults increases confidence in the adult findings and suggests that the absence of a linear repetition effect is unlikely to be an artefact of poor trial retention for the older participants.

This contrast can be explained in two potential ways. The first explanations is that this may be a result of developmental and maturation of attentional processes and their underlying neural mechanisms in adults which may not be fully matured in adolescence (Luna et al., 2004; Luna 2009). It is possible that the more pronounced effects in the adolescent samples reflect a neural system that is less matured and therefore more sensitive to experimental manipulations and individual differences in traits before it fully stabilises in adulthood. During adolescence, the balance between exploration and exploitation appears to perform optimally, with adolescents displaying more optimal exploratory tendencies compared to both children and adults (Gopnik et al., 2017; Gil-Gómez de Liaño et al., 2022; Lloyd et al., 2023). Thus, it is possible that the robust effects observed in adolescents (the linear decline in disengagement costs in the eye tracking study and the theta/P1 modulation from the EEG study may reflect a neural system that remains sensitive to experimental manipulations before stabilising in adulthood.

The second explanation could be the ecological validity of the stimuli used in our experiments and the motivations of the adult participants. It is possible that our stimuli overall may be better suited to younger participants, keeping them engaged and entertained (especially in the case of the EEG study using animated *Fantasia* as video stimuli), whereas adult participants – mostly undergraduate psychology students – remained unengaged with the experimental set ups. This poses a particular challenge when our theoretical models, and key experimental measures pertain to the processes of attentional engagement and disengagement as they are reliant on internal motivation and arousal based on Esterman and Rothlein (2019)'s model. Therefore the lack of consistent findings in the adult samples may not necessarily reflect an ineffective experimental paradigm, nor lack of influence from individual differences, but may be due to unengaged participants who's interest we were not able to adequately capture in these paradigms.

The use of clinical measures in the adolescent population with a sizable clinical population recruited can also be contrasted with the use of self-report measures of ASD and ADHD in largely non-clinical healthy adult population. While there is benefit to testing in this population, adopting the neurodiversity approach as we have and recruited large adult samples (total N= 173 in adult eye tracking experiments), an important consideration is that self-report measures may not always capture clinically relevant traits (Banker et al., 2025). It has also been reported to be confounded by factors like sleep deprivation (Ottoni et al., 2012). The usage of ADOS-2 and Conners-4 in the adolescent sample provided us with reliable, clinically relevant quantification of individual differences.

## 6.3 Limitations and future directions

As mentioned above a limitation of this work, in relation to the adult participants collected is ecological validity of the paradigms and stimuli used. The use of simplified laboratory tasks have been beneficial when collecting data in adolescents, especially in the clinical group where complicated instructions or responses would not be yield accurate results, however these tasks may have limited the quality of data collected from the adult samples. The *Fantasia* clips may not have been sufficiently engaging for adult participants, and the nature of the stimuli used in the gap-overlap paradigm is unlike regular multi-sensory environments in which we typically forage for information. Future work should employ more age appropriate stimuli, using videos that may engage adult participants or stimuli that are

visually richer. Hart et al., (2013) reported that perceptual elements of stimuli such as contrast and brightness significantly impacts fixation trends and improves detection.

A notable methodological limitation is from the findings of our follow up experiments in Chapter 5. We intended to employ a low information gain and high information gain version of the initial experiment (Chapter 4 and Chapter 5, Experiment 1). We found that increasing information gain potential (Experiment 3) yielded results that were comparable to Experiment 1, which indicates that information gain in both Experiment 1 and Experiment 3 were equivalent (and both represent high information gain potential set ups). However, due to not eliciting the expected disengagement cost in Experiment 2, it leaves a critical gap in our attempt to test different information gain variants of the gap-overlap paradigm. We cannot conclusively determine whether the lack of repetition effect – that was present in the adolescent sample – was due to high information gain occurring too early or too easily within the paradigm and therefore no change in disengagement cost was seen across repetitions, or whether there is an alternative explanation. Future research must attempt once more to design a gap-overlap paradigm with reduced information gain potential at each presentation of the repeating image. As mentioned above, one method may be by using highly complex or highly informative stimuli which keeps participants engaged in addition to acting as a richer information source, needing more than one trial to extract a high level of information.

An additional methodological consideration for the eye-tracking studies is the difference in trial retention between adolescents and adults in the eye-tracking experiments. While adults maintained fixation well even without a fixation cross, adolescents showed more gaze drift, resulting in more rejected trials. This difference in data quality means that direct comparisons between age groups should be made with caution, as the paradigms were not equally challenging across developmental stages. Future studies should consider including a fixation cross in gap conditions for adolescent participants to improve trial retention.

