
Usage Guidelines:
Please refer to usage guidelines at lib-eprints@bbk.ac.uk.

or alternatively contact lib-eprints@bbk.ac.uk.
Early visual foraging in relationship to familial risk for autism and hyperactivity/inattention: A preliminary study

Teodora Gliga1*
Tim Smith1
Noreen Gilhooly1
Tony Charman2
Mark H. Johnson1

1 Centre for Brain and Cognitive Development, Birkbeck College, University of London, UK
2 Institute of Psychiatry, Psychology and Neurosciences, King's College London, UK

*corresponding author: t.gliga@bbk.ac.uk

Author contributions: T.G. designed the eye-tracking study, T.G., T.S. and N.G. analyzed the data; T.G. and T.S. wrote the paper with contribution from M.H.J. and T.C. M.H.J. and T.C. lead the BASIS programme.

Acknowledgments: This work was supported by MRC Programme Grant G0701484 and the BASIS funding consortium led by Autistica (www.basisnetwork.org).
Abstract

Information foraging is atypical in both Autism Spectrum Disorders (ASD) and attention deficit hyperactivity disorder (ADHD), however while ASD is associated with restricted exploration and preference for sameness, ADHD is characterized by hyperactivity and increased novelty seeking. Here we ask whether similar biases are present in visual foraging in younger siblings of children with a diagnosis of ASD with or without additional high levels of hyperactivity and inattention. Fifty-four low-risk controls (LR) and 50 high-risk siblings (HR) took part in an eye-tracking study at 8 and 14 months and at 3 years of age. At 8 months, siblings of children with ASD and low levels of hyperactivity/inattention (HR/ASD-HI) were more likely to return to previously visited areas in the visual scene than were LR and siblings of children with ASD and high levels of hyperactivity/inattention (HR/ASD+HI). Thus, we reveal a paradoxical effect, in which additional family risk for ADHD core-symptoms mitigates the effect of ASD risk on visual information foraging.

Keywords: visual foraging, infants, ASD, hyperactivity/inattention, eye-tracking
Many human activities rely on information foraging, i.e., the seeking out and processing of information. Theoretical accounts of information foraging draw heavily on general models for resource foraging, which describe a trade-off between effort or energy expenditure and the amount of resources the organism manages to secure. Optimal foraging requires shifting between exploitation, i.e., taking advantage of the resource at hand, and exploration of the environment to discover new resources (Cohen, McClure & Yu, 2007). There is no absolute best combination of exploration and exploitation, but rather organisms gain from flexibly adapting foraging to environmental conditions (i.e., it is better to exploit the resources at hand if the further environment is uncertain, and better to explore when faced with competition (Kacelnik, Houston & Krebs, 1981). Any strong inherent bias, either towards exploratory behavior or for resource exploitation, might therefore interfere with optimal foraging. This paper deals with the development of such potential biases in foraging for information in infants at familial risk for ASD or for attentional problems.

