Precise minds in uncertain worlds: Predictive coding in autism

Sander Van de Cruys[1], Kris Evers[1,2,3], Ruth Van der Hallen[1,2,3], Lien Van Eylen[2,4], Bart Boets[2,3], Lee de-Wit[1], Johan Wagemans[1,2]

[1]Laboratory of Experimental Psychology, KU Leuven, Belgium; [2]Leuven Autism Research (LAuRes), KU Leuven, Belgium; [3]Department of Child Psychiatry, UPC-KU Leuven, Belgium; [4]Parenting and Special Education Research Unit, KU Leuven, Belgium

[In press, please cite as Van de Cruys, S., Evers, K., Van der Hallen, R., Van Eylen, L., Boets, B., de-Wit, L., Wagemans (2014). Precise minds in uncertain worlds: Predictive coding in autism. *Psychological Review*, in press.]

There have been numerous attempts to explain the enigma of autism, but existing neurocognitive theories often provide merely a refined description of one cluster of symptoms. Here we argue that deficits in Executive Functioning, Theory of Mind, and Central Coherence can all be understood as the consequence of a core deficit in the flexibility with which people with Autism Spectrum Disorders (ASD) can process violations to their expectations. More formally we argue that the human mind processes information by making and testing predictions, and that the errors resulting from violations to these predictions are given a uniform inflexibly high weight in ASD. The complex, fluctuating nature of regularities in the world, and the stochastic and noisy biological system through which we experience it, requires that, in the real world, we not only learn from our errors, but we also need to (meta)learn to sometimes ignore errors. Especially when situations (e.g., social) or stimuli (e.g., faces) become too complex or dynamic, we need to tolerate a certain degree of error in order to develop a more abstract level of representation. Starting from an inability to flexibly process prediction errors, a number of (seemingly) core deficits become logically secondary symptoms. Moreover, an insistence on sameness or the acting out of stereotyped and repetitive behaviors can be understood as attempts to provide a reassuring sense of predictive success in a world otherwise filled with error.

Keywords: autism spectrum disorder; predictive coding; uncertainty; adaptive control; learning

[Funes] was disturbed by the fact that a dog at three-fourteen (seen in profile) should have the same name as the dog at three-fifteen (seen from the front). His own face in the mirror, his own hands, surprised him on every occasion ... He was the solitary and lucid spectator of a multiform world which was instantaneously and almost intolerably exact ... he was not very capable of thought. To think is to forget a difference, to generalize, to abstract. In the overly replete world of Funes there were nothing but details, almost contiguous details.

— Jorge Luis Borges, 1942

Corresponding author: Sander Van de Cruys, Laboratory of Experimental Psychology (University of Leuven), Tiensestraat 102, 3000 Leuven, Belgium. Email: sander.vandecruys@ppw.kuleuven.be

Autism Spectrum Disorder (ASD) refers to a group of neurodevelopmental conditions with an early onset, and characterized by socio-communicative impairments and stereotyped, restricted behavior patterns and interests (DSM-5, American Psychiatric Association, 2013). Although ASD has a strong polygenetic component with heritability around 70% (Geschwind, 2011), no biological marker is available yet and thus, diagnosis mainly relies on behavioral assessment. The prevalence of ASD is estimated to be 1%, with males being more affected than females (Baird et al., 2006; Pinborough-Zimmerman et al., 2012). ASD is associated with increased comorbidity for other disorders (e.g., ADHD, anxiety disorders, tic disorders, learning disabilities and epilepsy) (J. L. Matson & Nebel-Schwalm, 2007). In addition, a significant proportion of the ASD population is intellectually disabled (Elsabbagh et al., 2012).

The neurocognitive frameworks put forward to account

for behavioral symptoms in ASD can be broken down into two groups, depending on which symptoms they consider to be central and preceding the others. Social first theories put problems with social cognition or motivation front and center. The most prominent contender is the Theory of Mind framework (Baron-Cohen et al., 2000) focus on the social problems and argue that the core deficit lies in the understanding of the behavior of others in terms of their underlying mental states. Nonsocial theories, on the other hand, consider general cognitive or perceptual problems to be the primary causal factor. Among them, the Weak Central Coherence theory (WCC; Frith & Happé, 1994; Happé & Booth, 2008; Happé & Frith, 2006) and the Enhanced Perceptual Functioning theory (EPF; Mottron & Burack, 2001; Mottron, Dawson, Soulières, Hubert, & Burack, 2006) focused on the perceptual peculiarities in ASD and argued for a locally (as opposed to globally) oriented processing style in individuals with ASD. Accounts which, prompted by the symptom cluster of repetitive and inflexible behavior patterns, situate the core deficit in an executive dysfunction (e.g., E. L. Hill, 2004), also belong in this group of nonsocial theories.

These theoretical frameworks are not mutually exclusive but focus on different behavioral symptoms. Each theory was highly influential in shaping the field, shifting the research and clinical focus from an exclusively descriptive behavioral approach towards an enhanced desire to understand the atypical neurocognitive mechanisms in ASD. Nevertheless, serious limitations with these frameworks have become evident over the years. First, whilst local processing styles, Theory of Mind difficulties and executive problems are common in ASD, they are neither specific to the disorder, nor are they universally apparent in all cases. Second, although they are called neurocognitive, they do not readily connect to underlying neural mechanisms, except in terms of broad networks of neural activation associated with each domain of function. Part of the problem is a lack of specificity in the proposed cognitive mechanism. Finally, while each of these frameworks attempts to incorporate more than the symptom cluster or behavior on which it is based, this often seems contrived, precisely because each theory is too closely intertwined with the cluster of symptoms in question.

We will argue that the way in which individuals with ASD process and respond to errors (or violations to their predictions) provides an excellent candidate for a primary dysfunction which, when viewed in the context of a complex developmental trajectory, provides a mechanistic explanation for the different symptoms of ASD. This imbalance in the brain's handling of prediction errors could result from different genetic and neurophysiological pathways, thus highlighting that different pathogenetic factors could in fact contribute to a common information processing imbalance (Geschwind, 2011).

We structured the current article as follows. In the first section (Section 1) we briefly introduce the predictive coding framework as it originated from perception research, but evolved into a unifying theory of brain functioning. In the second part (Section 2) we propose a specific etiological mechanism for ASD, which will then be applied to the different symptom clusters and clinical observations in ASD. Because of the developmental nature of the disorder, we start with a discussion of exploration and development (Section 3). Next, we discuss how perceptual and cognitive alterations in ASD can originate from our theory (Section 4). In the subsequent sections, sensorimotor and affective consequences will be covered (Section 5 and 6). In Section 7 core principles from earlier sections come together to explain problems in social functioning in ASD. Then, we briefly consider possible neural substrates of the proposed cognitive deficit (Section 8). Before reaching our conclusions, we cover a few related approaches of ASD to discuss commonalities and indicate the added value of our approach (Section 9).

1 The anticipating brain

Prediction is central for adaptive, intelligent systems (Hawkins & Blakeslee, 2004). It allows us to efficiently prepare for impinging circumstances that may foster or threaten continued subsistence. However, prediction-based computations can only succeed when there are in fact reasonably predictable contingencies in the world. Prediction, therefore, depends upon an animal's sensitivity to statistical regularities in the environment and in its interaction with it. Some of this structure is readily available, other parts are accessible only through higher order correlations. Our understanding of the role of predictions in shaping information processing has recently taken a step forward by the development of "predictive coding" models (Clark, 2013b). This computational scheme is heavily inspired by perception-as-inference (Helmholtz, 1910/1962) or perception-as-hypothesis (Gregory, 1980) ideas, which assume that the brain continually generates predictions on what input comes next based on current input and learned associations. Predictive coding, however, does not just stipulate that predictions are generated, but that these predictions are compared (at many levels of the system) to incoming sensory input, and that the comparison leads to the computation and representation of an error signal. These prediction errors are important, because they signal that the current generative model of the world —the one used to generate the currently best prediction— is not up to the task of explaining (predicting) the world. Once a prediction error has been signaled, the system still has to employ some degree of flexibility in deciding what do to with that error signal. In an uncertain world, experienced

via an inherently noisy biological processing system, errors will sometimes be spurious and uninformative. Thus, whilst prediction errors should sometimes be taken very seriously in updating one's predictive model, it is also critical that some prediction errors are essentially ignored. It is in the imbalance between these options that we think the symptoms of ASD are to find their cause.

In terms of neural architecture, predictive coding assumes a dual computational role for every level of processing (Egner, Monti, & Summerfield, 2010). Representation or prediction units compute predictions that are fed back, while prediction error units compute the difference between sensory input and top-down prediction. These prediction errors then serve as feed-forward input for the next level. The biological plausibility of this specific architecture is still under investigation, but the importance of prediction errors and predictive processing in the brain in general is well-established. Predictive coding can account for fundamental stages of perceptual processing, such as the emergence of extra-classical receptive field effects measured with single cell recordings in the primary visual cortex (Rao & Ballard, 1999). It can also account for the complex dynamics between predictions made and input received at very different stages of the system (den Ouden, Daunizeau, Roiser, Friston, & Stephan, 2010). Furthermore, it can explain neural dynamics such as the apparent adaptation to predictable stimulus contingencies (Summerfield, Monti, Trittschuh, Mesulam, & Egner, 2008). Finally, there is some evidence for the existence of separate representations for input and error signals in the recent discovery of differential sensitivity to predictable stimuli in separate clusters of voxels in the Fusiform Face Area (de Gardelle, Waszczuk, Egner, & Summerfield, 2012).

The computational scheme of predictive coding is assumed to repeat on every level of the perceptual hierarchy (Diuk, Tsai, Wallis, Botvinick, & Niv, 2013; Wacongne et al., 2011). Each higher level can capture a higher order regularity in input, relating events spanning more time or space, because it can work on the representational "language" of the previous level. Perceptual inference is guided in a top-down way through higher-level, conceptual predictions that can be passed downwards generating a chain of interdependent predictions to match on different levels, from complex features to low-level stimulus characteristics.

Formally, predictive coding is equivalent to Bayesian inference with the priors replaced by predictions and sensory evidence replaced by prediction errors, reflecting the mismatch between the input and the predictions. However, the differences between these two related approaches have important implications. A first distinction from Bayesian approaches concerns the more specific claims about the neural implementation of predictive coding. Second, replacing sensory evidence by prediction errors emphasizes that incoming information is put in context from the very start. It immediately becomes input relative to the organism, its models of the world and its current state. It also emphasizes that processing does not start with the onset of a stimulus. Pre-existing, intrinsic activity of the brain is considered formative as it reflects the continuous predictive activity of the pro-active mind-brain (Bar, 2009). Another advantage of predictive coding is that it allows a natural connection to other neurobehavioral domains, where prediction errors are known to play a crucial role, like midbrain dopaminergic processing of reward (Schultz, Dayan, & Montague, 1997), hippocampal processing for contextual memory (Honey, Watt, & Good, 1998) and amygdalar processing for fear learning (Boll, Gamer, Gluth, Finsterbusch, & Büchel, 2013). This suggests we may be a step closer to a general theory of the brain as a prediction engine in which prediction errors emerge as the lingua franca of neural information processing (den Ouden, Kok, & Lange, 2012).

Critically to our theory of ASD, predictive coding operates on two time scales (Dayan, 2012; Friston, 2010). Predictions are used here-and-now to shape one's online estimation of the state of the world (albeit through an iterative process), but the resulting prediction errors also shape plasticity and learning over longer time scales. In this way, today's prediction errors shape tomorrow's predictions (paraphrasing a famous Bayesian dictum). Because the world is not static, predictable contingencies that used to hold can change, and predictive coding has to track these dynamics. No two experiences are ever completely the same, thus, prediction error will always be present to some degree. However, the brain has no direct, independent means of differentiating mere noise from actual changes in the world (Feldman, 2013). It is, therefore, critical that predictive coding incorporates a mechanism to flexibly alter the extent to which the prediction errors generated by online estimation affect future learning and plasticity.

A solution to this can be found in terms of a flexible adjustment of what Friston (2010) describes as the *precision* of the prediction errors. To explain precision, one can draw the parallel with the means comparison in a t-test, in which the numerator represents the prediction error, which is weighted by the estimated standard error (precision or confidence) (Friston, 2009). As in the t-test, precision is not given in perceptual inference, it has to be estimated as well. In an optimal system, precision has to increase when there still are learnable regularities in the environment, and decrease when it is estimated that remaining deviations can be attributed to noise that is unlikely to repeat in next instances or to other irreducible uncertainties in input. Distinguishing between irreducible and reducible un-

certainties is a fallible process, relying on complex meta-predictions for a given context. The system, therefore, has to attribute a value or weight to prediction errors in order to determine to what extent they should induce new learning. The role of precision is conceptually the same as that of the learning rate parameter in Rescorla-Wagner learning (see Courville, Daw, & Touretzky, 2006; O'Reilly, 2013, for a full discussion on learning in volatile environments). Setting precision consequently relies on a form of meta-learning: learning what is learnable (Gottlieb, 2012) or estimating the predictability of new contingencies. It is clear from all of this that precision should be a context-sensitive measure, to be flexibly optimized dependent on the current class of input and the state of an organism. Indeed, precision is assumed to be the mechanism of attention within predictive coding. At its core, attention is the process of deciding where to look next, to allocate resources to that information with the highest value, understood precisely as input containing reducible uncertainty (Dayan, Kakade, & Montague, 2000; Gottlieb, 2012). Neurally, precision is assumed to be represented by the gain of bottom-up neural units representing the prediction errors, probably mediated by neuromodulators (Friston, 2009; see Section 8).

From this brief overview it should be apparent that predictive coding provides a framework that allows us to go beyond unidirectional views of information processing. Bottom-up information streams (predictions errors) are inherently dependent on top-down influences (predictions), which in their turn are shaped by previous prediction errors. This complex interplay also means that the dysfunction of one will automatically have consequences for the other. Disturbances in the relative contribution of top-down versus bottom-up information flow have been at the heart of two influential cognitive theories of ASD, representing apparently diametrically opposing positions (WCC and EPF). A predictive coding approach provides a principled and refined view on the influence of top-down versus bottom-up processes and their complex interplay.

2 Predictive coding in ASD

To bring into focus what we believe is the core processing deficit in ASD, we have to emphasize again the distinction between reducible and irreducible uncertainty (prediction errors). Irreducible uncertainty is due to the inherent stochastic nature of the world and the inherently noisy biological apparatus with which we sample from that world. Differentiating between reducible and irreducible uncertainty requires an estimation of "expected uncertainty" based on previous prediction errors (Preuschoff & Bossaerts, 2007; Yu & Dayan, 2005). If through learning, you estimate the outcomes of a stochastic process to vary with $\pm$ 3 (hypothetical) units, a prediction error of 2 should not surprise you, and therefore should not urge you to update your model (prediction). When the size of a prediction error is smaller than the expected variability (based on past prediction errors) for this event, the current prediction error should be scaled down. Reducible uncertainty, on the other hand, is present when associations in the world (or our interaction with it) are not fully learned yet. The latter is sometimes called unexpected uncertainty. This is the case when previously predictive cues have changed and become invalid, so a real update of the model is necessary. More formally, it is about situations in which correlations between predictions and prediction errors have changed.

In relatively unambiguous situations, people with ASD can successfully learn and apply new contingencies (M. Dawson, Mottron, & Gernsbacher, 2008). Problems arise, however, when the predictive value of learning cues changes (i.e., in volatile environments). For that reason, we situate impairments in ASD in meta-learning: learning which cues of all present stimuli are learnable, i.e., can reliably predict future situations relevant for the task at hand. This meta-capacity, estimating for which cues predictive progress can be made, allows typically developing (TD) individuals to distinguish random variability in input from actual, learnable changes in environmental regularities. Here, we advocate that individuals with ASD overestimate the amount of changes in environmental regularities, because they give too much weight to their prediction errors.

Another way to conceive of this meta-learning capacity is in terms of knowing where gains can be made in predicting the world. If you know where predictive progress can be made, you know which prediction errors matter, hence which prediction errors should be assigned high precision. Precision is the mechanism of attention in predictive coding because in this way it affects the further sampling of the sensory world. Atypical attention happens to be among the earliest signs of ASD, described in terms of the flexible and appropriate assignment of salience to stimuli (Elison et al., 2013; Zwaigenbaum et al., 2005). In ASD, the atypical distribution of attention has been attributed to slower encoding, which is consistent with the thesis that too many resources are invested in sensory processing because precise prediction errors cannot be discounted and thus attract further processing.

Hence, derived from a general theory of information processing, predictive coding, and our analysis of what could be the key problems in ASD, we situate the core deficit in the High, Inflexible Precision of Prediction Errors in Autism (HIPPEA). Low-level sensory prediction errors are generally set at a level of precision that is too high and independent of context (Palmer, Paton, Hohwy, & Enticott, 2013; Van de Cruys, de-Wit, Evers, Boets, & Wagemans, 2013). As mentioned before, it is useful to consider the consequences with regard to online inference

versus those regarding learning separately. If prediction errors during online inferences get an unduly high precision, these will urge new learning for every new event. The predictions that result from this learning will be shaped by noise that is unlikely to repeat in the future, hence these predictions will almost never be applicable. In neural network learning studies, overfitting takes place when errors for the training set are reduced to an exceedingly low level (Bakouie, Zendehrouh, & Gharibzadeh, 2009). It is a suboptimal form of learning because new data (acquired with each new experience) will generate large errors, meaning that there is little or no generalization. If errors are always deemed important, every new instance will be handled as an "exception", different from previous experiences. In the long run, however, those affected by this dysfunction, may succumb to a sort of learned helplessness: too much learning with no fruits. This may have an especially demotivating effect on particularly "noisy" interactions, such as those involved in social situations (see Section 7).

With regard to the consequences for online inference and behavior we have to distinguish situations in which an exact match from cue to prediction exists and is functional from situations in which exact matches will rarely happen or are even dysfunctional. In the case of exact matching, it is well-known that people with ASD cope incredibly well (Mottron et al., 2013). They often excel in rigid, exact associations (rote learning). Here, their overfitted predictions serve them perfectly well, precisely because they suffer less from interference from similar instances. They seem to trade off the ability to generalize with a more accurate memory. Hence, according to HIPPEA, the core processing deficits in ASD become most evident when some disregard for details and some generalization is needed. Generalized inferences are required in situations where exact matches are not present, which is the rule rather than the exception in natural situations, especially those involving social interactions.