An important avenue for further research is the finding of sustained theta engagement in ASD which was significant only when participants were median split into high and low scoring groups. Although this makes it a less robust finding, when taken together with previous literature which has found altered theta band activity in ASD (Hornung et al., 2019; Cohen et al., 2008; Wang et al., 2013; Shephard et al., 2017) suggests a compelling line of research for future studies. We mentioned that this pattern of sustained engagement in the

face of a depleting source of information may be linked to prediction error signalling (Gomot & Wicker, 2012; Lawson, Reed, & Friston, 2014). However, the neurophysiological mechanisms underlying this, and the implications of this sustained theta band activity warrant deeper investigation. There have been studies that have reported increased power in the theta band range in resting-state in children and adults with ASD (Lazarev et al., 2015; Mathewson et al., 2012) and has been interpreted as hyper-excitability and immature neural circuitry. Although in our sample we do not report significantly higher theta overall in ASD, it is not unlikely that consistent or sustained theta band activity may draw from the same underlying mechanisms.

Another explanation into atypical theta pattern could be that it reflects altered arousal and preferences in this experimental set up. It is true that when stimuli are repeating, and therefore depleting in information, individuals are likely to disengage and thereby present declining theta power. In ASD however, where preference for repetition is widespread, it is possible that these repeating videos – although declining in information – may still be preferred due to its familiarity and therefore maintain steady state of engagement (Uljarevic et al., 2016). Indeed, research as previously shown that predictable stimuli are inherently rewarding in autism (Clements et al., 2018). Research methods that measure pupil size change overtime can give us better insights into arousal.

### *6.4.1 Barriers in data collection*

The research reported in this thesis was shaped by several significant barriers encountered during data collection. The most substantial challenge was recruiting adolescent participants with clinical diagnoses who had the capacity to engage with the intensive testing protocol. The 2.5-to-3-hour session, which included EEG setup and recording, questionnaire completion, eye-tracking, and the ADOS-2 assessment. While all the participants that we recruited in our sample, were motivated, engaged, and interested throughout the process, it is important to note that these participants were likely a sample of high-functioning adolescents, who already possessed interest in science and psychology. The long testing sessions, in addition to sensory sensitivities that are likely to be triggered by EEG testing (use of gel on the scalp) was likely prohibitive for many families.

We used the ADOS-2 as a test for ASD related traits (or symptoms), and whilst it is a reliable diagnostic instrument, there are challenges associated with administering the ADOS as a single-session assessment. As a semi-structured interview, its accuracy depends heavily

on the participant's state on the day of testing; a young person who is fatigued, anxious, or simply having an off day may not fully engage with the interactive tasks, potentially resulting in scores that do not fully reflect their typical presentation outside the laboratory setting. Situational anxiety or sensory overload from the preceding EEG session could temporarily amplify certain behaviours, leading to scores that overestimate trait severity on that particular day. This state-dependent variability is a well-recognised limitation in autism research (Charman & Gotham, 2013; Lord et al., 2012). In the decision-making process prior to testing, we considered the trade-off between having various tests of individual differences that can offer a more complete and reliable profile of neurodiversity, versus having a singular test that reduces the time spent in the lab and allows participants to express themselves in their own words. The ADOS proved to be an excellent choice which allowed me as the researcher to ask questions directly about their interests, friends, hobbies, and thoughts. While there are limitations associated with the ADOS as a test, from our perspective, it played a significant role in making a majority of participants feel more comfortable, heard, and gave them space to lead conversations.

An important note is also the lack of ADHD-diagnosed participants in our sample. The process of recruitment and data collection was unexpectedly and significantly more time consuming than we anticipated. Despite efforts to recruit participants through multiple channels, the adolescent sample only consisted of one participant with ADHD-only diagnosis.

Participant engagement emerged as a recurring challenge across both adolescent and adult samples. In the EEG paradigm, the Fantasia video clips, while effective in engaging infants (Piccardi et al., 2021), may not have been optimally engaging for adults potentially explaining the absence of the expected linear decline in theta band power across repetitions in the adult sample. Similarly, in the eye-tracking experiments, the images of everyday objects may not have been interesting enough to engage active information gathering, particularly during the lengthy 132-trial sessions. This issue was compounded by the long questionnaires used to measure individual differences. The adult participants completing the AQ, BAPQ, ASRS, and WURS may have experienced boredom or reduced attention, potentially compromising the accuracy of trait measurement. Despite breaks being offered throughout sessions for the adolescent sample and surveys being completed separately prior to the experiment in the adult samples, the length of the protocol inevitably affected participant motivation and engagement. These challenges and barriers represent the trade-offs that are

inherent when conducting large, comprehensive multi-method research with neurodiverse populations.