*Atypical foraging in ASD and ADHD.* Individual differences in foraging behavior have been related to particular neural systems. For example, biases for exploratory behavior were associated with differences in dopaminergic and noradrenergic function (Frank et al, 2009; Jepma & Niewenhuis, 2011). Similarly, stimulating dopaminergic function in monkeys led to an increase in preference for novel stimuli over old stimuli with larger reward value (Costa et al., 2014). Explorative choices, i.e., choosing a new over an old item, were shown to be preceded by increases in pupil diameter, which index locus coeruleus/noradrenergic function (Jepma & Niewenhuis, 2011). Both Autism Spectrum Disorders (ASD) and Attention
Deficit Hyperactivity Disorder (ADHD) have been associated with atypicality in dopamine (e.g., Solanto, 2002; Kriete & Noelle, 2015) and noradrenaline functions (Biederman & Spencer, 1999; Blaser et al., 2014) as well as, separately, with atypical information foraging. In ASD, decreased exploratory behavior was documented in a task in which children had to discover objects hidden in different containers. Children with ASD spent less time in active exploration and explored fewer containers with these behaviors related to anatomical differences in the cerebellum, such as differences in the size of the vermal lobules (Pierce & Courchesne, 2001). In another similar foraging study, Pellicano et al. (2011) reported less systematic searches and a higher proportion of revisitations in children with ASD. Differences were also found in free-viewing visual scanning (Elison et al., 2012). Typically developing (TD) and ASD participants aged 2 to 18 years were presented with large arrays of images. The number of images visited per unit of time increased with age in both groups, but with a steeper slope in TD participants (Elison et al., 2012). Thus, across measures, ASD seems to be associated with less exploratory behavior. When findings could not be explained by specific preferences related to the disorder, as is the case in both Pellicano et al., (2011) and Pierce & Courchesne, (2001), returning to already explored locations/objects could be driven by incomplete information processing or poor memory. However, IQ was not a predictor of performance in either Elisson et al., (2012) or in Pierce & Courchesne, (2001) and groups were matched in IQ in Pellicano et al., (2011). Thus these biases seem to manifest independently of concurrent information processing difficulties.

In contrast to ASD, ADHD is associated with extreme novelty seeking, especially in the case of the hyperactive and combined (hyperactive and inattentive) subtypes (Salgado et al, 2008). Intriguing evidence that genes associated with ADHD
(e.g., particular DRD4 gene variants) are more frequent in populations that have a history of migration (Chen et al., 1999; Mathews & Butler, 2011), led to the suggestion that hyperactivity might be an adaptation to the food-scarce and volatile environment our ancestors lived in (Jensen et al., 1997). Some rather indirect evidence for a bias towards exploratory behavior in ADHD comes from a study in which the number of regions visited when free-viewing a visual scene correlated with differences in curiosity (Risko et al., 2012), which some have associated with ADHD (Williams & Taylor, 2006).

Thus, the existing literature suggests ASD and ADHD might be associated with opposing biases in foraging. However, since ASD and ADHD often co-occur (approximately 20% of UK 7-year-olds with ASD meet criteria for ADHD, and vice versa, Russell et al., 2014), this raises the question of how ASD and ADHD-specific foraging biases interact during development. Additive phenotypic effects were previously described in children with co-morbid ASD and ADHD, for example neural processing of human gaze in these children was similar to both profiles of children with ASD only or with ADHD only (Tye et al., 2013). An increase in symptom severity, compared to the single diagnosis cases, was also documented (e.g., Goldin et al., 2013; Craig et al., 2015). These findings raise the intriguing possibility that where disease-specific phenotypes are at opposite ends of a spectrum (e.g., increased exploration in ADHD and decreased exploration in ASD), risk for one disorder may mitigate the effects of risk for the other disorder.

We were therefore interested in investigating the impact ASD and ADHD risk has on information foraging during development. Both occulo-motor behaviors and object manipulation have been used to measure information foraging in infants.
(Bornstein, Hahn & Suwalsky, 2013). Of the two measures, visual scanning is less confounded by general motor development.

**Visual foraging and its development.** It has been suggested that the type of cognitive processes that derived from spatial foraging for resources such as food, may also be associated with the control of visual attention (Hills, Todd & Goldstone, 2008). For example, when freely exploring new visual scenes, participants make shorter fixations and longer saccades during the first few seconds, covering the whole visual scene. However, as participants continue to look, their fixations gradually become longer and their saccades shorter (Pannasch et al., 2008; Fischer et al, 2013). This suggests that the optimum visual information strategy relies on an initial exploratory phase, where a new scene/environment is mapped to discover potential interesting sights. This is then followed by information exploitation, where chosen locations (sources of information) are repeatedly investigated (Krebs, Kacelnik & Taylor, 1978). Failure to explore the scene might lead to missing out on important information while prolonged exploration might prevent learning. There is still limited understanding of developmental changes in visual foraging, but the limited existing evidence suggests an increase in exploration with age. During the first month after birth, infants restrict their scanning to a small portion of an image, but by 3 months they produce longer saccades and gaze patterns that are more systematically distributed over visual scenes (Bronson, 1991). However, even beyond 6 months of age, scanning patterns remain restricted to particular locations of the visual scene and are therefore highly predictable (Schesinger & Amso, 2013). In the current study we will ask whether measures of visual scanning capture information foraging atypicalities early in development.
The current study. The current work is a re-analysis of a previously published dataset (Elsabbagh et al., 2013). The “face-pop out” paradigm was designed to measure face orienting as an early marker for ASD. Infants freely explored displays containing a face and 4 other objects whilst their eye-movements were measured with an eye tracker (Fig 1). Because infants had 15 seconds to explore each new display, by analyzing the sequences of visits to faces and other objects/areas of interest (AOIs), this paradigm can inform about visual foraging strategies.