In everyday life, multiple cues impinge simultaneously on an individual. At first exposure this may cause sensory overload, because selectivity is lost when the informational (predictive) value of cues cannot be estimated immediately. Predictions are tested but violated because they are based on spurious correlations. Individuals with ASD may cope with perceived repeated changes in contingencies by executing prepotent, impulsive or "model-free" behaviors, described as repetitive, stereotyped behaviors in the ASD symptomatology (for a discussion on the role of precision in arbitrating between model-free and model-based behavior, see Clark, 2013a; Daw, Niv, & Dayan, 2005). In a second stage, individuals with ASD may "give up", and select cues just to evade and cope with prediction errors. On their own scale, these cues may be highly predictable, even though they are not functional in the situation at hand. Thus, attention and behavior become dominated by one or a few cues (cf. stimulus overselectivity; Lovaas, Koegel, & Schreibman, 1979), singled out seemingly arbitrarily.

Note that computing prediction errors as such is not impaired in ASD according to this view. Individuals with ASD can still compare their predictions with actual input. These prediction errors, however, have to be weighed in accordance to an estimation of their reliability, i.e., the extent to which they are caused by learnable (changes in) regularities. Attesting to the fact that prediction error computations are intact in people with ASD, their detection and discrimination performance seems to be similar to typically developing individuals, if not superior (see Section 4.2).

Importantly, one can distinguish between two mechanisms that both can result in inflexibly high precision of prediction errors. First, it is possible that the neural mechanism for precision is directly affected in ASD, fixing precision at a high level and preventing meta-learning (which may take place anyway) to have an effect on perception and learning. Aberrant neuromodulatory mechanisms of precision, as discussed in Section 8, may be responsible here. Second, the meta-learning prior to the setting of precision may be deficient in ASD and hence does not provide the needed basis for proper, context-dependent estimation of precision. Neural regions and mechanisms that may be central for this capacity are discussed in Section 8.

In a nutshell, HIPPEA consists of the following basic premises. The starting point is a high *and* inflexible estimation of precision of prediction errors in ASD. This indicates meta-learning is deficient or short-circuited. Indiscriminately high precision will mean that unrepeated, accidental variations in the input receive disproportionate weight. This in turn, prevents abstract representations to be formed, because matching will continue on a more specific level, closer to the input. Indiscriminately high precision also induces superfluous learning, leading to narrowly defined, lower-level predictions, and incomplete hierarchical models. Finally, indiscriminately high precision entails a loss of autonomous, flexible attentional selection based on informativeness (deciding what information to sample based on the different types of uncertainty in input).

HIPPEA thus situates problems in ASD at the intersection of perception, attention, learning, and executive functioning (adaptive control). Further key symptoms of ASD may emerge from this impairment, but this will be fleshed out in the sections below. We argue for an impairment in general information processing rather than in one single domain (e.g., social cognition), supported by the fact that problems in ASD are not limited to one such domain, but are pervasive. However, this also puts the burden of explanation with us as to why some domains (specifically

the social) would be affected more than others (see Section 7).

3 Development and exploration

The meta-learning deficit in HIPPEA is very consistent with the developmental nature of the disorder. The very process of moving from one "simpler" developmental stage to the next "complexer" one is impaired when an organism cannot estimate where predictive progress can be made. If any prediction error is deemed as valuable as the next, an inappropriate lingering on stimuli is expected to occur. As a result, the kind of exploration that optimizes learning is lacking, because estimating where predictive progress can be made helps an organism to avoid the large regions of input space that cannot be learned (fully) and those that are too difficult at this stage of development. In short, this principle gives a rationale for the importance of intermediate levels of complexity in development (Berlyne, 1966; Gibson, 1969; Oudeyer, Baranes, & Kaplan, 2010). If predictive gain can be properly estimated, exploration can be guided such that it is aimed at regions with a difficulty just above current ability, which leads to discernable progressive stages of increasing complexity, as modeled in developmental robotics (Oudeyer, Kaplan, & Hafner, 2007). Particularly in noisy, variable environments the mechanism can be expected to realize more efficient learning. It is easy to see that if this capacity for active exploration is missing, as we think is the case in ASD, an individual has to rely much more on the scaffolding provided by caregivers, explicitly guiding progression from simple to more naturalistic situations. Apart from prenatal genetic and neural components, differing degrees of this environmental scaffolding may account for heterogeneity in symptom severity and developmental trajectories in ASD.

The link between prediction violation and exploration is elegantly illustrated in a study by Legare (2012), investigating how TD children explain evidence violating their predictions and illustrates how this mechanism may shape development. Different shapes were put on top of boxes that could light up, depending on the shape, and those shapes that caused the box to light up were subsequently labeled as a "blicket". Children were then confronted with a violation of the established prediction (no light for a blicket) and Legare (2012) asked them to explain what had happened. She could distinguish two main types of explanations; about half of the children tried to explain *why* the block did not light up (e.g., no batteries, block was not placed properly), while a third of the children explained the situation by referring to the category membership (e.g., "It's not really a blicket; it only looks like one"). Most interestingly, however, the kind of explanation children gave predicted the way they played with the objects later on. While children who gave a causal explanation explored the objects more thoroughly, testing different combinations and experimenting with the placement and orientation of objects to find out what would happen, children who explained inconsistency in terms of the categories primarily went about sorting the objects in two different categories based on what had happened when they first placed them on the box. This sorting behavior was a less sophisticated form of exploration, and less likely to foster deep understanding of the underlying sources of inconsistencies.

Arguably, the difference hinges on the ability to model uncertainty in associations in the input. This modeled uncertainty becomes a handle to dissect underlying causes. The precision of low-level inconsistency can, with a proper model of uncertainty, be down-regulated such that the general rule (prediction) is not violated and so does not have to be abandoned. Rather, modeling uncertainties in the task opens the door to contextual modulations of the general rule, which are always at play in practice. When uncertainty is not accounted for, and precision is continuously high, every minor violation will induce new learning. An inconsistent finding is categorized anew or considered a "special case" unlike previous instances. The latter is what HIPPEA proposes to be the case in ASD. Though Legare's (2012) study only included TD children, the sorting behavior found in spontaneous play for the subset of children that gave non-causal explanations, is reminiscent of what is observed in autistic play. Her results show that whether and how people explain prediction error is linked to the kind of exploration they will engage in. In our line of thinking, the difference already emerges in the way people process perceptual input that runs counter their predictions and this may have far-reaching consequences for exploration and further development, notably with regard to finding out about why the world functions as it does.

Considering this change to the nature of exploration in ASD, it is informative to revisit the so-called "dark room problem" within predictive coding (Friston, Thornton, & Clark, 2012; Froese & Ikegami, 2013). This problem arises because if, as the fundamental thesis of predictive coding has it, an organism acts to minimize the prediction errors it experiences, then the simplest solution would be to seek out a dark room, devoid of prediction errors. Nevertheless, most organisms venture out of their black boxes and explore the world. The most obvious way to counter this is by noting that generalized predictive coding not only involves learned mental models and perceptual predictions, but also bodily predictions, predictions embodied by the very structure of the body, homeostasis, biomechanics and the "gross initial neural architecture of the agent" (Friston et al., 2012). Evolution equips organisms with a limited set of expected states (cf. homeostasis) that is compatible with their continued existence (survival). A dark room will not

remain a low prediction error environment, for instance, when food is not available.

One means of ensuring that organisms venture out to meet their needs for survival, is to equip them with an ability to tolerate the prediction errors associated with new unpredictable environments. Organisms can adjust the precision of prediction errors based on the expected volatility of their environment. If precision of low-level prediction errors is overly high, however, the organism may very well prefer to stay in a dark room-like environment. In fact, the typical autistic state of stereotypic self-stimulation and indifferent withdrawal from the world and from others, can be regarded as "abnormal yet effective ways of reducing prediction errors" (Froese & Ikegami, 2013). By their caregivers, children with ASD are often decribed as detached from the world, as if they are living in their own walled world. Not because they are unhappy, or unable to move or sense, but because they are satisfied with the current level of complexity of the environment. The prediction error minimization principle says that "we harvest sensory signals that we can predict" (Friston et al., 2012). Hence, it seems that children with ASD, since they (initially) cannot predict more complex environments, are perfectly content to stick to the confined space and motion they know.

4 Cognitive functioning

In the following sections we review the most relevant literature illustrating the implications of prediction errors with chronically high precision in cognitive and perceptual domains. At the end, we will also describe the special perceptual and cognitive skills that some individuals with ASD have developed ("savant skills"), which can result from the potential benefits of high-precision prediction errors when applied to certain domains. The problems in reasoning about mental states ("mentalizing"), which are also a central aspect of cognition in ASD, will be covered in Section 7 on social functioning.

4.1 Attention and executive functioning

An interesting pattern of findings has emerged from attention studies in ASD, comprising both superior performance in certain tasks and severe deficits in others. Below we substantiate that the specific pattern is largely consistent with HIPPEA. We start considering visual search tasks, then move to more complex attention tasks with a larger executive component, and finally make new predictions based on our account and propose suitable designs to test these.

Visual search studies demonstrate that performance on some attentional tasks can be intact or even enhanced in ASD. Superior visual search has been found both when the target is defined by a single feature and for conjunctive targets (Keehn et al., 2009; O'Riordan, Plaisted, Driver, & Baron-Cohen, 2001; Plaisted, O'Riordan, & Baron-Cohen, 1998). Moreover, performance seems to correlate with symptom severity (Joseph, Keehn, Connolly, Wolfe, & Horowitz, 2009). Group differences are especially present in conjunction search tasks or tasks with higher difficulty. A predictive coding account of visual search would start from the predictability within search displays. When every item in a display reinforces a particular "prediction", an anomaly (the "odd one out") causes an "error" that becomes salient. Heightened precision of this prediction error means enhanced salience of this oddball, which facilitates quick detection. Thus, individuals with ASD seem to be just as good or even better at exploiting predictability in a display.

In more complex attentional settings however, performance usually declines substantially in autism. As we saw, precision (or weight) of prediction errors should be flexibly adapted based on meta-learning (learning what the relevant features in a task are). When precision of prediction errors is uniformly high, the selective force is lost when processing a context with multiple cues. Hence, difficulties in allocating attention may be expected. Phenomenally, this may manifest itself as attention to irrelevant features, on the one hand, and as lack of disengagement or perseverative attention, on the other hand. Yet, this problem occurs only when multiple cues compete. If only one cue is present, ensuring that the selection process is clearly imposed by the task itself, performance remains intact (Burack, 1994).

A study by Pierce, Glad, and Schreibman (1997) confirms this. When ASD children, TD children and mentally disabled children were presented with video fragments of social interactions containing one or more cues, ASD children performed worse than the other two groups when asked to answer a set of questions right after, but only in the multiple cue conditions. We argue the problem is one of autonomous selection; the relevance or redundancy of the cues is not recognized. Consistent with this, task performance in ASD is expected to suffer most when it is dependent on autonomous exploration and efficiently probing of available cues rather than fixed instruction (clear top-down selection).

Others before us (e.g., Elsabbagh et al., 2009; Keehn, Müller, & Townsend, 2013) have situated the origin of problems in ASD in attentional difficulties, more specifically in disengaging attention. However, we consider these disengagement problems not as primary but as an effect of the lack of adaptive precision of prediction errors. This kind of overselective (perseverative) attention does not stand in contradiction with what we said before on the lack of autonomous selectivity in ASD. It is the flexible adapting of selectivity in a task-dependent way that is lacking in ASD. Uniformly high precision will create

a prolonged processing of all stimuli (and an associated deficit in disengaging). This is also apparent in studies by Sasson and colleagues (Sasson, Elison, Turner-Brown, Dichter, & Bodfish, 2011; Sasson, Turner-Brown, Holtzclaw, Lam, & Bodfish, 2008), demonstrating perseverative attention (longer fixation times per image explored) and more intensive, detail-oriented exploration of a limited number of images in ASD. Hohwy and Palmer (2014) note that increased precision could lead to longer sampling of incoming signal in order to attain the precise signal people with ASD expect, before making a decision. If so, such longer sampling may as well help to explain larger reaction times for diverse tasks, often reported in ASD. In any case, we surmise that lacking disengagement is not the core mechanism but rather one of the consequences of HIPPEA. However, often perseveration and overselectivity may be strategically replaced by avoidance and underreactivity.

It is clear that the proposed difficulties in autonomous cue selection will cause broader problems in executive functioning, in particular with regard to cognitive flexibility or set shifting. According to the executive functioning theory, the latter functions are impaired in ASD and this impairment is assumed to underlie the restricted, repetitive and stereotyped pattern of behavior and interests (B. R. Lopez, Lincoln, Ozonoff, & Lai, 2005). Though problems with flexibility have clearly been found in daily life (Gioia, Isquith, Kenworthy, & Barton, 2002; Mackinlay, Charman, & Karmiloff-Smith, 2006), studies measuring cognitive flexibility in a clinical or research setting have yielded less consistent evidence. Overall, studies using the Wisconsin Card Scoring Task (WCST) report clear deficits, reflected by a higher number of perseverative responses when a rule switch should occur, while more controlled task-switching paradigms generally fail to find cognitive flexibility problems in ASD (Geurts, Corbett, & Solomon, 2009; Van Eylen et al., 2011).

Recently several researchers suggested that these inconsistencies may be due to differences in the extent to which explicit task instructions are given, denoted as open-endedness (Van Eylen et al., 2011; White, 2013). When task instructions contain no explicit indication of the rules to be applied, nor that a rule switch will occur (as in the WCST), results show rather consistent cognitive flexibility deficits in ASD. In this case, participants have to be able to autonomously filter-out and focus on relevant information in a situation where multiple cues compete. There is evidence that individuals with ASD have difficulties doing so and overly focus on irrelevant, often low-level details (Stoet & López, 2011). In contrast, when a cue explicitly indicates which rule to apply and when to switch, all studies report intact performance in ASD. Hence, the act of switching does not seem to be a problem per se (Poljac & Bekkering, 2012).

All this is very compatible with our interpretation of ASD in terms of an overweighing of prediction errors. As we saw, the informativeness of cues has to be derived from meta-models, which should adjust the precision with which errors based on these cues are weighed. A loss of this capacity would lead to a deficit in the ability to autonomously select cues that have predictive value in situations where multiple cues compete. Learning a new unambiguous contingency in itself is not a problem, but individuals with ASD struggle with spontaneously noticing that the predictive value of particular information changed. This leads to cognitive flexibility deficits on open-ended tasks, but not on tasks were explicit instructions are provided about what is informative and when. Testing a range of executive functions in ASD, White, Burgess, and Hill (2009) corroborated that all open-ended tasks generated group differences, while none of the more constrained tasks did. Hence, this reasoning might also explain some of the inconsistencies in studies of other executive functions (Gioia et al., 2002; White et al., 2009).

Open-ended, generative sorting tasks provide converging evidence. For example, in a free sorting task with children books (Ropar & Peebles, 2007), ASD children relied less on category labels (games versus sports) and more on purely perceptual features (color and size) than TD children. More one-dimensional sorting was found in free sorting of shapes by children with ASD, especially in more complex stimulus sets (Edwards, Perlman, & Reed, 2012). In a twenty-questions game, children with ASD consistently generated questions ("predictions" in our context) of lower quality, especially more concrete ones that eliminated fewer items at a time (Alderson-Day & McGonigle-Chalmers, 2011). Analyses indicated that difficulties in managing relevant and irrelevant information were likely sources of the problems of children with ASD. This cognitive control problem, which is at the heart of HIPPEA, also explains why individuals with ASD are particularly slower in early blocks of categorization learning, when flexibly switching the focus of attention from one dimension to another dimension is needed (e.g., Soulières, Mottron, Giguère, & Larochelle, 2011).

To clearly summarize our hypothesis: When real environmental changes go together with random changes, disentangling the two is particularly difficult for people with ASD. They seem to be able to learn changes in contingencies, when they are clearly indicated, as in some set shifting tasks. Similarly, they can learn fixed contingencies, even in probabilistic environments and without explicit instructions, as implicit learning studies show (J. Brown, Aczel, Jiménez, Kaufman, & Grant, 2010; Nemeth et al., 2010; Pruett et al., 2011). However, these two combined create the clearest deficits. Therefore, we hypothesize

that adding noise by using a probabilistic switching task, would increase their flexibility impairments. This has indeed been observed by D'Cruz et al. (2013) in a reversal learning task with intermittent non-reinforcement. Moreover, these switching problems correlated with severity of repetitive and restrictive behaviors. From our perspective, this kind of task will be most sensitive in picking up deficits in executive functioning for ASD.

Although these findings are largely compatible with the prediction derived from HIPPEA, future attentional studies should test our hypothesis more directly. A modified version of Posner's attention cueing task as developed previously (Vossel et al., 2013; Yu & Dayan, 2005) could contribute to this. In the typical Posner cueing task, a simple cue (a briefly presented flash) only indicates the actual (valid) location of the target in a certain percentage of trials (e.g., 75%). Typically participants will learn to use the cue information to improve their detection speed to the extent that the cue is reliable. This improvement may also be present for individuals with ASD, but we predict that things will go awry in ASD when the probabilistic structure changes unexpectedly during the experiment, for instance, when the predictability of a cue changes across blocks. In such a volatile environment, the validity of the cue (the extent to which it predicts the target location) varies over the course of the experiment. Prediction errors usually lead to the updating of beliefs (predictions) about the environment, but the impact of these prediction errors should be tuned to whether additional learning is expected to be still possible. In a fully learned stable phase, new prediction errors are probabilistic noise that should lead to little or no update of predictions. However, when new learning is estimated to be possible, for example when probability structure changed, recent prediction errors should significantly update current predictions. This task shows the importance of contextual, flexible setting of precision.