## 6.5 Conclusions

In conclusion, this thesis provided a multifaceted investigation into the foraging account of attention. We used f high-level cognitive engagement measured by EEG as well as more precise motor act of disengagement measured by eye-tracking. Our findings demonstrate that the core principle of information foraging – that attentional engagement and disengagement should be governed by the information value of a stimulus – is robust in the adolescent population and less clear in the adult population. In both samples however, the way in which individual differences shape this process is not consistent. The proposed account with ASD and ADHD presenting at opposite ends of the spectrum is too simplistic to account for the trends we see in our data. The evidence from our experimental paradigm suggests a unique account of neurodiversity: that ADHD traits appear to have inefficient trade-off between engagement and disengagement, leading to a noisier, less stable attentional states which is consistent with the neural gain model described by Hauser et al., (2016). In contrast, ASD traits appear to alter baseline sustained engagement and the reward that may be associated with information leading to persistence with current stimuli or current location. This work has challenged our proposed model yet lays the groundwork for a more refined framework for understanding attention and information processing in neurodiversity.

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# 8. Appendices

## Appendix A

*A. Questions asked at the end of 6 pre-selected videos and the response expected from the participant*

| **Question** | **Response** |
|---|---|
| The first ostrich in the video starts the dance by going to the LEFT of the screen (from your perspective). | Click the RIGHT button for TRUE and LEFT button for FALSE |
| Was the colour of the curtain on stage pink? | Click the RIGHT button for YES and LEFT button for NO |
| Did the colour of the shoes and headband of the ostriches match? | Click the RIGHT button for YES and LEFT button for NO |
| The bubbles only come out of the trunk of the elephants. | Click the RIGHT button for TRUE and LEFT button for FALSE |
| Were the sequence of scenes in this video altered in any way? | Click the RIGHT button for YES and LEFT button for NO |
| Did all the hippos have 4 teeth each? | Click the RIGHT button for YES and LEFT button for NO |

## Appendix B

*B. Recruitment email and fliers posted on community pages.*

Dear _____

Thank you for reaching out and for your interest in our research! I think your son would be well suited to take part.

We would like to invite you and your child to take part in a series of activities and studies that will take place at the University of East Anglia. The total time commitment for this is expected to be around 3hrs in one visit. You will be with your child all the time, in the same room or in an adjacent room – whichever they prefer.

In our research, we are interested in understanding how your child pays attention to information. We also want to see how this relates to the presence of certain personality traits that are related to Autism and ADHD. Measuring these traits takes around 1 to 1.5hrs with some interactive activities, involving the researcher playing games and having discussions about their interests, and some involving questionnaires that your child will answer. In addition to this, we will also complete two laboratory tasks:

The first one is an Electroencephalography ("EEG") experiment. This involves us placing a cap with sensors onto your child's head, and they will be asked to watch some child friendly cartoons for 20 minutes. Placing the cap requires us to use small amounts of water-based gel on their scalp but it is easily removed with water. In total this task might take up to 1hr.

The second experiment uses eye tracking. This involves your child sitting in front of a screen and attending to images while we measure their eye movements. This part of the study takes 20 minutes.

There will be opportunities for short breaks throughout the experiments. We want you and your child to have an enjoyable and comfortable experience so please let us know if there is anything we can do to meet your child's needs (e.g., to manage sensory sensitivity, or anxiety).

To thank you for your time and effort, you will also receive a £30 shopping voucher along with a picture of your child in the laboratory.

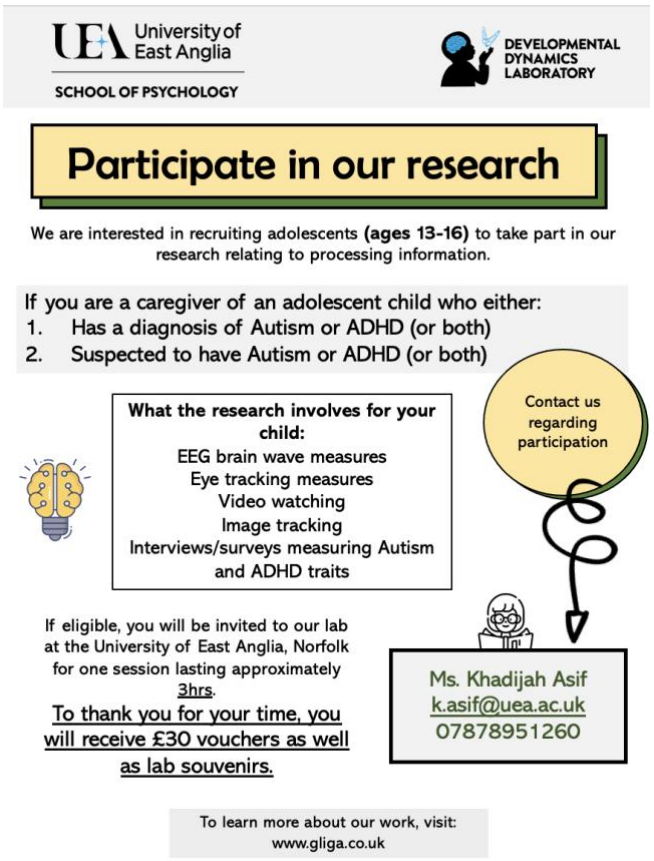
I have attached to this email information sheets containing more details relating to the study. There is a detailed information sheet which is directed to the parent/carer of the child and a child-friendly version which your child may choose to read and sign themselves or have it read to them by someone.