Study participants were infants with an older sibling with ASD, who were therefore themselves at-risk for ASD. Approximately 20 % of children at-risk will receive a diagnosis of ASD themselves (Ozonoff et al, 2011), and another 30% will manifest elevated levels of ASD symptoms (Messinger et al., 2013). Thus, by comparing visual foraging in this population to a low-risk cohort, we aim to capture differences due to genetic susceptibility for ASD. As mentioned earlier, ASD and ADHD are often co-occurring and, like ASD, ADHD has moderate heritability (Larsson et al., 2014). To investigate the effect ADHD risk has on visual foraging, we characterized the older siblings’ hyperactivity and inattention profile using the Strengths and Difficulties Questionnaire (SDQ). The SDQ has been extensively used as a screener for ADHD and has good sensitivity, especially for children with the combined subtype (hyperactivity/inattention) (Ullebo et al., 2011; Carballo et al., 2014). To reflect the fact that the SDQ is a screening, not a diagnostic, instrument we refer to the risk conferred by a high score on the SDQ as hyperactivity/inattention risk (HI risk). We can thus compare the effect of ASD risk, and that of additional HI risk, on visual foraging during early development. Infants contributed data at three age points (8 months, 14 months and 3 years). We analyzed the temporal dynamics of
attention as participants scanned the different areas of interest (AOIs), focusing in particular on the likelihood of images being revisited. We expected ASD risk to be associated with decreased exploration (i.e., higher likelihood of revisiting AOIs) and that additional HI risk may moderate this effect.

Methods

Participants

Fifty four infants at high familial risk (HR) and 50 infants at low familial risk (LR) for ASD took part in a longitudinal study. Infants attended lab-based testing 3 times, first between 6 and 10 months, a second time around 14 months and a third time around 3 years of age. Only a sub-set of participants contributed data at the 3-year visit. At the time of enrolment, none of the infants had been diagnosed with any medical or developmental condition. HR infants all had an older sibling (proband) with a community clinical diagnosis of ASD. Proband diagnosis was confirmed by two expert clinicians (xx, xx) based on information from the Development and Wellbeing Assessment (DAWBA) and the parent-report Social Communication Questionnaire (SCQ). Infants in the low-risk group were recruited from a volunteer database at the xxx. All low-risk infants had at least one older sibling with typical development and no first-degree relatives with ASD. None of the older siblings scored above instrument cut-off for ASD on the SCQ ($\geq$15, 1 score missing).

Assessing Hyperactivity/Inattention-risk

Proband ADHD risk was attributed based on scores on the Strengths and Difficulties Questionnaire (SDQ), filled in by the parent of HR participants. The SDQ is composed of 25 items that ask about behavioral attributes of the child and are
combined to form five subscales. The hyperactivity/inattention subscale covers restlessness, fidgeting, concentration, distractibility and impulsivity. Each item can be answered with: ‘Not true’ ‘Somewhat true’ or ‘Certainly true’, and they are scored 0, 1 or 2 respectively, giving a total score out of a possible 10 for each subscale. Screening studies suggest a cut-off of 8/10 as providing the best ADHD diagnostic accuracy (Carballo et al., 2014). Using a cut-off of 8, we separated the HR ASD group into a HR/ASD-HI group (n=26), including children whose probands scored below 8 (average=5.19; SD=1.7) and a HR/ASD+HI group (n=20), when probands scored 8 or above (average=9.25; SD=0.78). Hyperactivity and Inattention subscale scores did not relate to proband social communication abilities (Social Communication Questionnaire, Rutter & Bailey, 2003; r = .246, p > .1).