Another variation of the Posner task that could provide a useful test of our theory has been developed by Yu and Dayan (2005). In this version, a set of cues (e.g., differently colored arrows pointing left or right) precede the target. For any one trial, one particular cue (color) from the set predicts the target location with a certain probability (e.g., >.5). This cue type and validity remain active for a considerable amount of time, creating a stable environment. Then, unbeknownst to the participant, this context is suddenly changed: A different cue now predicts the target location with a different cue validity. Note the similarity with traditional set switching tasks, although the rules there usually are deterministic, rather than probabilistic. Participants with ASD will have distinct problems with this task, again because two forms of uncertainty are pitted against each other, as described above. An added benefit of these tasks is that a hierarchical Bayesian model can be used to quantify precision (or learning rate) on a trial-by-trial basis (Behrens, Woolrich, Walton, & Rushworth, 2007; Yu & Dayan, 2005), and on a subject-dependent basis (Mathys, Daunizeau, Friston, & Stephan, 2011; Vossel et al., 2013), pin-pointing exactly whether and how the learning style of ASD subjects differs from that of TD individuals.

From the above it should be clear that HIPPEA has a natural way of explaining the discrepancy between the experimental data in contrived laboratory contexts and the clinical observations in daily life. As most ASD researchers know, it is surprisingly difficult to find statistically significant group differences in the lab that should occur according to everyone's expectations based on the major problems that people with ASD experience every day (J. L. Amaral, Collins, Bohache, & Kloos, 2012). Natural circumstances are often much more unpredictable and open-ended with lots of accidental variability, and hence lead to clear deficits in ASD (Kenworthy, Yerys, Anthony, & Wallace, 2008). The lab, in contrast, usually provides a well-controlled environment, in which it is made very clear what is expected (explicit instruction, practice trials) and with multiple instances of the same (often simple) task (repeated trials). Little autonomous control is needed here. Where many TD children easily get bored in such a context and start talking to the experimenter, kids with ASD usually like these repetitive, computerized tasks, and they are motivated to do well in them.

4.2 Perceptual processing

Research on visual processing in ASD has been dominated by two related theoretical frameworks that each emphasized a different side of the coin: WCC theory emphasized reduced global processing (Frith & Happé, 1994), while EPF theory emphasized enhanced local processing (Mottron & Burack, 2001). More recent accounts describe the peculiar aspects of visual processing in ASD more in terms of a bias or perceptual style, a disinclination for global or a preference for local processing (Happé & Booth, 2008; Happé & Frith, 2006; Mottron et al., 2006). Despite a vast amount of research on visual perception in ASD, the atypical profile of visual processing is only partly understood, and the empirical evidence for the original ideas is mixed (for recent reviews, see Behrmann, Thomas, & Humphreys, 2006; Dakin & Frith, 2005; Simmons et al., 2009).

HIPPEA is compatible with both EPF and WCC, but it offers a more specific foundation, describes dynamics in learning and inference, and hence has different implications. According to HIPPEA, precision of bottom-up information is uniformly amplified, an idea that is consistent with EPF, but we can better specify how and why perception is enhanced. The detectable size of prediction errors

is not smaller, but rather the weights (precision) these errors receive are higher. HIPPEA does not reduce problems to a purely bottom-up way of perceptual processing. Because it is embedded in the inherently bidirectional predictive coding framework, the mutual, constructive interaction of bottom-up and top-down information flows is central. Specifically, increased precision of prediction errors will have important consequences with regard to the kind of predictions that will be formed based on prediction errors with unusually high precision. Perceptual inference and learning will not progress to higher-level, more abstract representations because of the emphasis given to violations to those higher-level representations at lower levels of processing. Learning will result in predictions tuned sharply to exact perceptual input cues. As a result, primarily low-level predictions will be formed, which will have limited applicability, while higher-level predictions will be triggered less automatically by incoming information, an idea that is consistent with WCC.

In ASD, stimuli are treated in an idiosyncratic manner, because slight deviations are perceived as informative and all experiences are thus more readily treated as new instead of as belonging to a known category. More concretely, the focus on prediction errors at lower levels causes individuals with ASD to focus on concrete but irrelevant changes in viewpoint or illumination, which impede the ability to progress to the more relevant, abstract levels of description in terms of shape or object identity. Note however, that the predictive machinery in ASD is not deficient in our view: Predictions are still formed and prediction error is computed correctly. Hence, global interpretations are not necessarily lost in ASD; they just require more experience and they will appear only under more constrained conditions. So, while a familiar representation may not pop-up automatically when a related stimulus appears, top-down activation of holistic, Gestalt-like templates and global processing are often still possible, but as a conscious strategy, when task instructions require it and enough time is available. For individuals with ASD, it is not the default, automatic processing mode. This accords nicely with the recent move in the field towards differences in default preference or bias (often measured by initial choice responses or reaction times) rather than in distinct inabilities (measured by error rates). This interpretation receives support from a recent meta-analysis of the mixed evidence from a variety of local-global perceptual processing tasks, which demonstrates that global processing takes time in individuals with ASD (Van der Hallen, Evers, Brewaeys, Van den Noortgate, & Wagemans, 2014). Moreover, the inconsistencies in the literature also make sense in this perspective. Laboratory tasks mostly use standardized stimuli, and often do not incorporate the noise that is usually present in real-life stimuli. In these constrained circumstances, individuals with ASD can actually perform on a typical level.

Low-level perception. According to HIPPEA, low-level differences will get boosted and sent upwards influencing behavior and learning. Setting precision high by default may give an advantage for lower-level processing (but impedes building and using of a hierarchy of predictions for generalization). In the auditory domain this is reflected in the frequently reported enhanced pitch perception in children and in a subgroup of adolescents and adults with ASD, especially those with early developmental language delay and language-related difficulties (for reviews, see Haesen, Boets, & Wagemans, 2011; O'Connor, 2012). Superior pitch processing has been established regardless of stimulus complexity (i.e., pure tones, complex tones, speech sounds, nonwords, words) using a variety of psychophysical tasks (e.g., identification, discrimination, memory) (e.g., Bonnel et al., 2010, 2003; Jones et al., 2009). Relevant in this context is also the increased prevalence of absolute pitch and musical savants in the ASD population (e.g., Heaton, Williams, Cummins, & Happe, 2008).

In visual perception, findings are more mixed. Most studies have found little or no group differences for visual acuity (Simmons et al., 2009). One study observed a small group difference indicating superior contrast sensitivity in individuals with ASD (Bertone, Mottron, Jelenic, & Faubert, 2005). Another study found evidence for superior visual acuity (Ashwin, Ashwin, Rhydderch, Howells, & Baron-Cohen, 2009), but has been disputed on methodological grounds (M. Bach & Dakin, 2009) and replication attempts have failed (Bölte et al., 2012; Kéïta, Mottron, & Bertone, 2010). Based on HIPPEA, however, there is still potential for well-controlled studies to find detection differences. In particular, it may be relevant to look at classic effects of perceptual gain control (Hillyard, Vogel, & Luck, 1998) in ASD, because precision is thought to rely on gain control of the output of neural units representing the perceptual prediction errors (Friston, 2009). Foss-Feig, Tadin, Schauder, and Cascio (2013) very recently found that detection of motion direction of a single clearly visible grating can be done based on significantly shorter presentation times in ASD compared to controls. The improvement was not present for low contrast gratings, for which gain control is negligible. Hence, it seems it is caused by reduced contrast saturation of high contrast gratings in ASD, consistent with a deficient gain control.

Local versus global perception. A common paradigm to study a more locally focused processing style in ASD is to examine their susceptibility to visual illusions. Overall, these studies yielded mixed to positive effects. While some authors did not find a difference in performance for ASD (C. Brown, Gruber, Boucher,

Rippon, & Brock, 2005; Rouse, Donnelly, Hadwin, & Brown, 2004), most others showed a diminished illusion susceptibility in ASD (e.g., Bölte, Holtmann, Poustka, Scheurich, & Schmidt, 2007; Mitchell, Mottron, Soulieres, & Ropar, 2010). This diminished susceptibility has been taken to imply that individuals with ASD are, in general, less influenced by contextual or prior information, remaining closer to the actual sensory input, an idea that is perfectly consistent with HIPPEA. For instance, when Ropar and Mitchell (2002) asked participants to estimate the shape of an illuminated disc presented at a slanted angle in a darkened room, control participants reported a more circular shape (closer to the inferred distal stimulus, discounting the slant), while participants with ASD reported a more elliptic shape (closer to the proximal stimulus, not discounting the slant).

The global-local processing issue is standardly studied using the block design task and the embedded figures task. The first study showed enhanced performance in both of these tasks in individuals with ASD (Shah & Frith, 1993), which was interpreted as evidence for reduced interference by the automatic processing of the global level. Later studies, however, yielded mixed results (e.g., Bölte, Hubl, Dierks, Holtmann, & Poustka, 2008; Ropar & Mitchell, 2001). Collectively, these results point to a difference in degree of efficiency or ease with which the task is performed, rather than a discrete performance difference (Van der Hallen et al., 2014).

Another domain in which the visual abilities in ASD have received a lot of attention is the perception of motion. A study by Bertone, Mottron, Jelenic, and Faubert (2003) revealed intact first-order (luminance-defined) motion processing but impaired second-order (texture-defined) motion processing. Motion coherence studies, in which observers have to track the presence or direction of coherently moving (luminance-defined) dots among differing proportions of randomly moving dots, generally yielded higher motion coherence thresholds in individuals with ASD (e.g., Milne et al., 2002; Pellicano, Gibson, Maybery, Durkin, & Badcock, 2005; Spencer et al., 2000), although there are also exceptions (De Jonge et al., 2007; Del Viva, Igliozzi, Tancredi, & Brizzolara, 2006; Saygin, Cook, & Blakemore, 2010). A recent study may explain this inconsistency (Robertson, Martin, Baker, & Baron-Cohen, 2012) by reporting a deficit in perception of motion coherence at short exposure durations which reduces with increasing exposure durations.

The finding that added noise is especially detrimental for global motion perception in ASD is one that follows directly from HIPPEA. Distinguishing noise and signal is particularly important in these paradigms. As explained before, people with ASD attribute unduly high value to noise that is unlikely to repeat, in an attempt to properly fit the input. Global motion will more readily "break" down for them, because they end up with errors that are too important to fit with an abstracted, global pattern. When the noise is absent, as in the plaid motion stimuli in Vandenbroucke, Scholte, van Engeland, Lamme, and Kemner (2008), global motion perception seems to be intact in ASD.

Research with bistable figures suggests that people with ASD can generate and maintain top-down predictions, because when guided to do so, they easily succeed in making the different interpretations of ambiguous figures (Ropar, Mitchell, & Ackroyd, 2003). However, we would advise to use binocular rivalry in future studies (rather than the pen-and-paper type face-vase or duck-rabbit tests used so far) because it has been proposed to be explained by predictive coding (Hohwy, Roepstorff, & Friston, 2008). Indeed input related to the suppressed image in binocular rivalry can be considered prediction error, because it is unexplained by the currently dominant percept. Only two studies have been performed so far with one showing unaltered binocular rivalry in ASD (Said, Egan, Minshew, Behrmann, & Heeger, 2013) and the other finding lower switch rates and more mixed percepts (Robertson, Kravitz, Freyberg, Baron-Cohen, & Baker, 2013). Mixed percepts could be the preferred way to minimize prediction errors in ASD, i.e. less explaining away through higher level constructs and hence staying "closer to the input". Note that care should be put into finding the right stimuli for use in autism, since availability of top-down templates evidently also influences rivalry. For example, the first study uses gratings while the second uses familiar objects. The less familiar (or semantically high-level) the better for use in ASD probably, at least when the focus is really on switching dynamics. Future binocular rivalry studies in ASD should specifically look at mixed percepts and fusion, because this the expected result according to predictive coding if precise prediction errors are present for both "hypotheses" (Hohwy et al., 2008). Another yet to be tested prediction from HIPPEA would be that adding noise (prediction error) to the input has a stronger effect on the breaking of one percept (and possibly inducing a switch) in ASD compared to controls.

Face and speech perception. Face and speech perception are crucial for smooth and successful social interactions, and therefore prominent targets of ASD research. Deep difficulties here can go a long way in explaining communication problems so central in ASD. Interestingly, face and speech perception are also prime examples of the hierarchical "analysis by synthesis" approach inherent to predictive coding. Normally this would provide inferences on high-level semantic sources of incoming sensory information (a generative model) that can cascade into multiple levels of predictions for activity in regions below, sup-

pressing or explaining away new input, as long as it is sufficiently well-predicted. Yet, what is sufficient has to be learned as well (meta-learning), given the requirements of speech or face understanding. If precision of prediction errors is invariably high, individuals with ASD will have more difficulties in "abstracting away" the short-term, contingent, low-level features of the stimuli. Behaviorally, this is expressed in individuals with ASD as a superior access to the underlying low-level visual or acoustic representations. On the other hand, they cannot fully exploit the higher-level predictions. This disadvantage is particularly felt in naturalistic face or speech-in-noise perception. The problem of which variations to encode in a given situation and which to disregard, comes to the forefront in both speech and faces, which is exactly one of the problems for individuals with ASD, according to HIPPEA. The brain does not only have to pick up and learn small auditory or visual differences, it also has to learn which ones are informative, in the sense of predictive for different kinds of social goals, and which differences to discard.

In speech, invariant phonetic cues are embedded within a variety of acoustic cues (e.g., fundamental frequency, accent, intonation, timbre, etc.) and can only be extracted by integrating and interpolating information, a process which is supported by higher-level linguistic guidance through phonotactic, semantic and syntactic constraints (predictions). During development very young TD children learn to generalize consonants, vowels and words across voices (e.g., of different gender), disregarding irrelevant absolute pitch cues in favor of more complex relative distances. However, in ASD we see increased access to fine-grained acoustic features of complex sounds in ASD (e.g., disembedding tones from musical chords; Heaton, 2003; Mottron, Peretz, & Menard, 2000) and superior perceptual processing of acoustic features of speech (e.g., Heaton et al., 2008; Jarvinen-Pasley, Peppe, King-Smith, & Heaton, 2008). Consistent with HIPPEA, it has been suggested that these individuals generate overly specific categories of sounds that impedes learning of higher-level abstract patterns (Crespi, 2013) needed for speech development. Early developmental language delays as well as broader linguistic impairments later in life are indeed prevalent in the individuals who show superior acoustic processing of pitch (Bonnel et al., 2010; Jones et al., 2009). Additionally, noise with similar characteristics as the signal (speech), substantially hinders performance in ASD (E. G. Smith & Bennetto, 2007), because these "errors" are not easily ignored.

A similar challenge is posed by faces, characterized by a very high intra-class similarity, with small and rather subtle differences amongst many dimensions distinguishing two human faces from each other. Countless transformations of an individual face amongst several dimensions should be ignored. A face has to be recognized despite variability in, for instance, lighting conditions, face orientation, changeable facial features (e.g., facial hair, spots, wrinkles, freckles), and extra-facial features (e.g., hair style, hats). Due to their meta-learning problems, individuals with ASD may fail to make this distinction between relevant and irrelevant variability, and hence get lost in non-functional characteristics. This may explain their poorer face memory and their face identity recognition problems (Weigelt, Koldewyn, & Kanwisher, 2012).

Paralleling evidence on global-local processing in general, there is no strong evidence for a reduced global or enhanced local face processing style in ASD. For example, no reduced face inversion effect, no attenuated composite face illusion, no diminished part-whole effect, and no decreased susceptibility to the Thatcher illusion was found in ASD (for a review, see Weigelt et al., 2012). More implicit measures, which are less prone to compensatory strategies, do find differences in face processing, contrary to most behavioral studies. For example, children with ASD fail to show the typical longer looking times (van der Geest, Kemner, Verbaten, & van Engeland, 2002) and the typical larger pupil dilation (Falck-Ytter, 2008) for upright than for inverted faces. Moreover, ERP studies demonstrated that the typical differential response to upright versus inverted faces is not present in adults with ASD (McPartland, Dawson, Webb, Panagiotides, & Carver, 2004; Webb et al., 2012). These findings all point to less efficient face processing, because selection and emphasis of predictive cues is missing, throwing face perception back to processes similar to those used for inverted faces. It also fits with HIPPEA that when global face processing deficits are found, they will disappear if participants with ASD are explicitly cued (e.g., "look at the eyes"), as shown by B. Lopez, Donnelly, Hadwin, and Leekam (2004).

Studies finding disturbed formation of face prototypes in ASD may also confirm our account (Gastgeb, Rump, Best, Minshew, & Strauss, 2009; Gastgeb, Wilkinson, Minshew, & Strauss, 2011). Typically, forming a face prototype requires the use of the central tendency in all encountered exemplar-faces to arrive at an implicit, average representation ignoring the within-category variability (Valentine, 1991). In ASD, however, the emergence of a familiar, broad face prototype will not occur automatically. For categorization to work, new instances have to be recognized as similar to previously experienced examples. The chronically high precision of prediction errors will impede this ability by overemphasizing the extent to which new input deviates from previous examples or learned templates. Consistently, Plaisted et al. (1998) found that high-functioning adults with ASD learned to discriminate between configurations of colored disks to higher levels of accuracy than controls, but when tested with slightly dif-

ferent exemplars of the same overall configurations, normal controls showed transfer from the learned exemplars to the novel ones, while individuals with ASD did not. As a result, individuals with ASD may be slower at categorization learning (e.g., Klinger & Dawson, 2001; Soulières et al., 2011) and they may be less spontaneously extracting a prototype from a series of exemplars (e.g., Gastgeb, Dundas, Minshew, & Strauss, 2012; Vladusich, Olu-Lafe, Kim, Tager-Flusberg, & Grossberg, 2010).

Finally, impaired formation of a familiar, broad face prototype can also be seen in the reduced face adaption aftereffects (Pellicano, Jeffery, Burr, & Rhodes, 2007; Rutherford, Troubridge, & Walsh, 2012, e.g.,). Though these findings may mean that perception is less influenced by prior knowledge (in this case the shifted prototype) (Pellicano & Burr, 2012), we would propose that it is the consequence of an abnormal updating of representations (prototypes). An adapting exemplar may not update the main prototype, because it contains important enough differences for individuals with ASD, to deserve creation of a novel, narrow prototype. Future studies of lower-level feature adaptation, currently lacking in ASD, may be able to resolve this debate.