If you are happy to go ahead, then please let us know which time period suits you from below, and then I will follow up with exact timings:

- ➔ During the half term break between February 17th till 24th February (including weekends)
- ➔ Either before or after the half term break in February on a WEEKDAY
- ➔ Either before or after the half term break in February on a WEEKEND

If you have any questions or concerns, do not hesitate to contact me by replying to this email or by giving me a call at 07878951260.

**a**




**b**

# Appendix C

*C.1 Participant information sheet and consent sheet adolescents (parental)*

Miss Khadijah Asif
PhD Candidate

02 August 2023

Faculty of Social Sciences
School of Psychology
University of East Anglia
Norwich Research Park
Norwich NR4 7TJ
United Kingdom

Email: K.asif@uea.ac.uk
Tel: 07878951260
Web: www.uea.ac.uk

## Participant information sheet

Thank you again for your interest in our research. To help you decide whether you and your child would like to take part, we have created this information sheet with all the important information. Please read this sheet carefully and if you have any questions or would like more information, feel free to contact the researcher or let us know in the session at the University.

Keep in mind that you and your child's participation in this study is voluntary and the following information will be explained again in your visit to the lab.

**What is this research about generally?**

The main purpose of our research is to understand the different attention profiles of adolescent children and what these profiles can tell us about how they process things in their daily life.

We do this by:

1. Measuring your child's executive functioning (ie: cognitive flexibility, inhibition, etc)
2. Surveying individual traits that might relate to Autism
3. Surveying individual traits that might relate to ADHD

We also run two experiments to see how children's attention and engagement changes over time:

1. One is an EEG (electroencephalography) experiment where your child will watch videos on the screen
2. The other is an Eye tracking experiment where your child will look towards images that are presented on the screen

**What does this study involve for you and your child?**

Taking part in this study means you will have to visit our lab at the University of East Anglia where this study is taking place. We expect this visit to last no longer than 3hrs. For your child, we will use a survey that measures the presence of Autism traits which involves a series of make-believe play, storytelling, and discussions. In addition, using questionnaires we will also measure the presence of ADHD traits and general executive function ability. This also includes some parent-report questionnaires.

There are two experiments in this study. The first one is an EEG study where we will place a mesh cap with sensors on your child's head. We will use small amounts of water-based gel in the sensors and then they will be asked to watch a selection of child friendly cartoon videos on the screen while we monitor their brain waves. After the EEG experiment is complete, your child may choose to wash the gel out of their hair. In the lab we provide hair washing sink as well as shampoo and towels.

The second experiment is an Eye tracking experiment. Here your child will be asked to sit in front of the computer, resting their chin on a chinrest and look towards images appearing on the screen.

It is important to note that none of these experiments should cause any discomfort at any point. We will offer breaks in between measurements of traits and the experiments, but you or your child can request for additional breaks when needed. You may decide to accompany your child throughout the whole session or for part of the session, and this may be in the same room or in an adjacent room. it is up to you and your child to see what works best. You or your child can stop the study at any time, and for any reason.

Approximate timings:

Autism survey (40-60mins)

ADHD and executive function questionnaires (30mins)

EEG experiment (60mins)

Eye tracking experiment (20mins)

**Do I have to take part? Can I change my mind?**

It is up to you to decide whether you and your child would like to take part. If you agree to let your child take part, we will ask you to sign a consent form when you arrive for your lab visit. We also have an information sheet and consent sheet with child-appropriate language which your child may choose to read and sign, or if you wish, you may read it to them.

You are free to withdraw your child's participation at any time, without giving a reason. This would not affect you nor your child in any way. Likewise, you and your child can stop the study at any time if your child seems uncomfortable, unhappy, or simply does not want to continue. You can also withdraw your child's data from the study up to one month after your participation has ended by contacting the researcher (Khadijah Asif) by phone (07878951260) or email (k.asif@uea.ac.uk), indicating that you would like the data withdrawn. The data will be destroyed and will not be included in the final report.

**What will happen if I agree to take part?**

If after reading this information sheet, you agree to take part in our study, you will be invited to our lab at the University of East Anglia in the LSB (Lawrence Stenhouse Building).

When you arrive at the UEA you will be guided to our lab where we will run most of the session. We will explain again what participation in this study entails and what is expected of the participants. After this we will run study as detailed above. We will also provide refreshments such as tea, coffee, and small snacks. Once this is finished, we will thank you and your child for your participation and answer any additional questions you may have.