Visual scanning task and procedure

The same stimuli and procedure as previously described in Elsabbagh et al, (2013) was used. At 8 and 14 months, infants saw 14 different slides (example in Figure 1), each for 15 seconds. At 3 years, only 10 slides were shown. Each slide contained 5 images, one from each of the following categories: faces, mobile phones, birds, cars and scrambled faces. A central attention getter was presented before each slide/trial to re-orient infant’s attention. Infants were seated on their parent’s lap in front of the eye-tracking monitor (at 8 and 14 months) or on their own in front of the eye-tracking monitor (3 years of age). Parents were asked to refrain from pointing to the screen or naming any of the images.

Revisitation analysis. Gaze data was recorded with a Tobii 1750 eye-tracker at 50 Hz. Data was parsed into fixations defined as ‘gaze remaining within a 30 pixel radius (∼.80° of visual angle) for a minimum of 60 ms’. A list of fixations, in the
order in which they occurred and their corresponding AOI, was extracted for each participant and used to compute visit durations and order during each trial. A *visit* was defined as the sum of all consecutive fixations made within an AOI. Each visit was coded as either a first visit (the first visit to that AOI, coded as 0) or a revisit (the AOI had been visited before within the trial, coded as 1). The probability of choosing an old item was always zero at the first visit, since none of the other items had been fixated at this point within the trial. Likewise, the probability of choosing an old item at the second visit was always zero, because all of the possible target AOIs were new items; the only old item was the AOI the eyes had just left. We coded up to 10 visits per trial. For each participant, we calculated the *revisitation likelihood* for each visit, from the 1st to the 10th, by averaging across all trials.

*Younger sibling outcome characterization*

A standard measure of mental development level, the Mullen Scales for Early Learning (MSEL; Mullen, 1995), was collected. The MSEL is a standardized direct developmental assessment that yields a standardized score (mean = 100, SD = 15) of overall intellectual ability (Early Learning Composite standard score). The HR group was also assessed with the parent-report Autism Diagnostic Interview – Revised (ADI-R; Lord et al., 1994) and the Autism Diagnosis Observation Schedule (ADOS, Lord et al, 2000). A ‘best estimate clinical consensus’ approach to diagnosis was taken following a review by experienced clinical researchers (xxx) taking account of all information about the child (i.e. MSEL, informal observation), in addition to information from the ADI-R and ADOS-G. Children were included in the ASD group if they met ICD-10 (World Health Organisation, 1993) criteria for any pervasive developmental disorder (PDD). Given the young age of the children, and in line with
the changes to DSM-5 (American Psychiatric Association, 2013), no attempt was made to assign specific sub-categories of PDD/childhood autism diagnosis. Children from the HR group were considered typically developing (high-risk Typical) if they (i) did not meet ICD-10 criteria for an ASD; (ii) did not score above the ASD cut-off on the ADOS or ADI; (iii) scored within 1.5 SD of the population mean on the Mullen Early Learning Composite (ELC) score (>77.5) and Receptive Language (RL) and Expressive Language (EL) subscale T scores (>35). Children from the HR group were considered to have atypical development if they did not fall into any of the above groups. That is, they either scored above the ADOS or ADI cut-off for ASD or scored <1.5SD on the Mullen ELC or RL and EL, but did not meet ICD-10 criteria for an ASD. From the 47 At-risk participants taking part in this task, 17 met criteria for an ASD diagnosis, 18 were At-risk Typical and 12 were in the HR Atypical group (9 scoring above the ADOS ASD cut-off, 1 scoring above the ADOS ASD cut-off and <1.5SD Mullen ELC cut-off, 1 scoring above the ADI ASD cut-off and 1 scoring <1.5SD Mullen ELC cut-off).