Mismatch negativity. While the predictive coding account has originally been conceptualized in the visual domain, a growing number of studies has also investigated predictive coding phenomena in the auditory modality (Arnal & Giraud, 2012; Winkler, Denham, & Nelken, 2009). In this regard, auditory mismatch negativity (MMN) research has been particularly informative. Here, presentation of an unexpected oddball stimulus within a sequence of repeated predictable stimuli, elicits a novelty response in the event-related potential. Originally, the MMN was interpreted as reflecting change detection on the basis of a passive bottom-up process of adaptation to the repeated stimuli (May & Tiitinen, 2010). Recent evidence, however, has shown that the MMN does not reflect release of repetition suppression, but is the result of a violated prediction rather than a physical stimulus change (e.g., Todorovic, van Ede, Maris, & de Lange, 2011; Wacongne, Changeux, & Dehaene, 2012). A series of studies further made plausible that the perceptual cortex indeed implements a hierarchy of predictions and prediction errors, with repetition suppression attenuating neural responses in a very early time window (40-60 ms), stimulus expectation on the basis of unconscious local predictions attenuating the intermediate stage of processing (100-200 ms, i.e., the typical MMN which originates in sensory areas), and stimulus expectations on the basis of more global, integrative and conscious predictions modulating the later P3b novelty response (300-600 ms, originating from a broader frontoparietal predictive network) (Todorovic & de Lange, 2012; Wacongne et al., 2011). Regarding the MMN, a number of studies observed larger amplitudes and/or earlier latencies to infrequent pitch changes in tones and vowels in ASD relative to TD controls, thus suggestive of hypersensitivity and superior recognition of pitch change (e.g., Ferri et al., 2003; Gomot, Giard, Adrien, Barthelemy, & Bruneau, 2002; Lepisto et al., 2005 but see Dunn, Gomes, & Gravel, 2008). Interestingly, Gomot et al. (2011) showed that these electrophysiological abnormalities were significantly more pronounced in children who displayed greater difficulties in tolerating change. The MMN response to infrequent phonemic changes in vowels or consonants, however, is typically smaller and/or delayed in ASD, thus suggestive of impaired recognition of the more global phonetic characteristics of speech (e.g., Kujala, Lepisto, Nieminen-von, Naatanen, & Naatanen, 2005; Lepisto et al., 2006). Finally, the later P3b component, presumably characterizing more global and integrative violations of expectations, exhibits smaller amplitudes in ASD relative to controls (e.g., G. Dawson, Finley, Phillips, Galpert, & Lewy, 1988; Kemner, Verbaten, Cuperus, Camfferman, & Van Engeland, 1995). Comparing neurophysiological findings pertaining to MMN versus P3b processing suggests that the brains of individuals with ASD are tuned to register low-level local changes in transition probabilities (cf. enhanced and earlier MMN sensory responses towards simple stimuli), but have difficulty picking up changes in the broader fronto-parietal predictive system which is tuned towards more global, higher-level patterns. This is at least compatible with the view that increased low-level precision hinders the formation of appropriate predictions higher up.

4.3 Savant skills

"Autistic savants" are individuals with ASD with co-occurring excellence in an isolated skill, i.e., an "island of genius" which contrasts with the individual's general lower-than-average abilities. Savantism has been identified in a wide range of neurological and neurodevelopmental disorders, but is most frequently reported in ASD. Savant skills are estimated to be present in one out of 10 autistic individuals, with males outnumbering females (approx. 6:1) (Howlin, Goode, Hutton, & Rutter, 2009; Treffert, 2009). Savant skills usually fall within one of five general categories, i.e, musical abilities, calendar calculating, mathematics, art and mechanical or spatial skills (Treffert, 2009). Although the savant skill of an individual may evolve over the years, the skill should not fade or disappear over time, but remain a peak in performance.

Several scholars attempted to explain the mechanism behind the savant skills. Plaisted (2001) suggested a reduced ability to process similarity at the perceptual and attentional level which results in a reduced tendency to generalize information. Baron-Cohen (2006) postulated an in-

creased drive to construct or analyze, which he referred to as "hyper-systemizing". The alleged adaptive function of the systemizing mechanism is to serve as a law-detector and a change-predicting mechanism. He argues that people with ASD prefer either no change, or systems which change in highly lawful or predictable ways (i.e, systems with rule-bound change, such as mathematics, physics, objects that spin or recur, music, machines, collections), and why they become disabled or "change-resistant" when faced with systems characterized by "complex" change (such as social interaction). Mottron et al. (2006) and Mottron et al. (2013) emphasized the putative role of enhanced feed-forward low-level perception and suggested that individuals with ASD have a developmental predisposition to "veridical mapping" of data and information. Although these accounts provide insight into the origin of such a skill, HIPPEA makes more specific claims about the underlying mechanisms.

Our predictive coding approach explains why similarity is not processed in the same way in ASD, consistent with Plaisted (2001). It also elucidates why complex change is challenging (Baron-Cohen, 2006): this is where meta-learning should lead to distinguishing mere noise from actual environmental changes. Finally, the veridical mapping can also be seen as a consequence of the constant drive to reduce even irrelevant prediction errors (Mottron et al., 2013). While predictions shaped by noise and irrelevant details will often result in impaired or slow processing, doing this for a specific, limited topic of interest can be quite possible and, above all, rewarding. Developing such a skill becomes extrinsically motivating (e.g., getting praise and attention) but also intrinsically, as making successful predictions in this particular domain will result in feelings of reward, and the notion that the generally unpredictable world is more controllable. For example, phone numbers have an exact but arbitrary mapping (Mottron et al., 2013). All known examples of savant skills, for instance, 3D drawings or musical play from memory, combine two factors: an exquisite discriminative sensory ability and an exceptional (rote) memory capacity (A. L. Hill, 1978; Treffert, 2009). The first is a general feature of ASD, we would argue, originating from high precision low-level prediction errors. A lack of abstraction is actually an advantage here. Clearly, this discriminative ability can only fully be put to use in the case of high memory capacity. This may be the feature that is specific to savants, but even then resource constraints may seriously limit the savant domain.

5 Sensorimotor abilities and a sense of self

Within the predictive coding theory, actions also entail predictions, namely of their proprioceptive and exteroceptive consequences. According to Friston, Daunizeau, Kilner, and Kiebel (2010), movement is defined by "what we want to see (or feel), rather than what we want to do". In this view, actions can be regarded as being aimed at fulfilling predictions (reducing prediction errors) of perceptual input. Several ASD symptoms can be readily interpreted from this perspective. Given that actions generate prediction errors, those actions that reduce these prediction errors to extreme minima should be preferred. Accordingly, some of the most characterizing symptoms in ASD are the stereotypical, repetitive (predictable) behavior patterns (Turner, 1999). These patterns establish controllable and thus very predictable proprioceptive (kinesthetic) feedback, that helps individuals with ASD to better cope with their environment (Ornitz, 1974). In a similar vein, the repetitive handling of lighting and spinning objects, and the repetitive tactile self-stimulation can be regarded as manners of creating a predictive environment to reduce and cope with prediction error. Especially unpredictable surroundings may be expected to elicit this kind of behavior, with the aim of reestablishing predictability and reducing stress (see Section 6). Ornitz (1974, p. 204) observes that "In their spontaneous activity autistic children are continually spirting, twirling, flicking, tapping, or rubbing objects. Furthermore, they repetitively flap, writhe, wiggle, or oscillate their extremities while regarding them intently." This latter part is significant because it indicates that while TD children might progress to more complex kinds of "play" (learning), children with ASD continue to be engaged in and learn from these simpler patterns.

According to HIPPEA, atypical behavior has the aim of regulating excessive amounts of prediction errors. At first sight, this seems very similar to the explanation invoked by the EPF theory, namely reducing excessive perceptual input (Mottron et al., 2006). However, in our view, individuals with ASD only aim to reduce that part of the perceptual input that cannot be predicted, and moreover actively attempt to create predictability to compensate. Interestingly, reports of autistic children screaming all day, despite being hypersensitive to noise themselves, might be understood as a way of dealing with prediction errors by making the sensory environment more predictable. The active desire for predictable sensory experience is brought even more clearly into light by Temple Grandin, an autistic woman who built a mechanic body squeeze machine, because she liked the feeling of being touched and hugged, but wanted it to be perfectly controlled (i.e., predictable) instead of the unpredictable overstimulating human touch (Edelson, Edelson, Kerr, & Grandin, 1999; Grandin, 1992). In a similar vein, the "high systemizing" concept used by Baron-Cohen, Ashwin, Ashwin, Tavassoli, and Chakrabarti (2009) to characterize the cognitive style of individuals with ASD can underscore that predictable patterns are formed and are important in their minds. The obsession with regularity can be seen as

borne of an overweighing of deviations.

The sense of self and of agency has also been related to (interoceptive) predictive coding (Apps & Tsakiris, 2013; Seth, Suzuki, & Critchley, 2012). It is through the tightly cross-modally correlated proprioceptive, tactile and visual input of self-induced movements that we construct the sense of a self that acts in the world. The high-level concept of the self is the most plausible prediction explaining low-level regularities in cross-modal input. This view of the emergence of the self via the observed correlations between proprioceptive, tactile and visual modalities can also explain why artificially created correlations can create the illusion that extra-corporeal objects are part of our own body (e.g., rubber hand illusion; Apps & Tsakiris, 2013; Botvinick & Cohen, 1998). Awareness of self and body as distinct from the world is thus dependent upon a certain degree of tolerance derived from the active, successful suppression of interoceptive prediction errors (Seth et al., 2012). The presence of repetitive, stereotyped movements in ASD during early development suggests that an abnormally large amount of correlated input is needed to establish a sense of self as separated from the surroundings (see also Brincker & Torres, 2013).

Two recent studies using the rubber hand illusion, an illusion of perceived arm position induced by correlated (synchronized) stimulation (Palmer et al., 2013; Paton, Hohwy, & Enticott, 2012), support this view. Both in individuals with ASD and in those with high but nonclinical ASD traits, the consequences of experiencing the illusion (on drift and movement) were reduced. A higher estimated precision of prediction errors may indeed lead to a reduced illusory percept, requiring more tightly correlated input (than is usually provided in this rubber hand procedures) for the illusory percept to fully establish itself. More generally, motor coordination problems, often noted in ASD (Fournier, Hass, Naik, Lodha, & Cauraugh, 2010; M. L. Matson, Matson, & Beighley, 2011), may be another consequence of over-precision of movement prediction errors in contexts that actually have a considerable amount of uncertainty (Palmer et al., 2013).

The observation that the repetitive, self-focused behaviors often decrease during development (Richler, Huerta, Bishop, & Lord, 2010) suggests that extensive exposure may eventually lead to a more stable sense of self. However, the typical "insistence on sameness" (Kanner, 1943) remains or increases with age, indicating that exteroceptive prediction errors generally remain precise. This insistence on routine or rituals, and resistance to trivial changes in the surroundings, again demonstrate that children with ASD do develop clear predictions on what should happen next in the current situation, in contrast to theories positing a uniformly weaker application of predictions in ASD (Pellicano & Burr, 2012). Therefore, insistence on sameness may be considered a hallmark of HIPPEA: It signals a clear grasp (prediction) on how the world should behave, while assigning too much importance to incidental changes.

6 Chronic unpredictability and its affective consequences

One of the most prominent clinical observations in individuals with ASD is their unusual reactivity to sensory stimuli. Numerous clinical and personal reports describe the presence of both hyper- and hyposensitivity to sensory stimulation. Hypersensitivity has been described in various modalities (Blakemore et al., 2006; Kern et al., 2006; Khalfa et al., 2004). Enhanced sensitivity to loud and unexpected sounds is particularly evident in children with ASD (e.g., Grandin, 1995; Tomchek & Dunn, 2007), and appears to decrease with age, with adults with ASD becoming more similar to TD adults (Kern et al., 2006). Yet, feelings of stimulus overload and hypersensitivity to noise are also common in adults on the autistic spectrum (in particular in social situations, like receptions or parties), and can cause great distress and anxiety. Enhanced sensitivity to visual stimuli is less common in ASD, but does occur, e.g., under the form of enhanced discomfort to bright light (Kern et al., 2001). When the gain of the neural units representing the prediction errors is fixed at a high level, it is easy to see that hyper-sensitivity becomes very likely, especially for unexpected input, as is the case in ASD. Overweighting of irrelevant prediction errors causes sensory overload.

Seeing that unpredictability is at the core of the sensory overload, we can also attempt to explain its negative affective impact. Uncertainty has long been identified as a factor that intensifies stress and anxiety (Herry et al., 2007; Miller, 1981). In addition to leading to increased stress and anxiety, persistent significant prediction errors may actually by themselves generate negative affect (Huron, 2006; Van de Cruys & Wagemans, 2011). When predictions are invoked, there is actually something at stake, namely the success of current internal models of the environment. When prediction errors signal the need for extra resources, aimed at updating the internal model, they may have negative affective value. For example, supposedly neutral perceptual prediction errors activate the habenula, a region known to code prediction errors of negative valence (Schiffer, Ahlheim, Wurm, & Schubotz, 2012; Schiffer & Schubotz, 2011). Originating from the cognitive dissonance tradition, recent frameworks in social psychology center precisely on the link between expectation violation (or uncertainty) and anxiety, with much of human cognition and behavior interpreted as efforts to reestablish a coherent, predictable world model (Hirsh, Mar, & Peterson, 2012; Proulx, Inzlicht, & Harmon-Jones, 2012).

The taxing, negative experience described in ASD as sensory overload or oversensitivity is, according to HIPPEA, a logical consequence of a brain continuously signaling that prediction errors merit the recruitment of more resources for learning. The proactive (predictive) investment of the system makes this a particularly aversive experience. Conversely, making progress in predicting the world (reducing prediction errors) may genuinely feel rewarding. Note that not the static state of low prediction error but rather the transition (change) from a state of high prediction errors to a state of low errors may induce positive affect (Joffily & Coricelli, 2013; Oudeyer et al., 2010; Van de Cruys & Wagemans, 2011). This kind of reward arguably is the driving force for further exploration and learning (cf. Section 3). However, difficulties in estimating where predictive progress can be made could largely rob a person from experiencing this type of reward, with detrimental implications for intrinsic motivation. Indeed, problems in general motivation and exploration are reported in ASD (Koegel & Mentis, 1985; Ozonoff et al., 2008), from very early on in development (Zwaigenbaum et al., 2005).

The combination of increased uncertainty-related anxiety and decreased reward of exploration may have particularly incapacitating and far-reaching effects in the longer term. We already referred to learned helplessness to indicate the anxious avoidance and lack of motivation caused by repeated frustration in predicting one's surroundings. By caregivers this may be interpreted as hypo-reactivity (Ben-Sasson et al., 2009; Tomchek & Dunn, 2007). Social interactions might suffer most from this lack of motivation (Chevallier, Grezes, Molesworth, Berthoz, & Happe, 2012), with obvious consequences with regard to the willingness to engage in social relations. We do not consider social motivation problems to be the origin of ASD, but our account agrees with social motivation theories (Chevallier et al., 2012) that this is an important aggravating factor in the syndrome. Indeed, social interactions are not perceived that enjoyable or rewarding in individuals with ASD (Chevallier et al., 2012). Unsurprisingly, a lot of interventions focus on increasing the reward of social interactions. If social situations are avoided from early on in life, the number of social learning experiences decreases, and so, in a vicious circle, even more social impairments ensue.

Taken together, these factors arguably make individuals with ASD more vulnerable to mood and anxiety problems, which are indeed overrepresented in ASD (Kim, Szatmari, Bryson, Streiner, & Wilson, 2000). Hence, mood problems, anxiety and anxious avoidance should in our view be considered as secondary symptoms, originating from accumulated experience with (irreducible) prediction errors, and from repeated frustration in learning. Consistent with this, anxiety and mood problems seem to increase during childhood in ASD (Kim et al., 2000).

7 Social functioning

Social interaction problems are amongst the first described symptoms of ASD (Asperger, 1944; Kanner, 1943) and are crucial pieces in the DSM-classification (American Psychiatric Association, 2013). Social impairments stand out strongly in the clinical phenotype (demonstrated by the existence of ASD questionnaires focusing merely on the social symptoms; e.g., Constantino, 2002), and retrospective studies often report early signals in the social domain (Volkmar, Chawarska, & Klin, 2005). The phenomenal and clinical prominence of social deficits spurred a wealth of evidence on social impairments. Therefore, a central challenge for core information processing dysfunction theories of ASD is to explain why abnormalities manifest themselves most clearly in the social domain.

What sets social situations apart from non-social situations? Or better: what distinguishes social tasks in the lab from the tasks used for other (lower-level) domains? Like Simmons et al. (2009), we wonder whether *social* may just be a synonym of *complex* here. However, our approach allows us to pinpoint exactly what this complexity may entail with regard to the difficulties in ASD. Most ingredients have been provided in the previous sections, but in the social domain they come together and are expressed to the fullest.

7.1 Social complexity

Our brief overview of face and speech processing impairments in ASD did not strongly speak for a special status of faces or speech as such. Here too, we do not want to treat social judgements differently from other processing. It is more fitting, we argue, to view them as just another kind of inference, in this case inference about other people's emotions or intentions from their facial expressions, gaze, bodily postures, etc. (Hohwy & Palmer, 2014; Zaki, 2013). Therefore, the same mix of accidental uncertainty and informative changes determines the social problems in ASD. No two social scenarios are identical. Numerous accidental properties in the rich social environment are mostly uninformative and should be ignored. This is ideally what tuning down precision should accomplish. Individuals should (meta-)learn which aspects are informative and which are irrelevant to the social rules governing the current situation. This is particularly difficult when these noisy social contingencies are changing and context-bound, which they mostly are (Barrett, Mesquita, & Gendron, 2011). There is rarely a one-to-one mapping between social signals and their meaning. For example, happiness can be expressed with an obvious loud laughter, but an enigmatic Mona Lisa smile is possible too. A similar laugh can signify consent (humor) or rejection (irony). Subcultures (e.g., youth culture) invent new

meanings for old signals (e.g., words) or new signals for old meanings. In addition, low-level input can be dramatically different while the same social rules apply. Instead of flexibly adjusting the precision of prediction errors based on previous and current experiences, individuals with ASD will get flooded by the wealth of available information in a social situation.