**Are there any problems with taking part?**

Our study does not have any risks beyond those typically experienced in everyday activities. It is possible that your child will become bored or tired of the study activities. You may ask for additional breaks or end the study session at any time. You can also choose to skip any questions if you or your child feels uncomfortable with providing an answer.

**Will it help me if I take part?**

Your child will not benefit personally from being in this study. However, we hope that in the future other people might benefit from this study, as this study will help us understand different children use their attention and process information around them.

If you suspect your child to have Autism or ADHD and have not yet received a diagnosis, it may be beneficial to receive insight from the surveys we conduct in the session. Note that we use Autism and ADHD measures for the purpose of research, therefore, a diagnosis would still need to be sought from a GP or a Clinical Psychologist, although we would be happy to provide you with our report on your child's traits relating to Autism and ADHD to aid the process.

**Will it cost me anything to be in this study?**

Free parking in designated bays for lab visitors if you are driving.

**Will I be paid for participating?**

You will receive a £30 gift voucher for your participation in this study, even if you or your child decides to stop participation before the completion of the study. We will also take a polaroid picture of your child while connected to the EEG system as a souvenir for you to keep.

**Who is running this study?**

The study is being carried out by Khadijah Asif (K.asif@uea.ac.uk) and is taking place under the supervision of Dr. Teodora Gliga (T.gliga@uea.ac.uk) and Prof Andrew Bayliss (Andrew.p.bayliss@uea.ac.uk).

**How will you store and use the information that I give you?**

The results from this study are part of my postgraduate research and may be presented in academic journals and at conferences. These results will always be presented as group data and individuals will never be identifiable. Your personal data and information will only be used as outlined in this Participant Information Sheet. Data management will follow the Data Protection Act 2018 (DPA 2018) and UK General Data Protection Regulation (UK GDPR), and the University of East Anglia's Research Data Management Policy.

The information you provide will be stored securely and your identity will be kept strictly confidential, except as required by law. Study data may also be deposited with a repository to allow it to be made available for scholarly and educational purposes. The data will be kept for at least 10 years beyond the last date the data were accessed. The deposited data will not include your name or any identifiable information about you.

**How do I know that this research is safe for me to take part in?**

All research in the University is looked at by an independent group of people called a Research Ethics Committee, to protect your safety, rights, wellbeing, and dignity. This research was approved by the Psychology Research Ethics Committee at the University of East Anglia on 07/09/2023.

**Will I be told the results of the study?**

You are able to receive the scores on assessments of individual traits for your child if you choose. As for the experiments, these are only analysed at group level so individual results would not be given. If you would like to read the overall findings of this study, they may be included in the monthly newsletter that we send out through the Developmental Lab at UEA. If you are interested in signing up for this, please notify me by email (k.asif@uea.ac.uk) at any time or let me know in the session.

ETH2223-2762

**What if I have concerns or complaints about the study?**

You can contact me via the University of East Anglia at the following address:

Miss Khadijah Asif

School of Psychology

University of East Anglia

NORWICH NR4 7TJ

k.asif@uea.ac.uk

07878951260

If you are concerned about the way this study is being conducted or you wish to make a complaint to someone independent from the study, please contact the Head of School of Psychology: Prof. Neil Cooper; Neil.cooper@uea.ac.uk; 01603592996

**Duty of care**

We will disclose to the proper authority, information you share with us concerning child abuse, child sexual abuse, or harming yourself or others.

**You are under no obligation to agree** to take part in this research.

If you do agree you can **withdraw at any time without giving a reason**

**Contact details:**

*If you have any questions or concerns about this research you can contact:*

Miss Khadijah Asif (k.asif@uea.ac.uk; 07878951260),

Dr. Teodora Gliga (T.gliga@uea.ac.uk) and

Prof Andrew Bayliss (Andrew.p.bayliss@uea.ac.uk).

**School of Psychology Ethics Committee**: ethics.psychology@uea.ac.uk; 01603 59714

**Head of School Professor Neil Cooper**: N.Cooper@uea.ac.uk; 01603 597145

**This information sheet is for you to keep**

ETH2223-2762

**PARTICIPANT CONSENT FORM (First Copy to Researcher)**

I, ..... [PRINT NAME], **am** willing to participate in this research study.

In giving my consent I state that:

- I understand the purpose of the study, what I will be asked to do, and any risks/benefits involved.
- I have read the Participant Information Sheet, which I may keep, for my records, and have been able to discuss my involvement in the study with the researchers if I wished to do so.
- The researchers have answered any questions that I had about the study and I am happy with the answers.
- I understand that being in this study is completely voluntary and I do not have to take part. My decision whether to be in the study will not affect my relationship with the researchers or anyone else at the University of East Anglia now or in the future.
- I understand that I may stop the research activity at any time if I do not wish to continue, and that unless I indicate otherwise any materials (e.g. images, recordings, text) will then be erased and the information provided will not be included in the study results. I also understand that I may refuse to take part in any of the activities that I wish.
- I understand that the results of this study may be published but that any publications will not contain my name or any identifiable information about me.
- I understand that personal information about me that is collected over the course of this project will be stored securely and will only be used for purposes that I have agreed to. I understand that information about me will only be told to others with my permission, except as required by law.