**Results**

*Preliminary analyses.* We computed the average number of AOI visits per trial for each age group. On average, participants contributed 7 visits (min average 3, max average 9.9) at 8 months, 8 visits at 14 months (min 2.42, max 10) and 9 visits at 3 years (min 6.8 and max 10). Thus, to avoid data loss, we restricted the analysis to the first three visits, which meant restricting the analysis of revisitation likelihood to visit 3 (since the likelihood of revisitation is zero at the first and second visit). Given that at the 3rd visit all previous visits had been on new AOIs, this measure will be unaffected by visit history (and by potential group differences in visit history).
Because at both 8 months and 3 years, the HR/ASD-HI group had the lowest scores on the Mullen Scales of Early Learning of the 3 groups, (Table 1), we investigated the relationships between revisitation likelihood and concurrent Mullen scores. None of these relationships reached significance (see SOM).

**Does ASD and HI risk influence visual foraging?**

We carried out a repeated measures ANOVA on likelihood of revisitation at the 3rd visit, with age (8, 14 months and 3-years) and group (24 LR, 20 HR/ASD-HI, 12 HR/ASD+HI) as within participant measures. This yielded a main effect of age (F(2,106) = 17.97, p < .001), as well as a significant effect of group (F(2,53) = 3.81, p = .028). With age, all groups became more exploratory, in the sense that they were increasingly more likely to visit a new AOI rather than returning to a previously seen one. Bonferroni-corrected post-hoc t-tests revealed that LR participants were less likely to re-visit than the HR/ASD-HI group (p = .051), whilst the HR/ASD+HI group was not significantly different from either the HR/ASD- HI participants (p = .091) or the LR participants. We followed-up on a significant age x group interaction (F(4,106) = 3.40, p = .012) with 3 repeated measures ANOVAs for each age (Figure 2). At 8 months the effect of group was significant (F(2, 95) = 7.39, p = .001; HR/ASD-HI were significantly different from both LR (p = .009) and HR/ASD+HI (p = .001), the last two being indistinguishable statistically). Likelihood of re-visitation was above chance level (.25) in all groups (LR: t(47) = 7.07, p < .001; HR/ASD-HI: t(25) = 10.54, p < .001; HR/ASD+HI: t(17) = 3.17, p = .006)

At 14 months, the effect of group was again significant, F(2,86) = 4.65, p = .012; only LR and HR/ASD-HI were significantly different from each other (p = .012). Likelihood of re-visitation was above chance level in all groups (LR: t(43) =
3.17, p = .003; HR/ASD-HI: t(23) = 4.53, p < .001; HR/ASD+HI: t(17) = 5.41, p < .001). Groups did not significantly differ at 3 years and at all ages likelihood of revisititation was at chance (LR: t(25) < 1; HR/ASD-HI: t(19) < 1; HR/ASD+HI: t(13) < 1). Thus, by 3 years of age, when encountering a new visual scene exploratory biases are apparent (i.e., the likelihood of return falling below chance level).

Given that a dimensional measure of HI risk was available (the SDQ scores vary between 0 and 10), and to strengthen our findings, we investigated the quantitative relationship between revisititation likelihood at 8 and 14 months and this measure. This analysis yielded a significant negative correlation between revisititation likelihood and SDQ scores at 8 months (r = -.430, p = .003; Figure 2), but not at 14 months.