Generalizing what we said about face perception, people with ASD fail to discriminate between informative and irrelevant properties when making social judgments (cf. the lack of autonomous selection in attention). The result is that social information does not seem to be particularly salient for them, or at least not more so than non-social stimuli. This deficit is most clearly illustrated by eye-movement studies. Individuals with ASD show a reduced attention to faces, but more attention towards bodies and objects in the background of a social scene (e.g., Klin, Jones, Schultz, Volkmar, & Cohen, 2002; Rice, Moriuchi, Jones, & Klin, 2012). Within faces too, differences in information selection are noticeable. They also do not seem to have learned the typical informativeness of the eyes region, crucial for face and emotion recognition. Instead, studies reveal a bias for the mouth region and scanning patterns towards the outer face characteristics (such as hair; Harms, Martin, & Wallace, 2010). From early childhood on, children with ASD do not show the usual preference for social stimuli (Klin, 1991, 1992). Two year old children with ASD rather attend to non-social physical contingencies instead of socially relevant biological motion (Klin, Lin, Gorrindo, Ramsay, & Jones, 2009). We think this should be explained by the steadier, lower-level predictability of the former.

The fact that the atypical viewing patterns and the emotion recognition deficits are most apparent when using complex stimulus material (Chevallier et al., 2012; Harms et al., 2010) also speaks for our hypothesis. While the distinction between relevant and irrelevant information may be rather clear-cut in simple social stimuli (e.g., isolated, well-controlled and re-used faces), using ecologically valid stimuli (e.g., noisy, dynamic social scenes) implies more competition from distracting (irrelevant) information.

Gradually, TD children form "social scripts": abstracted and broadly applicable knowledge structures, representing an organized sequence of actions, causes and consequences within a certain social context (e.g., making friends). In children with ASD this capacity to generate adequate social scripts is found to be impaired (Loth, Happe, & Gomez, 2010). It is easy to see that indiscriminate precision of social and non-social cues results in narrow and specific social scripts (e.g., making friends *when* I'm playing soccer), wrought with spurious, concrete features. Interventions that try to remedy social script deficits, select and describe the relevant cues for a given script, linking it with possible appropriate responses (for a meta-analysis, see Reynhout & Carter, 2011).

7.2 Multisensory integration

Adequate social understanding heavily relies on integration of multiple sources of information, both within modality and across modalities. The same facial expressions can receive completely opposite meanings depending on the bodily context in which they appear (Aviezer, Trope, & Todorov, 2012). In other situations different modalities provide complementary information, to be used to figure out emotions and intentions from face-to-face-communication. In such cases, additional information of another modality helps the interpretation. For instance, visual articulatory information aids speech perception, especially under noisy circumstances. Again we note that uncertainty of the different sources has to be taken into account in order to determine which information should have more saying in the eventual social judgment. Indeed, this can be formalized using Bayes' theorem (Zaki, 2013), which is already widely used in (non-social) perceptual cue integration studies. For optimal inference, the expected uncertainty (precision) of the different sensory sources should determine differential reliance (weight) on those sources.

Individuals with ASD are known to have difficulties with such multisensory integration (Iarocci & McDonald, 2006), for instance with the detection of inter-modal correspondence of facial and vocal affect (e.g., Loveland et al., 1995). If precision is fixed at a similarly high level for all sources, as HIPPEA maintains, optimal integration will not take place, because all cues, even redundant or very uncertain ones, will be weighed equally. Moreover, the spatiotemporal contiguity of two inputs required to perceive them as belonging to the same distal cause would be more strictly defined for people with high precision. Any minor spatiotemporal mismatch between two cues (e.g. visual-auditory in the ventriloquist effect or visual-haptic in the rubber hand illusion) will render it more likely that these will be experienced as distinct unimodal events rather than an integrated, multimodal event (Palmer et al., 2013). The attenuated McGurk effect found in ASD could similarly be explained (Mongillo et al., 2008; Taylor, Isaac, & Milne, 2010).

7.3 Mentalizing

"Theory of Mind" or "mentalizing" refers to the ability to read the (facial) expressions of other people, to understand their feelings, intentions, wishes and thoughts, and to use this —mostly implicit— knowledge to understand another individual's actions and guide one's own actions (Premack & Woodruff, 1978). A vast amount of research

in ASD has focused on the Theory-of-Mind problems in individuals with ASD, arguing that individuals with ASD have difficulties in placing themselves into the mental world of others and themselves, and sometimes described as "mindblindness" (e.g., Baron-Cohen, 2001; Frith & Frith, 2003). The discovery of mirror neurons (in monkeys) that are active both during the action observation or imagination (offline processing), but also during the online execution of an action (e.g., Kohler et al., 2002), led to the conjecture that action-understanding and even mentalizing crucially rely on this class of neurons. All discussions on the precise role and distribution of mirror neurons in the brain aside, this finding conclusively showed that action execution and action perception are closely intertwined. Predictive coding offers a new perspective on the implementation of goal and intention inference in the mirror system (e.g., Friston, Lawson, & Frith, 2013; Kilner, Friston, & Frith, 2007; Koster-Hale & Saxe, 2013). As mentioned before, actions could be conceived of as a series of hierarchical predictions (Hamilton, Brindley, & Frith, 2007), going from longer-term intentions and goals (e.g., to splash water in your friends' face) over short-term goals (e.g., to grasp a glass of water) and motor plans (movement sequences), down to the muscle commands and kinesthetics. At all levels, predictions will be matched with input, resulting in prediction errors that drive and guide proper action execution. Importantly, in predictive coding this same hierarchical model that is used for forward action generation also serves inverse inference: figuring out goals from observed actions (Kilner et al., 2007). Observed actions will both automatically generate expectations on the kinematics and muscle activation linked to it, and create discrepancies that can only be explained away by inferring an appropriate intention on the highest levels. How can this system distinguish then between own actions and another's actions? Put differently, observed action creates prediction errors because motor plans and goals are generated, while muscle and kinesthetics are inactive. How does the brain avoid automatically executing (mimicking) observed actions to reduce those low-level prediction errors?

The assumed mechanism is, again, precision (Clark, 2013a). The prediction errors have a high expected precision, which makes sure actions you initiate yourself are properly executed. These prediction errors will be suppressed by your own accurate predictions (goals), often inciting a sense of confidence or agency (see Section 5). For action observation however, estimated precision of motor prediction errors should be tuned down, such that they receive low weight and the thrust of processing moves to higher-level inference of goals and intentions. In this way, precision becomes the mechanism that allows organisms to exploit the learned hierarchical models for action execution, also for mentalizing and offline planning (Clark, 2013a).

Following this reasoning, a deficit in the flexible tuning of precision of prediction errors, resulting in an overly high estimation of precision, as HIPPEA assumes to be the case in ASD, may give rise to a couple of related problems. First, it may contribute to offline (motor) planning problems (Booth, Charlton, Hughes, & Happé, 2003; Hughes, 1996), with high precision preventing individuals to transcend the immediate input, as noted earlier. Second, failure to lower the precision of low-level prediction errors during action observation, may automatically lead to precise proprioceptive prediction errors, because the action is not executed. A possible strategy to reduce these errors is the mimicking of (formal aspects of) others' behavior. Indeed, hyper-imitation of formal aspects of behavior (Bird, Leighton, Press, & Heyes, 2007; Spengler, Bird, & Brass, 2010), and echolalia and echopraxia, the automatic copying of others' speech or behavior occur more frequently in the ASD population. We are cautious in pointing to this possibility, because precision of motor errors may be determined by a different neurotransmitter (dopamine) than perceptual errors (see Section 8) and not every child with ASD shows this automatic mimicking.

A third possible problem of inflexible tuning of precision, links back to our discussion on visual and auditory perception. We noted there that top-levels of hierarchical models may not get properly build (learned), because processing is stuck in low-level matching due to the high precision of low-level prediction errors. If for motor execution and planning too, individuals with ASD end up with incomplete hierarchical models, they may be unable to reach the higher levels of conceptual inferences of goal and intention. Consequentially, these individuals will experience difficulties in inferring emotions from their *own* bodily states and expressions (Seth et al., 2012). Indeed, alexithymia is often found in ASD, and has recently been shown to better predict poor recognition of emotional expressions than ASD as such (Cook, Brewer, Shah, & Bird, 2013). From the predictive coding standpoint, where one model is used for both emotion recognition and inferring own emotion, this makes a lot of sense. Brain responses related to empathy are also modulated by alexithymia rather than ASD (Bird et al., 2010). If these findings are corroborated, it may turn out the empathy and emotion recognition problems in ASD (see Harms et al., 2010; Uljarevic & Hamilton, 2013, for a meta-analysis and literature review, resp.) are not primary symptoms, but are inherently linked to alexithymia. The processing profile of ASD as we sketched may predispose patients to alexithymia, because high precision interoceptive prediction errors prevents adequate emotional inferences (Seth et al., 2012).

8 Neurobiological underpinnings

In the current paper we primarily wanted to articulate the cognitive, computational foundation of our account and its behavioral consequences. We do want to briefly survey plausible neurobiological underpinnings of the proposed mechanism as well, without giving an exhaustive review of the neurobiology of ASD (e.g., see D. G. Amaral, Schumann, & Nordahl, 2008; Bauman & Kemper, 2005). Clearly, a more systematic, extensive discussion of ASD neurobiology in light of HIPPEA will be needed in the future.

Using HIPPEA, we can tentatively divide neurobiological findings in three parts: first, studies directly targeting the neural regulation of precision, second, studies on the neural basis of models of uncertainty and meta-learning that feed into regulation of precision, and third, downstream consequences of high precision for neural plasticity and connectivity. We will only consider the first two here and leave the last part for later work.

Precision regulation. In Friston's predictive coding model, precision is regulated by neuromodulators that control the gain of the units representing prediction errors (Friston, 2010). This gain determines the impact of prediction errors on units that encode the predictions. Neuromodulators such as acetylcholine (ACh) and norepinephrine (NE) are long known to influence attention and learning, so they are likely candidates for this role. In particular, the neuromodulator ACh is assumed to enhance precision of perceptual prediction errors (Friston, 2010). Indeed, a pharmacological agent that increases ACh availability in cholinergic synapses increases the event-related response to deviations of predictions (Moran et al., 2013) and attenuates the decrease in responses with repeated stimulation (repetition suppression). However, Yu and Dayan (2005) proposed a different, complementary role of ACh and NE, in which only expected uncertainty, linked to the known stochasticity (lack of reliability) of a predictive relationship, is coded by ACh. NE, on the other hand, tracks unexpected uncertainty, that is, the actual, important changes in the regularities governing the relationships in the world (Duzel & Guitart-Masip, 2013; Payzan-LeNestour, Dunne, Bossaerts, & O'Doherty, 2013). A context-dependent modulation of the balance between these two must ensure that learning is enabled when learning is due (for actual changes).

The findings on (nicotinic) cholinergic signaling in ASD are very inconclusive at this stage, but a few studies report abnormalities (Lam, Aman, & Arnold, 2006), including in the main source of ACh, the basal forebrain (Bauman & Kemper, 1994; Perry et al., 2001). Raised NE signalling in ASD is suggested by elevated blood plasma levels (Lam et al., 2006) and by a tonically high arousal system as shown by a tonically elevated heart rate in autistic children, with reduced phasic response (Kootz & Cohen, 1981). For pupil size the same pattern has been reported: increased tonic pupil size, and increased latency, smaller constriction amplitude and lower constriction velocity for the pupillary light reflex, compared to TD children (Anderson & Colombo, 2009; Fan, Miles, Takahashi, & Yao, 2009). This is noteworthy, because of the known coupling of pupil size with the NE system, more specifically with activity in the principal source of NE projections, the locus coeruleus (Rajkowski, Kubiak, & Aston-Jones, 1993). Finally, prenatal overstimulation of the β_2-adrenergic receptor by an agonist is associated with increased risk of ASD (Connors et al., 2005).

Hence, available evidence already seems to point to some loss in the dynamic range of ACh and NE neuromodulation, but direct tests await. Pharmacological studies applying an agent that increases central cholinergic signaling should verify whether the ERP or behavioral response to expectation violation is modulated similarly in individuals with and without ASD (cf. Moran et al., 2013). If cholinergic signaling is already at ceiling in ASD, an additional boost of this system may not make a difference. Alternatively, a cholinergic antagonist may, in ASD, lead to "normal" performance on tasks that benefit from disregarding smaller differences (on which ASD subjects are usually worse). With regard to NE, there may be considerable potential in measuring pupil dynamics in ASD. Nassar et al. (2012) demonstrated that learning dynamics can be tracked by pupil size measurements, suggesting that NE arousal systems indeed can regulate learning. Their predictive inference task required adjusting of precision (learning rate), because predictive relationships changed at certain points ("change points") in the course of the task, as explained before (see Section 4.1). Apparently, pupil diameter change is monotonically related to change point probability, where prediction errors should indeed receive high weight. Additionally, average pupil size reflects "uncertainty that arises after change points and signals the need for rapid learning" (Nassar et al., 2012, p. 1043). Recall that this uncertainty was called reducible. If ASD is linked to increased precision of prediction errors across the board, as HIPPEA maintains, this should be apparent both in average learning rate and pupil metrics in this sort of task.

Finally, there is evidence that these neuromodulators can act as metaplastic signals regulating the potential of synapses to undergo activity-dependent long-term potentiation (e.g., Inoue et al., 2013). This provides another link with precision as a meta-learning signal, that needs to be explored more. Indeed, several of the genetic mutations linked to ASD have an important role in the regulation of plasticity (e.g., Delorme et al., 2013; Ebert & Greenberg, 2013; Hutsler & Zhang, 2010). Relatedly, the valproic acid

rat model of ASD shows twice the amount of long term potentiation of controls (Markram & Markram, 2010).

Models of uncertainty. We emphasized before that precision of prediction errors does not appear out of the blue. The brain builds meta-models, predictions of prediction errors, to estimate precision. These meta-models are formally not that different from regular predictive models assumed to take place across the perceptual hierarchy. Arguably then, these meta-models may be represented also in a distributed manner across the cortex. However, there is evidence that some regions are more involved than others in the processing of uncertainty.

Two regions that are good candidates for this *and* that have recently attracted researchers' interest in ASD are the insula and the anterior cingulate cortex (ACC). Both are thought to be central parts of the so-called salience network, the circuit involved in responding to behaviorally important stimuli and in cognitive control. Indeed, we could replace the somewhat vague term "salience" with "precision", because *in se* they have similar intent, namely determining value or relevance of input for behavior and learning. The salience network is closely connected to the motor system, suggesting a role in generating exploratory actions (Rushworth, Behrens, Rudebeck, & Walton, 2007), as we discussed in Section 3 on exploration in ASD. Also, it is deemed to be crucial in judging whether to persist in or switch the current attentional set (Dosenbach et al., 2006). Evidently, models of uncertainty in input are vital in such decisions. Finally, the ACC innervates the locus coeruleus-NE system (Aston-Jones & Cohen, 2005), perhaps allowing it to modulate gain (precision) of prediction errors in the (sensory) cortex.

A recent study found hyperactivation in dorsal ACC in response to visual oddbal stimuli in ASD (i.e., infrequently presented, deviant stimulus) (Dichter, Felder, & Bodfish, 2009), consistent with the idea that expectation violations are more salient. In healthy subjects ACC activity is found for behaviorally relevant prediction errors (Ide, Shenoy, Yu, & Li, 2013; Metereau & Dreher, 2013). Others have found evidence that the cingulate cortex not only represents the prediction errors, but also performs the computations underlying the adaptive regulation of precision (D. R. Bach, Hulme, Penny, & Dolan, 2011; Behrens et al., 2007).

The insula too is known to be involved in prediction under uncertainty. Activity in ACC and insula is strongly coupled, and critically, this coupling is modulated by prediction errors (Limongi, Sutherland, Zhu, Young, & Habib, 2013). Using a gambling game, Preuschoff, Quartz, and Bossaerts (2008) showed that activity in the anterior insula can code that part of uncertainty that cannot be reduced, due to the stochasticity of the associations at hand, also called known or expected uncertainty. There is evidence from dynamic causal modeling analyses that anterior insula is the entry point of the salience network and drives ACC activity (Ham, Leff, Boissezon, Joffe, & Sharp, 2013; Limongi et al., 2013). If true, a possible hypothesis is that the insula corrects incoming prediction errors for known stochasticity and thereby helps ACC and further regions to properly attribute salience (precision) of the prediction errors. In any case, insula, ACC and possibly neighboring frontal regions may cooperate to dissect uncertainty, with the aim of estimating where predictive progress can be made and setting precision accordingly (attending to the right aspects of input) (see also Karlsson, Tervo, & Karpova, 2012). When, as is the case for ASD, there is abnormal connectivity and activity of ACC and insula (Di Martino et al., 2009; Uddin & Menon, 2009), this estimation process may go awry, leading to unadaptive and possibly chronically high precision. Much more work is needed because existing neuroimaging work in ASD mostly uses task contrasts (often using faces or other complex stimuli) that are hardly informative in relation to our proposal. Fortunately, the needed paradigms have already been applied in nonclinical participants.

9 Related approaches

Several important theoretical frameworks of ASD can be usefully compared to ours. Some have been emphasized in previous sections, but here we more closely look at those that were not discussed before and that are most akin to our theorizing, in postulating a broader information-processing account. In the second part of this section, we will address the question whether a unified account is possible at all, in view of the heterogeneous nature of ASD. We will close this section with a discussion of a recent theory of schizophrenia, which is closely related to our theory of ASD.

9.1 Other information processing accounts of ASD

A straightforward, Bayesian way to conceptualize problems in ASD could be to assume broader (high uncertainty) priors or predictions that therefore have a weaker influence on the outcome of perceptual inference. Indeed, this road has recently been taken by Pellicano and Burr (2012) in a thought-provoking article (for a related approach, see Gomot & Wicker, 2012). These authors argued that this may cause perceptual outcomes to remain closer to the perceptual input, minimally biased by top-down, prior knowledge, an idea that is consistent with the WCC theory. Hence, this account explains why individuals with ASD may be less susceptible to visual illusions that are caused by prior knowledge or contextual interactions (see Section 4.2 on perception). In other words it would, according to the authors, result in a more accurate or "real" perception.