I consent to:

My child completing the traits assessments (for Autism/ADHD) YES ☐ NO ☐

My child taking part in EEG and Eye tracking experiment YES ☐ NO ☐

.....
**Signature**

.....
**PRINT name**

.....
**Date**

ETH2223-2762

*C.2 Participant information sheet and consent sheet adolescent (participant)*

University of East Anglia
Norwich Research Park
Norwich. NR4 7TJ

Email: k.asif@uea.ac.uk
Tel: 07878951260
Web: www.uea.ac.uk

*09/10/2023*

## Study Information Sheet

**Hello. My name is Khadijah, and I am a researcher at the University of East Anglia.**

**I am doing a study to learn more about how people in your age group have different attention styles.**

I am asking you to be in my study because I would like to learn more about how your brain works while you watch videos and images. I would also like to take part in some activities with you and ask you questions that can tell me more about your daily life and the things you like doing.

You can decide if you want to take part in the study or not, it's up to you.

This sheet tells you what I will ask you to do if you decide to take part in this study. Please read it carefully.

If you decide you want to be in the study and then you change your mind later, that's ok. All you need to do is tell me that you don't want to be in the study anymore. We will remove any information you may have already given us. You or someone who looks after you can email me before or after the study if you don't want to be in the study anymore (k.asif@uea.ac.uk).

If you have any questions; you or someone who looks after you can contact me on my email: k.asif@uea.ac.uk. Or if you want, you can speak to me during your visit to the university.

**What will happen if I say that I want to be in the study?**
If you would like to take part in this study, you, and your parent (or anyone who looks after you) will visit our lab at the University of East Anglia. When you arrive, we will take part in some activities together.
The activities are as follows:

- We will ask you to answer some questions on a computer or tablet. You can choose to do this by yourself or ask your parent or the researcher to help you fill it in.
- In another activity, we will speak about thing like your friends and what you want to be when you grow up. We will also play games like puzzles, make a story with some items, and read some pages of a picturebook together.
- To look at your brain waves, we will also place some sensors on your head and then ask you to watch some cartoon videos on the screen.
- Another activity we have, is for us to see how your eyes move between different images. Here you will be sitting in front of a screen and just watch the images that come up.

In these activities I will ask you questions, but you can choose which ones you want to answer, and if you don't want to talk about something, we don't have to talk about it, that's ok. If there is an activity that you do not like or do not want to take part in, you can just let me know and we don't have to do that activity. You can also change your mind even after the activity is started, if you don't enjoy it, we can stop and that's ok as well.

I will make sure you do not get too tired or bored, so we will have breaks in between the activities, and I will also try to bring some snacks! If you feel tired at any time in middle of the activities, we can also take a break then.

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Don't worry if you forget any of this, I will explain all this again on the day you visit us and you can ask me any questions you have.

**Will anyone else know what I say in the study?**

I won't tell anyone else what you say to me, except if you talk about someone hurting you or about you hurting yourself or someone else. Then I might need to tell someone outside of the study to keep you and other people safe.

All the information that I have about you from the study will be put in a safe place and I will look after it very carefully. I will write a report about the study and show it to other people, but I won't put your name in the report, and no one will know that you're in the study. I may also share your information with other researchers and use it for teaching and learning purposes, but I will always take out your name.

**How long will the study take?**

In total your whole time with us at the university should not be longer than **3 hours**. This time will be split between all the different activities you will take part in. Some will be short such as answering questions on the computer, and some will be a little longer such as the brain waves measure because it needs extra time to set up.

**Are there any good things about being in the study?**

There might not be any big helpful change in your daily life if you choose to take part. But the information you give us, will help us understand the different attention styles of people your age. Even though there is not going to be a big difference to your daily life, we hope that you will have a good time in your visit and that you will enjoy learning about some of the things we research about at the university. We will also give you and your parent a £30 voucher to thank you for your visit!

**Are there any bad things about being in the study?**

Taking part in this study will take up some of your free time, but there should be no bad effect on you if you choose to take part. It is possible you might get bored, but we will try our best to keep it fun and interesting for you!

**Will you tell me what you learned in the study at the end?**

Yes, I will if you want me to. There is a question on the next page that asks you if you want me to tell you what I learned in the study. If you circle Yes, when I finish the study, I will tell you what I learned.