Were group differences driven by returns to the face? Since many revisits at the 3rd visit are returns to the face (about 50% of first looks are to the face, Elsabbagh et al., 2013), it is important to know whether the group differences described mainly reflect differences in returns to this stimulus. To ask this question we ran a repeated measures ANOVA with type (Face, non-Face) and group (LR, HR/ASD-HI, HR/ASD+HI), at each age point. At both 8 and 14 months this analysis yielded significant main effects of type (8 months: F(1,88) = 42.35, p < .001; 14 months: F(1,83) = 23.14, p < .001) and group (8 months: F(2,88) = 3.64, p = .030; 14 months: F(2,83) = 3.47, p = .036), but a non-significant interaction of type x group (8 months: F(2,88) = 2.06, p > .1; 14 months: F(2,83) < 1), which suggests that group differences in revisititation likelihood concern both types of images. At 3 years, the effects of type and group were not significant (type: F(1,55) = 2.84, p > .05; group: F(2,55) < 1), but there was a marginally significant interaction between type and group (F(2,55) = 2.99, p = .058). However, follow-up paired t tests yielded no significant effects of type.
Is foraging related to the child’s ASD diagnosis?

Since ASD diagnosis was available for the younger sibling, we also ran a repeated-measures ANOVA on the revisit likelihood, with age as a within participant variable and outcome (LR, HR-TD, HR-Atypical and HR-ASD) as between-participants factor. This yielded a significant interaction between age and outcome ($F(6,108) = 2.43, p = .030$) and a non-significant effect of outcome ($F(3,54) < 1$). We followed up this analysis with 3 univariate ANOVAs for each age point. Although HR-ASD did have the highest likelihood of revisititation at 8 months, outcome was not a significant predictor ($F(3,102) = 2.23, p = .089$ (see Figure S1). Follow up paired t-tests against the LR group (Dunnett t, Dunnett 1955), yielded marginally significant effects (LR vs. HR-ASD, $p = .084$). At 14 months there was a significant effect of outcome ($F(3,92) = 2.78, p = .045$), but none of the follow-up paired t-tests were significant. The effect of outcome was not significant at 3 years.

To explore whether proband HI affected all outcome groups, at 8 months a second repeated measures ANOVA was run on the high-risk group only, this time with both outcome (HR-TD, HR-Atypical and HR-ASD) and HI level (low, high) as between participants variables. This yielded a significant effect of HI level ($F(1,43) = 10.51, p = .002$), which did not interact with outcome ($F(2,43) < 1$; see figure S2). Thus, high proband HI increased exploration in those siblings that developed ASD as well as in the typically developing children.

Discussion

Borrowing the foraging framework developed for appetitive decision-making
in animals (Cohen, McClure & Yu, 2007; Kacelnik, Huston & Krebs, 1981), we investigated the development of exploratory biases in visual information foraging. We observed age related changes. Six months olds showed a strong tendency to revisit previously seen AOIs, with 40% of their 3rd visits being revisits. In contrast, by 3 years of age, when faced with a new visual scene, children randomly choose whether to explore a new AOI or revisit. Thus, although we observe a decrease with age in exploitative choices (i.e., preferring to return to old AOIs), not even at 3 years of age does foraging become driven by exploration (i.e., consistently preferring to sample new AOIs). The main question that motivated this research was whether background family risk for ASD and hyperactivity and inattention (core symptoms of ADHD), impact on visual foraging early in life. In terms of the likelihoods of revisitation, the HR/ASD-HI group was more likely to return to old locations than were LR participants, at both 8 and 14 months. When the sibling’s own ASD diagnosis was taken into account, a trend was found for higher revisitation likelihood in 8-month-old HR-ASD, compared to LR controls. This is reminiscent of previous findings in populations with a diagnosis of ASD that also show high return rates when exploring visual scenes or real environments (Pierce & Courchesne, 2001; Pallicano et al., 2011, Elison et al., 2012).