In addition to spurring an interesting discussion (Brock, 2012; Friston et al., 2013; Teufel, Subramaniam, & Fletcher, 2013; Van de Cruys et al., 2013; van Boxtel & Lu, 2013), this stance has been criticized on theoretical and empirical grounds. Teufel et al. (2013) remind us that "a perceptual system that refines sensory information by prior knowledge provides a better estimate of real but hidden causes than perception that is based on the ambiguous sensory information on its own, because the former system exploits all the relevant information available." In this regard, broader priors would lead to less accurate perception because the actual input is always noisy and ambiguous. Even in the case of visual illusions, it is not "priors per se [that] render perception less accurate; rather, it is the application of the wrong prior that leads to the illusory percept" (Teufel et al., 2013). Furthermore, Brock (2012) notes that perception (the posterior) can move closer to perceptual input (likelihood) for two different reasons: either, as Pellicano and Burr (2012) argue, the prior is broader (higher uncertainty, lower precision) *or* the likelihood is sharper (lower uncertainty, higher precision). It should be clear that the proposal in HIPPEA is more akin the second option. Finally, there is evidence that individuals with ASD are very well capable of building precise expectations from experience (see Section 4.1). Indeed that may be the reason why they are so perturbed by information that deviates from this information. The problems, we argue, arise because these deviations receive too much salience. Instead of a lack of precision in predictions, there may be a heightened precision of prediction *errors* in ASD.

It is also interesting to distinguish the our view from approaches locating the core problem in ASD in a reduced signal to noise ratio in neural processing (Belmonte et al., 2004; Simmons et al., 2009). While increasing noise usually impairs psychophysical performance, it can improve detection under restricted conditions, a phenomenon called "stochastic resonance" (Goris, Wagemans, & Wichmann, 2008). Though speculative at this stage, increased internal noise in neural communication may in this way be able to explain both improved performance in a limited number of tasks and impaired performance on more complex, high-level tasks (Simmons et al., 2009). HIPPEA in contrast, does not necessarily assume increased internal noise in neural signaling, but rather a higher weighing of external and internal "noise" (accidental features), causing the system to attempt to capture this irrelevant, non-repeating noise. We believe that this view is more readily compatible with the broad range of behavioral peculiarities in ASD.

9.2 Unifying theories of ASD, in the face of its genetic and phenotypic heterogeneity

Several scholars have lamented the overgrowth of unifying theories on ASD, seeing that they fail to deliver a convincing account for every ASD symptom cluster. Heterogeneity in underlying genetics similarly seems to suggest that there is not one but rather a multitude of deficits underlying the ASD pathology (Happé & Booth, 2008). Finally, and most importantly, phenotypic variability is notorious in ASD (Rommelse, Geurts, Franke, Buitelaar, & Hartman, 2011). This causes, but may also be caused by, difficulties in diagnosing ASD. Questioning the view of ASD as singular entity even further is the fact that "virtually every symptom characteristic of ASD can be observed in children who do not fit this diagnostic category" (Bishop, 1989). This of course, does not necessarily imply that these symptoms when they appear together in ASD are just the result of the "worst of luck". Still, these observations have led Happé and Booth (2008) to describe ASD as a fractionable triad, with three independent components (communication problems, social interaction deficits, and repetitive and restricted behaviors and interests) coincidentally co-occurring. Only when the three conspire, subclinical signs become clinical symptoms meriting a diagnosis.

Naturally, we agree with Happé and Booth (2008) that a better characterization of the subcomponents of ASD is much needed, but an intrinsic coherence of the components may only shine through when the appropriate level of description has been found. As we progress towards more realistic models of the mind-brain, we may be able to formulate more fitting explanations of ASD within these broader models. HIPPEA can be considered a first step in that direction.

Furthermore, there may be more coherence in the ASD symptom clusters than these critical authors assume. For example, while executive functioning and attentional deficits may not be specific to ASD (cf. ADHD), the specific pattern of executive capacities impaired and intact may be distinguishable from other disorders, and may have a privileged relationship with social or emotional symptoms of ASD. It is no doubt a challenge to connect social and communicative symptoms to more basic processing differences, due to divergence in the pathways leading to such high-level dysfunctions and to possible compensatory mechanisms saving these capacities for others. Indeed, a truly developmental account like HIPPEA will predict quite some variability in the unfolding of clinical symptoms depending on interactions with the environment.

Finally, heterogeneity in underlying (epi)genetic and molecular paths towards the syndrome does not preclude the possibility that one main cognitive mechanism is impaired. There is little reason to expect a one-to-one map-

ping from cognitive processing to neurobiology. The previous section provided possible ways HIPPEA links up with neurobiological evidence.

9.3 ASD in relation to schizophrenia

Increasing evidence suggests ASD has common genetic risk factors and neuroanatomical overlap with schizophrenia (Carroll & Owen, 2009; Cheung et al., 2010; Serretti & Fabbri, 2013). Intriguingly, a recent theory of schizophrenia (Adams, Stephan, Brown, & Friston, 2013; Fletcher & Frith, 2009) invoked undue high precision of prediction errors to explain positive symptoms in schizophrenia (hallucinations and delusions). The authors proposed that high precision prediction errors cannot be reduced and are propagated to higher levels, where they induce radical updates of beliefs to somehow make sense of them. Hence, they result in the strange world views and delusions.

Briefly, it seems to us that inflexible, high precision prediction errors are a better fitting explanation for ASD than they are for schizophrenia. Overprecise prediction errors as a fundamental, indeed developmental, characteristic would be present from very early on in life. Hence, the relatively late onset of schizophrenia needs explaining. Also, overly high precise prediction errors arguably do not sufficiently explain the specific, improbable and utterly bizarre contents of delusional beliefs (Silverstein, 2013). Other things that may be important to consider are the specific level of origin of the prediction errors (conceptual or action vs perceptual prediction errors, Adams et al., 2013; Fletcher & Frith, 2009) and the subjective confidence level (precision) that top-down beliefs can take on (to explain their fervor).

While the cognitive commonality of schizophrenia and ASD may match their genetic and neuroanatomical overlap, it also highlights a central challenge for predictive coding theories of mental illnesses: if they want to provide more than over-accommodating just-so stories for mental disorders, these theories should be able to give good, constraining explanations for the cognitive and neural specificities of each disorder. Clearly more work is needed in this respect.

10 Conclusions

While one core deficit is unlikely to explain all heterogeneity in ASD, it is quite remarkable that our approach can accommodate a broad range of reported deficits and peculiarities. This also makes sense since meta-learning is central in development across domains. Meta-cognition, conceptualized as the ability to monitor and adaptively use uncertainty, is generally fragile, costly and only conclusively demonstrated in a few, cognitively higher developed species (Carruthers, 2008; J. D. Smith, 2009; J. D. Smith, Coutinho, Church, & Beran, 2013). Dysfunction of this capacity may impact higher-level functions such as emotion processing and social cognition, but it also has a pervasive effect on attention, cognitive control, perception and learning. Hence, HIPPEA is broader than earlier single-deficit accounts of ASD, because it is not linked to a certain symptom cluster. At the same time however, HIPPEA is more specific than those accounts, homing in on the disturbed mechanism.

Every existing neurocognitive theory is criticized for not being universal and not being specific for ASD. How does how HIPPEA fare on those accounts? First, does HIPPEA maintain that *every* individual with ASD shows inflexibly high precision of prediction errors (*universality*)? We argue that this is indeed the case, but leave room for two ways to arrive at this high precision: A direct, possibly neuromodulatory deficit in the precision mechanism, or a deficit in the extraction from experience of information that should be used to estimate precision (meta-learning). Second, does every individual with chronically high precision prediction errors suffer from ASD (*specificity*)? Again, we answer positively, but with the important qualification that HIPPEA is consistent with the existence of a spectrum of ASD traits. It distinguishes different perceptual, cognitive, emotional, and social processes according to the extent to which they can be affected by chronically high precision errors. This naturally leads to the notion of a "spectrum". Just how high and how fixed precision is, determines whether normal functioning is still possible. Indeed, some people may be able to turn their "deficit" into an asset in tasks that benefit greatly from their specific processing style (Gonzalez, Martin, Minshew, & Behrmann, 2013).

Evidence-based treatments and psycho-education for ASD that focus on early learning (such as applied behavioral analysis, Lovaas, 1987; Rogers & Vismara, 2008), could take inspiration from HIPPEA that also has learning at its core but demarcates the circumstances under which problems in ASD arise. Animal models of ASD-related diseases show that environmental enrichment can reduce risk of developmental disorders (G. Dawson, 2008). We also remarked that people with ASD may be able to learn and use high-level predictions, given extensive exposure to more and different situations. However, most of all, our approach reaffirms the importance of more scaffolding during learning (e.g., Bellon, Ogletree, & Harn, 2000; Odom et al., 2003). Our Section 3 on exploration made it clear that children with ASD need more support with the gradual progression from simple to naturalistic stimuli (e.g., using virtual environments), taking into account uncertainty *and* its causes. Finally, and slightly counter-intuitively, reducing intense concentration on learning experiences, preventing subjects from trying to match all details ("early stopping"), has also been proposed to be ben-

eficial (Bakouie et al., 2009).

Although we consider HIPPEA a rich and promising theory, much of what we have offered here is post-hoc. The specific theory of ASD we proposed in this paper is based on predictive coding in normal functioning, but so far most of the explanatory power is in our selective (albeit broad) synthesis of the literature on ASD. Future research will have to corroborate its unique predictive power. In the preceding sections, we have often added comments about shortcomings in the current literature as well as specific hypotheses derived from our theory that remain to be tested. With a very general theory like predictive coding, there is always a risk of non-falsifiability (see also the extensive discussion sparked by Clark, 2013b), but we are convinced that our theory of predictive coding in ASD is specific enough to be testable. Although we mainly addressed the functional (psychological) level in this paper, we are optimistic that HIPPEA is at least compatible with an explanation at the neural level. We hope the progress that is currently being made in filling in the neural mechanisms behind predictive coding will help answer the question of precisely why individuals with ASD end up with high, inflexible precision.

In sum, our intent with this paper was to sketch the breadth of implications of HIPPEA with regard to aberrant development, and to point to new empirical questions for ASD research flowing from this view. Ultimately this will give us a better handle on ASD, connecting clinical to neurobiological descriptions and providing a firmer foundation for treatment.

Acknowledgements

Supported by a Methusalem grant by the Flemish Government (METH/08/02) to JW. We are incredibly grateful to John Brock, Floris de Lange, Vebjörn Ekroll, Marie Gomot, Jakob Hohwy, Ilona Kovacs, Laurent Mottron, Colin Palmer, Jean Steyaert, Jeroen van Boxtel, Peter van der Helm and Raymond van Ee for support and invaluable feedback on an earlier draft of this paper, and to our colleagues from LAuRes for many interdisciplinary discussions.

References

Adams, R. A., Stephan, K. E., Brown, H. R., & Friston, K. J. (2013). The computational anatomy of psychosis. *Frontiers in Schizophrenia*, *4*, 47. Retrieved from http://www.frontiersin.org/Schizophrenia/10.3389/fpsyt.2013.00047/full doi: 10.3389/fpsyt.2013.00047

Alderson-Day, B., & McGonigle-Chalmers, M. (2011). Is it a bird? is it a plane? category use in problem-solving in children with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *41*(5), 555–565. doi: 10.1007/s10803-010-1077-9

Amaral, D. G., Schumann, C. M., & Nordahl, C. W. (2008). Neuroanatomy of autism. *Trends in Neurosciences*, *31*(3), 137–145. Retrieved from http://www.cell.com/trends/neurosciences/abstract/S0166-2236(08)00038-6 doi: 10.1016/j.tins.2007.12.005

Amaral, J. L., Collins, S., Bohache, K. T., & Kloos, H. (2012). Beyond the Black-and-White of autism: How cognitive performance varies with context. In H. Kloos (Ed.), *Current topics in children's learning and cognition.* InTech. Retrieved from http://www.intechopen.com/books/current-topics-in-children-s-learning-and-cognition/beyond-the-black-and-white-of-autism-how-cognitive-performance-varies-with-context

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-5.* Arlington, Va.: American Psychiatric Association.

Anderson, C. J., & Colombo, J. (2009). Larger tonic pupil size in young children with autism spectrum disorder. *Developmental Psychobiology*, *51*(2), 207–211. (PMID: 18988196) doi: 10.1002/dev.20352

Apps, M. A. J., & Tsakiris, M. (2013). The free-energy self: A predictive coding account of self-recognition. *Neuroscience & Biobehavioral Reviews*. (PMID: 23416066) doi: 10.1016/j.neubiorev.2013.01.029

Arnal, L. H., & Giraud, A. L. (2012). Cortical oscillations and sensory predictions. *Trends in Cognitive Sciences*, *16*(1879-307X (Electronic)), 390–398. Retrieved from PM:22682813 doi: 10.1016/j.tics.2012.05.003

Ashwin, E., Ashwin, C., Rhydderch, D., Howells, J., & Baron-Cohen, S. (2009). Eagle-eyed visual acuity: an experimental investigation of enhanced perception in autism. *Biological psychiatry*, *65*(1), 17–21. (PMID: 18649873) doi: 10.1016/j.biopsych.2008.06.012

Asperger, H. (1944). Die "autistischen psychopathen" im kindesalter. *Archiv fur Psychiatrie und Nervenkrakheiten*, *117*, 76–136.

Aston-Jones, G., & Cohen, J. D. (2005). An integrative theory of locus Coeruleus-Norepinephrine function: Adaptive gain and optimal performance. *Annual Review of Neuroscience*, *28*(1), 403–450. Retrieved from http://www.annualreviews.org/doi/abs/10.1146/annurev.neuro.28.061604.135709 (PMID: 16022602) doi: 10.1146/annurev.neuro.28.061604.135709

Aviezer, H., Trope, Y., & Todorov, A. (2012). Body cues, not facial expressions, discriminate between intense positive and negative emotions. *Science*, *338*(6111), 1225–1229. Retrieved from http://www.sciencemag.org/content/338/6111/1225 doi: 10.1126/science.1224313

Bach, D. R., Hulme, O., Penny, W. D., & Dolan, R. J. (2011). The known unknowns: Neural representation of Second-Order uncertainty, and ambiguity. *Journal of Neuroscience*, *31*(13), 4811–4820. Retrieved from http://www.jneurosci.org/cgi/doi/10.1523/JNEUROSCI.1452-10.2011 doi: 10.1523/JNEUROSCI.1452-10.2011

Bach, M., & Dakin, S. C. (2009). Regarding "Eagle-Eyed vi-

sual acuity: An experimental investigation of enhanced perception in autism". *Biological Psychiatry*, *66*(10), e19–e20. Retrieved from http://www.sciencedirect.com/science/article/pii/S0006322309006258 doi: 10.1016/j.biopsych.2009.02.035

Baird, G., Simonoff, E., Pickles, A., Chandler, S., Loucas, T., Meldrum, D., & Charman, T. (2006). Prevalence of disorders of the autism spectrum in a population cohort of children in south thames: the special needs and autism project (SNAP). *Lancet*, *368*(9531), 210–215. (WOS:000239095000032) doi: 10.1016/S0140-6736(06)69041-7

Bakouie, F., Zendehrouh, S., & Gharibzadeh, S. (2009). Does a kind of over-fitting occur in the brain of autistic patients? *Journal of Neuropsychiatry and Clinical Neurosciences*, *21*(3), 343. (PMID: 19776319) doi: 10.1176/appi.neuropsych.21.3.343

Bar, M. (2009). The proactive brain: memory for predictions. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *364*(1521), 1235–1243. Retrieved from http://rstb.royalsocietypublishing.org/content/364/1521/1235 doi: 10.1098/rstb.2008.0310

Baron-Cohen, S. (2001). Theory of mind and autism: A review. *International Review of Research in Mental Retardation, Vol 23*, *23*, 169–184. Retrieved from ://000165696200007

Baron-Cohen, S. (2006). The hyper-systemizing, assortative mating theory of autism. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *30*(5), 865–872. Retrieved from http://www.sciencedirect.com/science/article/pii/S027858460600011X doi: 10.1016/j.pnpbp.2006.01.010

Baron-Cohen, S., Ashwin, E., Ashwin, C., Tavassoli, T., & Chakrabarti, B. (2009). Talent in autism: hyper-systemizing, hyper-attention to detail and sensory hypersensitivity. *Philosophical Transactions of the Royal Society London B Biological Sciences*, *364*(1471-2970 (Electronic)), 1377–1383. Retrieved from PM:19528020 doi: 10.1098/rstb.2008.0337

Baron-Cohen, S., Ring, H., Bullmore, E., Wheelwright, S., Ashwin, C., & Williams, S. (2000). The amygdala theory of autism. *Neuroscience & Biobehavioral Reviews*, *24*(3), 355–364. Retrieved from http://www.sciencedirect.com/science/article/pii/S0149763400000117 doi: 10.1016/S0149-7634(00)00011-7

Barrett, L. F., Mesquita, B., & Gendron, M. (2011). Context in emotion perception. *Current Directions in Psychological Science*, *20*(5), 286–290. Retrieved from http://cdp.sagepub.com/content/20/5/286 doi: 10.1177/0963721411422522

Bauman, M. L., & Kemper, T. L. (1994). Neuroanatomic observations of the brain in autism. *The neurobiology of autism*, 119–145.

Bauman, M. L., & Kemper, T. L. (2005). *The neurobiology of autism*. JHU Press.