**What if I am not happy with the study or the people doing the study?**

If you are not happy with how I am doing the study or how I treat you, then you or someone who looks after you can:

- Tell me during the study.
- Email me (k.asif@uea.ac.uk; 07878951260)
- Contact my supervisors (Dr. Teodora Gliga T.gliga@uea.ac.uk and Prof Andrew Bayliss Andrew.p.bayliss@uea.ac.uk)
- Email my Head of School (N.Cooper@uea.ac.uk; 01603 597145)

**How do I know that this study is ok to take part in?**

All research I undertake is checked and approved by an Ethics Committee at the University of East Anglia before I can start it.

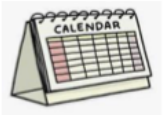
**What if I want to know more about the information collected on me in the study?**

When we talk in the study, I will collect some information which is unique to you. I can only collect this information if I have a reason to do so. My reason to do so for this study is because the study is helpful to understand children's brains and thinking.

This information is stored by me within the University of East Anglia. They help me protect your information and look after it.

If you want to know more about the information collected about you, you can email me (k.asof@uea.ac.uk) or you can email the University's Data Protection Officer (dataprotection@uea.ac.uk) who helps to protect your information. The Information Commissioner's Office (ICO) helps to protect everyone's information. If you are unhappy with mine or the University Data Protection Officer's responses about your information, you can speak to the ICO.

**Further information**




This sheet was last updated on 09/10/2023 I will update you if I make any changes to this sheet.

*This information sheet is for you to keep.*

**Participant Consent Form (*First Copy to Researcher*)**

If you are happy to be in the study, please:

- **write** your **name** in the space below.
- **sign** your **name** at the bottom of the next page.
- put the **date** at the bottom of the next page.

You should only say 'yes' to being in the study if you know what it is about and you want to be in it. If you don't want to be in the study, don't sign the form.

I, ..... [PRINT NAME], am happy to be in this research study.

In saying yes to being in the study, I am saying that:

- ✓ I know what the study is about.
- ✓ I know what I will be asked to do.
- ✓ Someone has talked to me about the study.
- ✓ My questions have been answered.
- ✓ I know that I don't have to be in the study if I don't want to.
- ✓ I know that I can pull out of the study at any time if I don't want to do it anymore and it will possible to remove any information I have already provided (if contacted within 1 month)
- ✓ I know that I don't have to answer any questions that I don't want to answer.
- ✓ I know that the researchers won't tell anyone what I say when I talk to them unless I talk about being hurt by someone or hurting myself or someone else.

**Now I am going to ask you circle 'Yes' or 'No' to tell me what you are happy to do or not do in the study.**

Are you happy to **speak just to me**?
**Yes** **No**

Are you happy to answer some **questions about daily life** on a computer?
**Yes** **No**

Are you happy for me to measure your **brain waves while you watch cartoons**?
**Yes** **No**

Are you happy for me to **measure your eye movements while you watch images**?
**Yes** **No**

Do you want me to tell you what I **learned** in the study?
**Yes** **No**

..... .....
**Signature** **Date**

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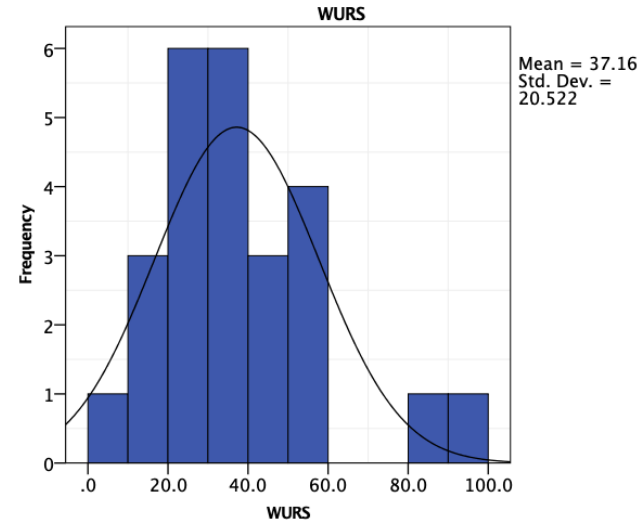
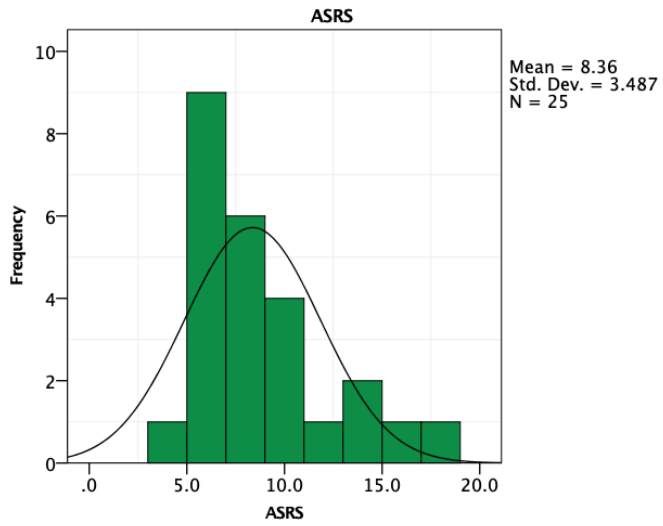
## Appendix D