We also predicted that proband hyperactivity/inattention would be associated with a bias towards exploration. Indeed, at 8 months likelihood to re-visit AOIs inversely relates to proband SDQ hyperactivity and inattention scores. However, when groups are compared, HR/ASD+HI participant performance was similar to that of the control group at 8 months and was not different from either the HR/ASD-HI or LR at 14 months. Our findings are compatible with an additive effect, where ASD and ADHD risk contribute opposing biases. Additive effects of dual ASD and ADHD
diagnosis have been previously documented (e.g., Tye et al, 2013). More often then not, the literature reports increased symptom severity in children with a dual diagnosis of ASD and ADHD (e.g., Goldin et al., 2013; Yerys et al., 2009). This is the first evidence for a potential moderating effect of one risk type on another. However, because we are assessing the effects of co-occurring ASD and HI risk and not those of co-occurrence of these symptoms in a particular individual, we are unable to say at this point whether HI risk acts to reduce the effects of ASD risk in the same children or whether the “typical” performance of the HR/ASD+HI group results from some children having inherited the ASD risk factors and other HI risk factors. Some indication that the former hypothesis might be true comes from the fact that HR/ASD+HI performance variance is comparable to those of the other two groups (Levene’s F(2,89) = 1.17, p > .1; Figure S1). It is important to highlight the fact that whether co-occurring conditions result in additive, multiplicative or a completely new phenotype may vary from one phenotypic trait to another and therefore the current findings should not be generalized.

Why are the effects of proband hyperactivity/inattention more prominent earlier in development? Transitory signatures of risk have been reported previously. For example, 9 month old visual search performance is a predictor of later ASD symptoms, but the performance of 15 month olds does not relate to later outcome (Gliga et al. 2015). Similarly, the amount of looking towards faces is a predictor of later face recognition when measured at 8 months, but not at 14 months (deKlerk et al, 2014). It is possible that these developmental changes reflect adaptive mechanisms following initial perturbations in brain functioning, mechanisms that eventually mask the perturbations, but not before development has been set on an atypical pathway (Johnson, Jones & Gliga, 2015). It also remains possible that our findings do not
reflect heritable genetic background but environmental effects. Growing up alongside a sibling with a developmental disorder may in itself affect development. Proband hyperactivity symptoms may be more perturbing when the sibling is younger, thus explaining the stronger relationship found at 8 months of age. Despite the uncertainty regarding the mechanisms behind the effects we document here, these findings remain novel and important as they add to our understanding of both early learning and psychopathology. How infants explore visual information could have long-term consequences on their learning abilities (see SOM). We also reveal putative mechanisms for resilience, where high levels of hyperactivity/inattention in older siblings, through shared genetic or environmental mechanisms, can have paradoxical effects, being associated with optimal information foraging in infancy.


Klein, R. M., & MacInnes, W. J. (1999). Inhibition of return is a foraging facilitator in visual search. Psychological science, 10(4), 346-352.


Table 1. Participant characteristics. Mullen ELC: Early Learning Composite standard scores; ADOS SC: Autism Diagnostic Observation Scale, Social Interaction and Communication Composite; * indicates a main effect of group; letters indicate significant paired comparisons (Bonferroni correction, p <.05); * number of participants contributing eye-tracking data at each age.