Behrens, T. E. J., Woolrich, M. W., Walton, M. E., & Rushworth, M. F. S. (2007). Learning the value of information in an uncertain world. *Nature Neuroscience*, *10*(9), 1214–1221. (PMID: 17676057) doi: 10.1038/nn1954

Behrmann, M., Thomas, C., & Humphreys, K. (2006). Seeing it differently: visual processing in autism. *Trends in Cognitive Sciences*, *10*(6), 258–264. doi: 10.1016/j.tics.2006.05.001

Bellon, M. L., Ogletree, B. T., & Harn, W. E. (2000). Repeated storybook reading as a language intervention for children with autism: A case study on the application of scaffolding. *Focus on Autism and Other Developmental Disabilities*, *15*(1), 52–58. Retrieved from https://stuiterproxy.kuleuven.be/content/15/1/,DanaInfo=foa.sagepub.com+52.short doi: 10.1177/108835760001500107

Belmonte, M. K., Cook, E. H., Anderson, G. M., Rubenstein, J. L. R., Greenough, W. T., Beckel-Mitchener, A., ... Tierney, E. (2004). Autism as a disorder of neural information processing: directions for research and targets for therapy*. *Molecular Psychiatry*, *9*(7), 646–663. Retrieved from http://www.nature.com/mp/journal/v9/n7/abs/4001499a.html doi: 10.1038/sj.mp.4001499

Ben-Sasson, A., Hen, L., Fluss, R., Cermak, S., Engel-Yeger, B., & Gal, E. (2009). A meta-analysis of sensory modulation symptoms in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *39*(0162-3257 (Print)), 1–11. Retrieved from PM:18512135 doi: 10.1007/s10803-008-0593-3

Berlyne, D. E. (1966). Curiosity and exploration. *Science*, *153*(3731), 25–33. Retrieved from http://www.sciencemag.org/content/153/3731/25 doi: 10.1126/science.153.3731.25

Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2003). Motion perception in autism: A "Complex" issue. *Journal of Cognitive Neuroscience*, *15*(2), 218–225. Retrieved from http://dx.doi.org/10.1162/089892903321208150 doi: 10.1162/089892903321208150

Bertone, A., Mottron, L., Jelenic, P., & Faubert, J. (2005). Enhanced and diminished visuo-spatial information processing in autism depends on stimulus complexity. *Brain*, *128*(10), 2430–2441. Retrieved from http://brain.oxfordjournals.org/content/128/10/2430 (PMID: 15958508) doi: 10.1093/brain/awh561

Bird, G., Leighton, J., Press, C., & Heyes, C. (2007). Intact automatic imitation of human and robot actions in autism spectrum disorders. *Proceedings of the Royal Society B-Biological Sciences*, *274*(1628), 3027–3031. (WOS:000251278900015) doi: 10.1098/rspb.2007.1019

Bird, G., Silani, G., Brindley, R., White, S., Frith, U., & Singer, T. (2010). Empathic brain responses in insula are modulated by levels of alexithymia but not autism. *Brain*, *133*(5), 1515–1525. Retrieved from http://brain.oxfordjournals.org/content/133/5/1515 (PMID: 20371509) doi: 10.1093/brain/awq060

Bishop, D. V. (1989). Autism, asperger's syndrome and semantic-pragmatic disorder: where are the boundaries? *The British journal of Disorders of Communication*, *24*(2), 107–121. (PMID: 2690915)

Blakemore, S., Tavassoli, T., Calo, S., Thomas, R., Catmur, C., Frith, U., & Haggard, P. (2006). Tactile sensitivity in asperger syndrome. *Brain and Cognition*, *61*(0278-2626 (Print)), 5–13. Retrieved from PM:16500009 doi: 10.1016/j.bandc.2005.12.013

Boll, S., Gamer, M., Gluth, S., Finsterbusch, J., & Büchel, C. (2013). Separate amygdala subregions signal surprise and predictiveness during associative fear learning in humans. *European Journal of Neuroscience*, *37*(5), 758–767. Retrieved from http://doi.wiley.com/10.1111/ejn.12094 doi: 10.1111/ejn.12094

Bölte, S., Holtmann, M., Poustka, F., Scheurich, A., & Schmidt, L. (2007). Gestalt perception and Local-Global processing in High-Functioning autism. *Journal of Autism and Developmental Disorders*, *37*(8), 1493–1504. Retrieved from http://link.springer.com/article/10.1007/s10803-006-0231-x doi: 10.1007/s10803-006-0231-x

Bölte, S., Hubl, D., Dierks, T., Holtmann, M., & Poustka, F. (2008). An fMRI-study of locally oriented perception in autism: Altered early visual processing of the block design test. *Journal of Neural Transmission*, *115*(3), 545–552. Retrieved from http://www.springerlink.com/content/p5115748m1u55547/ doi: 10.1007/s00702-007-0850-1

Bölte, S., Schlitt, S., Gapp, V., Hainz, D., Schirman, S., Poustka, F., ... Walter, H. (2012). A close eye on the eagle-eyed visual acuity hypothesis of autism. *Journal of Autism and Developmental Disorders*, *42*(5), 726–733. (PMID: 21660498) doi: 10.1007/s10803-011-1300-3

Bonnel, A., McAdams, S., Smith, B., Berthiaume, C., Bertone, A., Ciocca, V., ... Mottron, L. (2010). Enhanced pure-tone pitch discrimination among persons with autism but not asperger syndrome. *Neuropsychologia*, *48*(9), 2465–2475. Retrieved from http://www.sciencedirect.com/science/article/pii/S0028393210001661 doi: 10.1016/j.neuropsychologia.2010.04.020

Bonnel, A., Mottron, L., Peretz, I., Trudel, M., Gallun, E., & Bonnel, A. (2003). Enhanced pitch sensitivity in individuals with autism: A signal detection analysis. *Journal of Cognitive Neuroscience*, *15*(2), 226–235. Retrieved from http://dx.doi.org/10.1162/089892903321208169 doi: 10.1162/089892903321208169

Booth, R., Charlton, R., Hughes, C., & Happé, F. (2003). Disentangling weak coherence and executive dysfunction: planning drawing in autism and attention–deficit/hyperactivity disorder. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *358*(1430), 387–392. Retrieved from http://rstb.royalsocietypublishing.org/content/358/1430/387 doi: 10.1098/rstb.2002.1204

Botvinick, M., & Cohen, J. (1998). Rubber hands 'feel' touch that eyes see. *Nature*, *391*(6669), 756. (PMID: 9486643) doi: 10.1038/35784

Brincker, M., & Torres, E. B. (2013). Noise from the periphery in autism. *Frontiers in Integrative Neuroscience*, *7*. Retrieved from http://www.frontiersin.org/Integrative_Neuroscience/10.3389/fnint.2013.00034/full doi: 10.3389/fnint.2013.00034

Brock, J. (2012). Alternative bayesian accounts of autistic perception: comment on pellicano and burr. *Trends in Cognitive Sciences*, *16*(12), 573–574; author reply 574-575. (PMID: 23123383) doi: 10.1016/j.tics.2012.10.005

Brown, C., Gruber, T., Boucher, J., Rippon, G., & Brock, J. (2005). Gamma abnormalities during perception of illusory figures in autism. *Cortex*, *41*(3), 364–376. (PMID: 15871601)

Brown, J., Aczel, B., Jiménez, L., Kaufman, S. B., & Grant, K. P. (2010). Intact implicit learning in autism spectrum conditions. *Quarterly Journal of Experimental Psychology*, *63*(9), 1789–1812. (PMID: 20204919) doi: 10.1080/17470210903536910

Burack, J. A. (1994). Selective attention deficits in persons with autism: preliminary evidence of an inefficient attentional lens. *Journal of Abnormal Psychology*, *103*(3), 535–543. (PMID: 7930053)

Carroll, L. S., & Owen, M. J. (2009). Genetic overlap between autism, schizophrenia and bipolar disorder. *Genome Medicine*, *1*(10), 1–7. Retrieved from http://link.springer.com/article/10.1186/gm102 doi: 10.1186/gm102

Carruthers, P. (2008). Meta-cognition in animals: A skeptical look. *Mind & Language*, *23*(1), 58–89. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/j.1468-0017.2007.00329.x/abstract doi: 10.1111/j.1468-0017.2007.00329.x

Cheung, C., Yu, K., Fung, G., Leung, M., Wong, C., Li, Q., ... McAlonan, G. (2010). Autistic disorders and schizophrenia: Related or remote? an anatomical likelihood estimation. *PLOS ONE*, *5*(8), e12233. Retrieved from http://dx.doi.org/10.1371/journal.pone.0012233 doi: 10.1371/journal.pone.0012233

Chevallier, C., Grezes, J., Molesworth, C., Berthoz, S., & Happe, F. (2012). Brief report: Selective social anhedonia in high functioning autism. *Journal of Autism and Developmental Disorders*, *42*(7), 1504–1509. (WOS:000305231000022) doi: 10.1007/s10803-011-1364-0

Clark, A. (2013a). The many faces of precision. *Frontiers in Theoretical and Philosophical Psychology*, *4*:, 270. Retrieved from http://www.frontiersin.org/Theoretical_and_Philosophical_Psychology/10.3389/fpsyg.2013.00270/abstract doi: 10.3389/fpsyg.2013.00270

Clark, A. (2013b). Whatever next? predictive brains, situated agents, and the future of cognitive science. *Behavioral and Brain Sciences*, *36*(03), 181–204. doi: 10.1017/S0140525X12000477

Connors, S. L., Crowell, D. E., Eberhart, C. G., Copeland, J., Newschaffer, C. J., Spence, S. J., & Zimmerman, A. W. (2005). beta2-adrenergic receptor activation and genetic polymorphisms in autism: data from dizygotic twins. *Journal of Child Neurology*, *20*(11), 876–884. (PMID: 16417856)

Constantino, J. N. (2002). *The social responsiveness scale*. Los Angelos: Western Psychological Services.

Cook, R., Brewer, R., Shah, P., & Bird, G. (2013). Alexithymia, not autism, predicts poor recognition of emotional facial expressions. *Psychological Science*. Retrieved from http://pss.sagepub.com/content/early/2013/03/25/0956797612463582 (PMID: 23528789) doi: 10.1177/0956797612463582

Courville, A. C., Daw, N. D., & Touretzky, D. S. (2006). Bayesian theories of conditioning in a changing world. *Trends in Cognitive Sciences*, *10*(7), 294–300. Retrieved from http://www.sciencedirect.com/science/article/pii/S1364661306001288 doi: 10.1016/j.tics.2006.05.004

Crespi, B. (2013). Developmental heterochrony and the evolution of autistic perception, cognition and behavior. *BMC.Med.*, *11*(1741-7015 (Electronic)), 119. Retrieved from PM:23639054 doi: 10.1186/1741-7015-11-119

Dakin, S., & Frith, U. (2005). Vagaries of visual perception in autism. *Neuron*, *48*(3), 497–507. Retrieved from http://www.sciencedirect.com/science/article/pii/S0896627305008846 doi: 10.1016/j.neuron.2005.10.018

Daw, N. D., Niv, Y., & Dayan, P. (2005). Uncertainty-based competition between prefrontal and dorsolateral striatal systems for behavioral control. *Nature Neuroscience*, *8*(12), 1704–1711. Retrieved from http://www.nature.com/neuro/journal/v8/n12/full/nn1560.html doi: 10.1038/nn1560

Dawson, G. (2008). Early behavioral intervention, brain plasticity, and the prevention of autism spectrum disorder. *Development and psychopathology*, *20*(3), 775–803. (PMID: 18606031) doi: 10.1017/S0954579408000370

Dawson, G., Finley, C., Phillips, S., Galpert, L., & Lewy, A. (1988). Reduced p3 amplitude of the event-related brain potential: its relationship to language ability in autism. *J.Autism Dev.Disord.*, *18*(0162-3257 (Print)), 493–504. Retrieved from PM:3215878

Dawson, M., Mottron, L., & Gernsbacher, M. A. (2008). Learning in autism. In *J. H. Byrne (Series Ed.) & H. Roediger (Vol. Ed.). Learning and memory: A comprehensive reference: Cognitive psychology of memory* (pp. 759–772). London: Elsevier. Retrieved from http://www.epubbud.com/read.php?g=ET5HW22S&p=1

Dayan, P. (2012). Twenty-Five lessons from computational neuromodulation. *Neuron*, *76*(1), 240–256. Retrieved from http://www.sciencedirect.com/science/article/pii/S0896627312008628 doi: 10.1016/j.neuron.2012.09.027

Dayan, P., Kakade, S., & Montague, P. R. (2000). Learning and selective attention. *Nature Neuroscience*, *3*(1218-1223), 3758.

D'Cruz, A., Ragozzino, M. E., Mosconi, M. W., Shrestha, S., Cook, E. H., & Sweeney, J. A. (2013). Reduced behavioral flexibility in autism spectrum disorders. *Neuropsychology*, *27*(2), 152–160. (PMID: 23527643) doi: 10.1037/a0031721

de Gardelle, V., Waszczuk, M., Egner, T., & Summerfield, C. (2012). Concurrent repetition enhancement and suppression responses in extrastriate visual cortex. *Cerebral Cortex*. Retrieved from http://cercor.oxfordjournals.org/content/early/2012/07/17/cercor.bhs211 (PMID: 22811008) doi: 10.1093/cercor/bhs211

De Jonge, M. V., Kemner, C., De Haan, E. H., Coppens, J. E., Van Den Berg, T. J. T. P., & Van Engeland, H. (2007). Visual information processing in high-functioning individuals with autism spectrum disorders and their parents. *Neuropsychology*, *21*(1), 65–73.

Delorme, R., Ey, E., Toro, R., Leboyer, M., Gillberg, C., & Bourgeron, T. (2013). Progress toward treatments for synaptic defects in autism. *Nature Medicine*, *19*(6), 685–694. Retrieved from http://www.nature.com/nm/journal/v19/n6/full/nm.3193.html doi: 10.1038/nm.3193

Del Viva, M. M., Igliozzi, R., Tancredi, R., & Brizzolara, D. (2006). Spatial and motion integration in children with autism. *Vision Research*, *46*(8-9), 1242–1252. Retrieved from http://europepmc.org/abstract/MED/16384591/reload=0;jsessionid=QFIR1GTv4ZFd0o3xB30s.4 doi: 10.1016/j.visres.2005.10.018

den Ouden, H. E., Daunizeau, J., Roiser, J., Friston, K. J., & Stephan, K. E. (2010). Striatal prediction error modulates cortical coupling. *The Journal of neuroscience*, *30*(9), 3210–3219. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3044875/ (PMID: 20203180 PMCID: PMC3044875) doi: 10.1523/JNEUROSCI.4458-09.2010

den Ouden, H. E., Kok, P., & Lange, F. P. d. (2012). How prediction errors shape perception, attention, and motivation. *Frontiers in Perception Science*, *3*, 548. Retrieved from http://www.frontiersin.org/Perception_Science/10.3389/fpsyg.2012.00548/full doi: 10.3389/fpsyg.2012.00548

Dichter, G. S., Felder, J. N., & Bodfish, J. W. (2009). Autism is characterized by dorsal anterior cingulate hyperactivation during social target detection. *Social Cognitive and Affective Neuroscience*, *4*(3), 215–226. Retrieved from http://scan.oxfordjournals.org/content/4/3/215 (PMID: 19574440) doi: 10.1093/scan/nsp017

Di Martino, A., Ross, K., Uddin, L. Q., Sklar, A. B., Castellanos, F. X., & Milham, M. P. (2009). Functional brain correlates of social and nonsocial processes in autism spectrum disorders: an activation likelihood estimation meta-analysis. *Biological psychiatry*, *65*(1), 63–74. (PMID: 18996505) doi: 10.1016/j.biopsych.2008.09.022

Diuk, C., Tsai, K., Wallis, J., Botvinick, M., & Niv, Y. (2013). Hierarchical learning induces two simultaneous, but separable, prediction errors in human basal ganglia. *The Journal of Neuroscience*, *33*(13), 5797–5805. Retrieved from http://www.jneurosci.org/content/33/13/5797 doi: 10.1523/JNEUROSCI.5445-12.2013

Dosenbach, N. U., Visscher, K. M., Palmer, E. D., Miezin, F. M., Wenger, K. K., Kang, H. C., ... Petersen, S. E. (2006). A core system for the implementation of task sets. *Neuron*, *50*(5), 799–812. Retrieved from https://stuiterproxy.kuleuven.be/science/article/pii/,DanaInfo=www.sciencedirect.com+S0896627306003497 doi: 10.1016/j.neuron.2006.04.031

Dunn, M., Gomes, H., & Gravel, J. (2008). Mismatch negativity in children with autism and typical development. *J.Autism Dev.Disord.*, *38*(0162-3257 (Print)), 52–71. Retrieved from PM:17624605 doi: 10.1007/s10803-007-0359-3

Duzel, E., & Guitart-Masip, M. (2013). Not so uncertain at last: Locus coeruleus and decision making. *Neuron*, *79*(1), 9–11. Retrieved from http://www.sciencedirect.com/science/article/pii/S0896627313005394 doi: 10.1016/j.neuron.2013.06.023

Ebert, D. H., & Greenberg, M. E. (2013). Activity-dependent neuronal signalling and autism spectrum disorder. *Nature*, *493*(7432), 327–337. Retrieved from http://www.nature.com/doifinder/10.1038/nature11860 doi: 10.1038/nature11860

Edelson, S., Edelson, M., Kerr, D., & Grandin, T. (1999). Behavioral and physiological effects of deep pressure on children with autism: a pilot study evaluating the efficacy of grandin's hug machine. *American Journal of Occupational Therapy*, *53*(0272-9490 (Print)), 145–152. Retrieved from PM:10200837

Edwards, D. J., Perlman, A., & Reed, P. (2012). Unsupervised categorization in a sample of children with autism spectrum disorders. *Research in Developmental Disabilities*, *33*(4), 1264–1269. doi: 10.1016/j.ridd.2012.02.021

Egner, T., Monti, J. M., & Summerfield, C. (2010). Expectation and surprise determine neural population responses in the ventral visual stream. *The Journal of Neuroscience*, *30*(49), 16601–16608. Retrieved from http://www.jneurosci.org/content/30/49/16601 (PMID: 21147999) doi: 10.1523/JNEUROSCI.2770-10.2010

Elison, J. T., Paterson, S. J., Wolff, J. J., Reznick, J. S., Sasson, N. J., Gu, H., ... Piven, J. (2013). White matter microstructure and atypical visual orienting in 7-Month-Olds at risk for autism. *The American journal of psychiatry*. (PMID: 23511344) doi: 10.1176/appi.ajp.2012.12091150