*D. Graphs showing frequency plots for survey scores in WURS, ASRS, AQ, and BAPQ*




(a)

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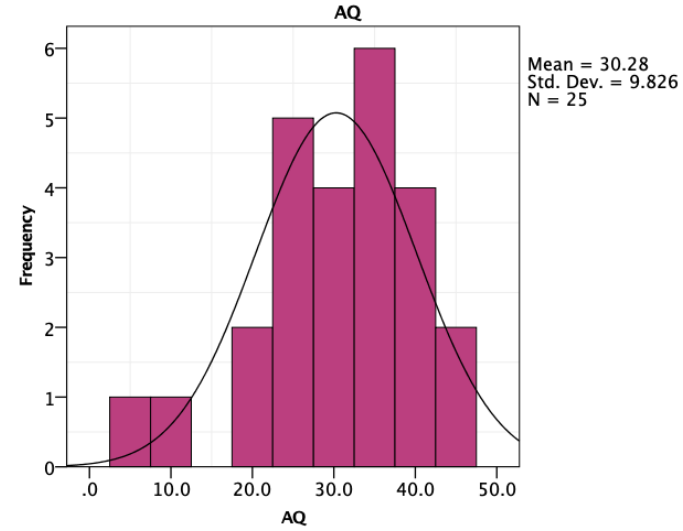
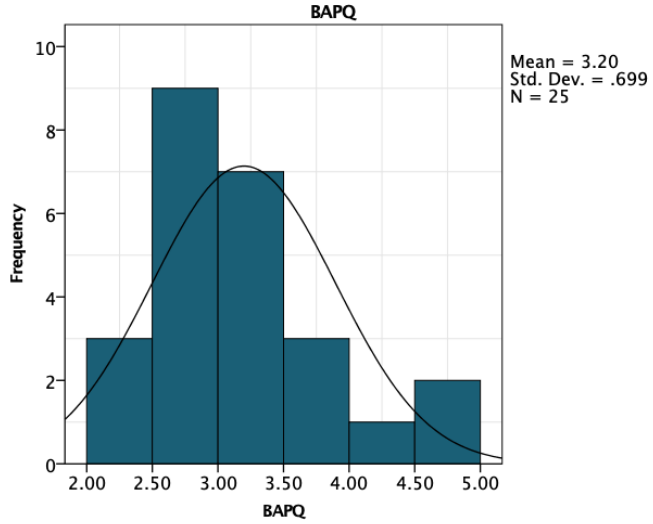



(c)




(d)

## Appendix E

E. Table explaining differences between different versions of gap overlap paradigm.

| Version | Task | Purpose | Gain |
|---|---|---|---|
| Typical | Images are presented for 1000ms throughout the experiment.<br>*In Gap condition:*<br>- Central image: 1000ms<br>- Gap time: 300ms<br>- Peripheral image: 1000ms<br>*In the peripheral condition*<br>- Central image: 1300ms (300ms of this is overlapped with presentation of peripheral image)<br>- Peripheral image: 1000ms<br>Repetitions in the central image position are randomized however, a repetition does not occur more than 7 trials followed a previous presentation of the same image. | Standard version. Images are attended to by the participant for 1000ms. | Medium |
| Blocked design | Images are presented for 1000ms throughout the experiment.<br><br>Repetitions in the central image position are ***not*** randomized. A repetition of an image immediately follows the presentation of the previous image. Ie: images that repeat twice are repeated in two trials following each other, and images that repeat four times, are repeated in four trials following each other. | More information gain. Without changing the length of time the images are presented to the participant (elongating the entire experiment and boring the participant), the trials are blocked. This allows processing of the repeated images closer to each other temporally, without processing of different images between trials of repeated images. | High |
| Short time | Images are presented for 500ms throughout the experiment. The design of this version is identical to the *typical* design, the difference between these two versions is the timings.<br><br>*In Gap condition:*<br>- Central image: 500ms<br>- Gap time: 200ms<br>- Peripheral image: 500ms<br>*In the peripheral condition* | Less information gain. Without changing the order of presentations (creating bigger gaps between repeated trials, allowing participants to forget some stimuli), the time participants attend to the image is reduced. Presentation of images are reduced by 50% and overlap time is reduced | Low |

| | | | |
|---|---|---|---|
| | - Central image: 700ms (200ms of this is overlapped with presentation of peripheral image)<br>- Peripheral image: 300ms<br><br>Repetitions in the central image position are randomized however, a repetition does not occur more than 7 trials followed a previous presentation of the same image. | by 30%: allowing sufficient time for both images to cause competition when presented. | |