<table>
<thead>
<tr>
<th></th>
<th>LR</th>
<th>HR/ASD-HI</th>
<th>HR/ASD+HI</th>
</tr>
</thead>
<tbody>
<tr>
<td>F:M</td>
<td>29:21</td>
<td>16:10</td>
<td>10:10</td>
</tr>
<tr>
<td><strong>Time 1</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age months (SD)</td>
<td>7.87 (1.1)</td>
<td>8.01 (1.1)</td>
<td>7.56 (1.4)</td>
</tr>
<tr>
<td>Trial no. (min-max)</td>
<td>13.83 (12-14)</td>
<td>13.85 (13-14)</td>
<td>13.17 (4-14)</td>
</tr>
<tr>
<td>Mullen ELC* (SD)</td>
<td>104.42 (11.31) (^{a,b})</td>
<td>91.30 (11.21) (^a)</td>
<td>95.21 (14.24) (^b)</td>
</tr>
<tr>
<td><strong>Time 2</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age months (SD)</td>
<td>13.91 (3.1)</td>
<td>14.48 (1.2)</td>
<td>13.80 (1.6)</td>
</tr>
<tr>
<td>Trial no. (min-max)</td>
<td>13.67 (4-14)</td>
<td>13.71 (11-14)</td>
<td>13.94 (13-14)</td>
</tr>
<tr>
<td>Mullen ELC (SD)</td>
<td>106.10 (15.72)</td>
<td>99.07 (17.31)</td>
<td>98.20 (18.94)</td>
</tr>
<tr>
<td><strong>Time 3</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age months (SD)</td>
<td>38.22 (3.05)</td>
<td>37.42 (1.4)</td>
<td>37.20 (1.7)</td>
</tr>
<tr>
<td>Trial no. (min-max)</td>
<td>9.60 (7-10)</td>
<td>9.95 (5-14)</td>
<td>10 (10-10)</td>
</tr>
<tr>
<td>Mullen ELC* (SD)</td>
<td>115.79 (16.27) (^a)</td>
<td>100.92 (22.61) (^a)</td>
<td>111.94 (22.61)</td>
</tr>
</tbody>
</table>
Figure 1. Example stimulus, areas of interest (AOIs) and visit coding. Successive fixations within and AOI were coded as one visit. The 3rd visit, in this example, is a revisit to the face.

Figure 2. Likelihood of revisitation for the 3rd visit in the trial; bars represent 1 SEM (left); Dimensional relationship between revisit likelihood at 8 months and proband Hyperactivity/Inattention score (right).
Supplemental Figures:

**Figure S1.** Scatter plot of re-visit likelihood at 8 months

**Figure S2.** Re-visit likelihood at 8 months, depending on ASD Outcome (left) and the same data split by outcome and HI risk (right)

**Supplemental Analysis:**
The relationship with concurrent and later learning abilities. Since higher proband hyperactivity and inattention (HI) is associated with foraging values that are closer to the chance level (even for high levels of HI, foraging does not become exploratory), this may reflect poor memory for previous visit locations rather than more optimal
information foraging (in both the Low risk and the HR/ASD+HI groups). This is where investigating the impact foraging measures have on learning, becomes important.

To ask whether early foraging and learning relate to each other during development, revisit likelihood at 8 and 14 months was entered in a cross-lagged autoregressive model with the Early Learning Composite (ELC) standard score at all ages (Figure S3). Because of the high data loss in the foraging measure at 3 years, we have not included this measure in the analysis. Autoregressive models test how the variance-covariance matrix changes over time and are thus ideal for dressing developmental hypothesis. The model fit well the data ($\chi^2 = 9.20; p = .16; CFI=.93$). Eight-months foraging measure significantly predicted 3-years ELC (STDEV standardized estimates: $\beta=-.198; S.E.=.07; p=.008$), with higher likelihood of return predicting lower ELS scores. Concurrent relationships between foraging and ELC were not significant.

After taking into account longitudinal relationships in foraging at different ages and also in learning abilities at different ages, as well as bi-directional relationships between foraging and learning at later time points, foraging at 8 months significantly predicted learning at 3 years of age, with decreased exploratory predicting poor learning. This direction of the relationship is very much in line with previous studies investigating object exploration (Bornstein, Hahn, & Suwalsky, 2013). However, since both highly explorative foraging (i.e. always driven to novel information and never to consolidation) and highly exploitative foraging (i.e. only extracting information from a restricted number of sources), restrict learning, a U-shaped relationship is expected to relate foraging to learning. It is probably because we investigated information foraging in a population at high-risk for ASD that we only observe one side of the U-shaped relationship. Future studies assessing populations at risk for only ADHD or populations with an ADHD diagnosis, might capture the other end of this relationship, where extreme exploration, i.e. rarely returning to re-inspect and consolidate information, may also be related to poor learning.

Figure S3. Auto-regressive cross-lagged model. Dark arrows depict significant relationships, and the numbers associated with them are the standardized estimates.