Elsabbagh, M., Divan, G., Koh, Y., Kim, Y. S., Kauchali, S., Marcín, C., ... Fombonne, E. (2012). Global prevalence of autism and other pervasive developmental disorders. *Autism Research*, *5*(3), 160–179. (PMID: 22495912) doi: 10.1002/aur.239

Elsabbagh, M., Volein, A., Holmboe, K., Tucker, L., Csibra, G., Baron-Cohen, S., ... Johnson, M. H. (2009). Visual orienting in the early broader autism phenotype: disengagement and facilitation. *Journal of Child Psychology and Psychiatry*, *50*(5), 637–642. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/j.1469-7610.2008.02051.x/abstract doi: 10.1111/j.1469-7610.2008.02051.x

Falck-Ytter, T. (2008). Face inversion effects in autism: a combined looking time and pupillometric study. *Autism Research*, *1*(5), 297–306. (PMID: 19360681) doi: 10.1002/aur.45

Fan, X., Miles, J. H., Takahashi, N., & Yao, G. (2009). Abnormal transient pupillary light reflex in individuals with autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *39*(11), 1499–1508. (PMID: 19499319) doi: 10.1007/s10803-009-0767-7

Feldman, J. (2013). Tuning your priors to the world. *Topics in Cognitive Science*, *5*(1), 13–34. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/tops.12003/abstract doi: 10.1111/tops.12003

Ferri, R., Elia, M., Agarwal, N., Lanuzza, B., Musumeci, S., & Pennisi, G. (2003). The mismatch negativity and the p3a components of the auditory event-related potentials in autistic low-functioning subjects. *Clinical Neurophysiology*, *114*(1388-2457 (Print)), 1671–1680. Retrieved from PM:12948796

Fletcher, P. C., & Frith, C. D. (2009). Perceiving is believing: a bayesian approach to explaining the positive symptoms of schizophrenia. *Nature Reviews Neuroscience*, *10*(1), 48–58. Retrieved from http://www.nature.com/nrn/journal/v10/n1/abs/nrn2536.html doi: 10.1038/nrn2536

Foss-Feig, J. H., Tadin, D., Schauder, K. B., & Cascio, C. J. (2013). A substantial and unexpected enhancement of motion perception in autism. *The Journal of Neuroscience*, *33*(19), 8243–8249. Retrieved from http://www.jneurosci.org/content/33/19/8243 doi: 10.1523/JNEUROSCI.1608-12.2013

Fournier, K. A., Hass, C. J., Naik, S. K., Lodha, N., & Cauraugh, J. H. (2010). Motor coordination in autism spectrum disorders: A synthesis and Meta-Analysis. *Journal of Autism and Developmental Disorders*, *40*(10), 1227–1240. Retrieved from http://link.springer.com/article/10.1007/s10803-010-0981-3 doi: 10.1007/s10803-010-0981-3

Friston, K. J. (2009). The free-energy principle: a rough guide to the brain? *Trends in Cognitive Sciences*, *13*(7), 293–301. Retrieved from http://www.sciencedirect.com/science/article/B6VH9-4WKY7C0-1/2/e079fb093d97ac028dd7d61c7b14a8f7 doi: 10.1016/j.tics.2009.04.005

Friston, K. J. (2010). The free-energy principle: a unified brain theory? *Nature Reviews Neuroscience*, *11*(2), 127–138. Retrieved from http://dx.doi.org/10.1038/nrn2787 doi: 10.1038/nrn2787

Friston, K. J., Daunizeau, J., Kilner, J., & Kiebel, S. J. (2010). Action and behavior: a free-energy formulation. *Biological Cybernetics*, *102*(3), 227–260. (PMID: 20148260) doi: 10.1007/s00422-010-0364-z

Friston, K. J., Lawson, R., & Frith, C. D. (2013). On hyperpriors and hypopriors: comment on pellicano and burr. *Trends in Cognitive Sciences*, *17*(1), 1. (PMID: 23218940) doi: 10.1016/j.tics.2012.11.003

Friston, K. J., Thornton, C., & Clark, A. (2012). Free-Energy minimization and the Dark-Room problem. *Frontiers in Psychology*, *3*. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3347222/ (PMID: 22586414 PMCID: PMC3347222) doi: 10.3389/fpsyg.2012.00130

Frith, U., & Frith, C. D. (2003). Development and neurophysiology of mentalizing. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, *358*(1431), 459–473. Retrieved from http://rstb.royalsocietypublishing.org/content/358/1431/459 (PMID: 12689373) doi:

10.1098/rstb.2002.1218

Frith, U., & Happé, F. (1994). Autism: beyond "theory of mind". *Cognition*, *50*(1-3), 115–132. (PMID: 8039356)

Froese, T., & Ikegami, T. (2013). The brain is not an isolated "black box," nor is its goal to become one. *Behavioral and Brain Sciences*, *36*(03), 213–214. doi: 10.1017/S0140525X12002348

Gastgeb, H., Dundas, E. M., Minshew, N. J., & Strauss, M. S. (2012). Category formation in autism: Can individuals with autism form categories and prototypes of dot patterns? *Journal of Autism and Developmental Disorders*, *42*(8), 1694–1704. doi: 10.1007/s10803-011-1411-x

Gastgeb, H., Rump, K. M., Best, C. A., Minshew, N. J., & Strauss, M. S. (2009). Prototype formation in autism: Can individuals with autism abstract facial prototypes? *Autism Research*, *2*(5), 279–284. (WOS:000272200900005) doi: 10.1002/aur.93

Gastgeb, H., Wilkinson, D., Minshew, N., & Strauss, M. (2011). Can individuals with autism abstract prototypes of natural faces? *Journal of Autism and Developmental Disorders*, *41*(12), 1609–1618. doi: 10.1007/s10803-011-1190-4

Geschwind, D. H. (2011). Genetics of autism spectrum disorders. *Trends in Cognitive Sciences*, *15*(9), 409–416. Retrieved from http://www.sciencedirect.com/science/article/pii/S1364661311001458 doi: 10.1016/j.tics.2011.07.003

Geurts, H. M., Corbett, B., & Solomon, M. (2009). The paradox of cognitive flexibility in autism. *Trends in Cognitive Sciences*, *13*(2), 74–82. Retrieved from http://www.sciencedirect.com/science/article/pii/S136466130800260X doi: 10.1016/j.tics.2008.11.006

Gibson, E. (1969). *Principles of perceptual learning and development*. East Norwalk, CT, US: Appleton-Century-Crofts.

Gioia, G. A., Isquith, P. K., Kenworthy, L., & Barton, R. M. (2002). Profiles of everyday executive function in acquired and developmental disorders. *Child Neuropsychology*, *8*(2), 121–137. (PMID: 12638065) doi: 10.1076/chin.8.2.121.8727

Gomot, M., Blanc, R., Clery, H., Roux, S., Barthelemy, C., & Bruneau, N. (2011). Candidate electrophysiological endophenotypes of hyper-reactivity to change in autism. *Journal of Autism and Developmental Disorders*, *41*(1573-3432 (Electronic)), 705–714. Retrieved from PM:20827502 doi: 10.1007/s10803-010-1091-y

Gomot, M., Giard, M., Adrien, J., Barthelemy, C., & Bruneau, N. (2002). Hypersensitivity to acoustic change in children with autism: electrophysiological evidence of left frontal cortex dysfunctioning 27. *Psychophysiology*, *39*(0048-5772 (Print)), 577–584. Retrieved from PM:12236323

Gomot, M., & Wicker, B. (2012). A challenging, unpredictable world for people with autism spectrum disorder. *International Journal of Psychophysiology*, *83*(2), 240–247. Retrieved from http://www.sciencedirect.com/science/article/pii/S0167876011002832 doi: 10.1016/j.ijpsycho.2011.09.017

Gonzalez, C., Martin, J. M., Minshew, N. J., & Behrmann, M. (2013). Practice makes improvement: How adults with autism Out-Perform others in a naturalistic visual search task. *Journal of Autism and Developmental Disorders*, 1–10. Retrieved from http://link.springer.com/article/10.1007/s10803-013-1772-4 doi: 10.1007/s10803-013-1772-4

Goris, R. L. T., Wagemans, J., & Wichmann, F. A. (2008). Modelling contrast discrimination data suggest both the pedestal effect and stochastic resonance to be caused by the same mechanism. *Journal of Vision*, *8*(15). Retrieved from http://www.journalofvision.org/content/8/15/17 (PMID: 19146300) doi: 10.1167/8.15.17

Gottlieb, J. (2012). Attention, learning, and the value of information. *Neuron*, *76*(2), 281–295. Retrieved from http://www.cell.com/neuron/abstract/S0896-6273(12)00888-4 doi: 10.1016/j.neuron.2012.09.034

Grandin, T. (1992). Calming effects of deep touch pressure in patients with autistic disorder, college students, and animals. *Journal of Child & Adolescent Psychopharmacology*, *2*(1044-5463 (Print)), 63–72. Retrieved from PM:19630623 doi: 10.1089/cap.1992.2.63

Grandin, T. (1995). *Thinking in pictures and other reports of my life with autism*. New York: Vintage Books Inc.

Gregory, R. L. (1980). Perceptions as hypotheses. *Philosophical Transactions of the Royal Society of London. B, Biological Sciences*, *290*(1038), 181.

Haesen, B., Boets, B., & Wagemans, J. (2011). A review of behavioural and electrophysiological studies on auditory processing and speech perception in autism spectrum disorders. *Research in Autism Spectrum Disorders*, *5*(2), 701–714. Retrieved from http://www.sciencedirect.com/science/article/pii/S175094671000173X doi: 10.1016/j.rasd.2010.11.006

Ham, T., Leff, A., Boissezon, X. d., Joffe, A., & Sharp, D. J. (2013). Cognitive control and the salience network: An investigation of error processing and effective connectivity. *The Journal of Neuroscience*, *33*(16), 7091–7098. Retrieved from http://www.jneurosci.org/content/33/16/7091 (PMID: 23595766) doi: 10.1523/JNEUROSCI.4692-12.2013

Hamilton, A. F. d. C., Brindley, R. M., & Frith, U. (2007). Imitation and action understanding in autistic spectrum disorders: How valid is the hypothesis of a deficit in the mirror neuron system? *Neuropsychologia*, *45*(8), 1859–1868. Retrieved from http://www.sciencedirect.com/science/article/pii/S0028393206004787 doi: 10.1016/j.neuropsychologia.2006.11.022

Happé, F., & Booth, R. (2008). The power of the positive: Revisiting weak coherence in autism spectrum disorders. *The Quarterly Journal of Experimental Psychology*, *61*(1), 50–63. Retrieved from http://www.tandfonline.com/doi/abs/10.1080/17470210701508731 doi: 10.1080/17470210701508731

Happé, F., & Frith, U. (2006). The weak coherence account: detail-focused cognitive style in autism spectrum disorders. *Journal of autism and developmental disorders*, *36*(1), 5–25. (PMID: 16450045) doi: 10.1007/s10803-005-0039-0

Harms, M., Martin, A., & Wallace, G. L. (2010). Facial emo-

tion recognition in autism spectrum disorders: A review of behavioral and neuroimaging studies. *Neuropsychology Review*, *20*(3), 290–322. Retrieved from http://dx.doi.org/10.1007/s11065-010-9138-6 doi: 10.1007/s11065-010-9138-6

Hawkins, J., & Blakeslee, S. (2004). *On intelligence* (Adapted ed.). Times Books.

Heaton, P. (2003). Pitch memory, labelling and disembedding in autism. *J.Child Psychol.Psychiatry*, *44*(0021-9630 (Print)), 543–551. Retrieved from PM:12751846

Heaton, P., Williams, K., Cummins, O., & Happe, F. (2008). Autism and pitch processing splinter skills: a group and subgroup analysis 57. *Autism*, *12*(1362-3613 (Print)), 203–219. Retrieved from PM:18308768 doi: 10.1177/1362361307085270

Helmholtz, H. v. (1962). *Treatise on physiological optics*. New York: Dover.

Herry, C., Bach, D. R., Esposito, F., Di Salle, F., Perrig, W. J., Scheffler, K., ... Seifritz, E. (2007). Processing of temporal unpredictability in human and animal amygdala. *Journal of Neuroscience*, *27*(22), 5958–5966. Retrieved from http://www.jneurosci.org/cgi/content/abstract/27/22/5958 doi: 10.1523/JNEUROSCI.5218-06.2007

Hill, A. L. (1978). Savants: Mentally retarded individuals with special skills. *International Review of Research in Mental Retardation*, *9*, 277–298. Retrieved from http://books.google.be/books?hl=nl&lr=&id=Tc6gzggdPCUC&oi=fnd&pg=PA277&dq=hill+1978+savant&ots=t_RhxZErFp&sig=8KzXEBmSLrdXbDgoCXkMpz82E0Y

Hill, E. L. (2004). Executive dysfunction in autism. *Trends in Cognitive Sciences*, *8*(1), 26–32. (PMID: 14697400) doi: 10.1016/j.dr.2004.01.001

Hillyard, S. A., Vogel, E. K., & Luck, S. J. (1998). Sensory gain control (amplification) as a mechanism of selective attention: electrophysiological and neuroimaging evidence. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *353*(1373), 1257–1270. Retrieved from http://rstb.royalsocietypublishing.org/content/353/1373/1257.full.pdf+html doi: 10.1098/rstb.1998.0281

Hirsh, J. B., Mar, R. A., & Peterson, J. B. (2012). Psychological entropy: a framework for understanding uncertainty-related anxiety. *Psychological Review*, *119*(2), 304–320. (PMID: 22250757) doi: 10.1037/a0026767

Hohwy, J., & Palmer, C. (2014). Social cognition as causal inference: implications for common knowledge and autism. In M. Gallotti & J. Michael (Eds.), *Social ontology and social cognition (forthcoming)*.

Hohwy, J., Roepstorff, A., & Friston, K. (2008). Predictive coding explains binocular rivalry: An epistemological review. *Cognition*, *108*(3), 687–701. Retrieved from http://www.sciencedirect.com/science/article/pii/S0010027708001327 doi: 10.1016/j.cognition.2008.05.010

Honey, R. C., Watt, A., & Good, M. (1998). Hippocampal lesions disrupt an associative mismatch process. *The Journal of Neuroscience*, *18*(6), 2226–2230. (PMID: 9482806)

Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2009). Savant skills in autism: psychometric approaches and parental reports. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *364*(1522), 1359–1367. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2677586/ (PMID: 19528018 PMCID: PMC2677586) doi: 10.1098/rstb.2008.0328

Hughes, C. (1996). Brief report: Planning problems in autism at the level of motor control. *Journal of Autism and Developmental Disorders*, *26*(1), 99–107. Retrieved from http://link.springer.com/article/10.1007/BF02276237 doi: 10.1007/BF02276237

Huron, D. (2006). *Sweet anticipation: Music and the psychology of expectation* (1st ed.). The MIT Press.

Hutsler, J. J., & Zhang, H. (2010). Increased dendritic spine densities on cortical projection neurons in autism spectrum disorders. *Brain Research*, *1309*, 83–94. Retrieved from http://www.sciencedirect.com/science/article/pii/S0006899309023117 doi: 10.1016/j.brainres.2009.09.120

Iarocci, G., & McDonald, J. (2006). Sensory integration and the perceptual experience of persons with autism. *Journal of Autism and Developmental Disorders*, *36*(1), 77–90. Retrieved from http://link.springer.com/article/10.1007/s10803-005-0044-3 doi: 10.1007/s10803-005-0044-3

Ide, J. S., Shenoy, P., Yu, A. J., & Li, C.-s. R. (2013). Bayesian prediction and evaluation in the anterior cingulate cortex. *The Journal of Neuroscience*, *33*(5), 2039–2047. (PMID: 23365241) doi: 10.1523/JNEUROSCI.2201-12.2013

Inoue, W., Baimoukhametova, D. V., Füzesi, T., Cusulin, J. I. W., Koblinger, K., Whelan, P. J., ... Bains, J. S. (2013). Noradrenaline is a stress-associated metaplastic signal at GABA synapses. *Nature Neuroscience*, *16*(5), 605–612. Retrieved from http://www.nature.com/neuro/journal/v16/n5/full/nn.3373.html doi: 10.1038/nn.3373

Jarvinen-Pasley, A., Peppe, S., King-Smith, G., & Heaton, P. (2008). The relationship between form and function level receptive prosodic abilities in autism. *Journal of Autism and Developmental Disorders*, *38*(0162-3257 (Print)), 1328–1340. Retrieved from PM:18172749 doi: 10.1007/s10803-007-0520-z

Joffily, M., & Coricelli, G. (2013). Emotional valence and the Free-Energy principle. *PLoS Comput Biol*, *9*(6), e1003094. Retrieved from http://dx.doi.org/10.1371/journal.pcbi.1003094 doi: 10.1371/journal.pcbi.1003094

Jones, C. R., Happé, F., Baird, G., Simonoff, E., Marsden, A. J., Tregay, J., ... Charman, T. (2009). Auditory discrimination and auditory sensory behaviours in autism spectrum disorders. *Neuropsychologia*, *47*(13), 2850–2858. Retrieved from http://www.sciencedirect.com/science/article/pii/S0028393209002644 doi: 10.1016/j.neuropsychologia.2009.06.015

Joseph, R. M., Keehn, B., Connolly, C., Wolfe, J. M., & Horowitz, T. S. (2009). Why is visual search

superior in autism spectrum disorder? *Developmental Science*, *12*(6), 1083–1096. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/j.1467-7687.2009.00855.x/abstract doi: 10.1111/j.1467-7687.2009.00855.x

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, *2*, 217–250.

Karlsson, M. P., Tervo, D. G. R., & Karpova, A. Y. (2012). Network resets in medial prefrontal cortex mark the onset of behavioral uncertainty. *Science*, *338*(6103), 135–139. Retrieved from http://www.sciencemag.org/content/338/6103/135 (PMID: 23042898) doi: 10.1126/science.1226518

Keehn, B., Brenner, L., Ramos, A., Lincoln, A., Marshall, S., & Muller, R. (2009). Brief report: Eye-movement patterns during an embedded figures test in children with ASD. *Journal of Autism and Developmental Disorders*, *39*(2), 383–387. doi: 10.1007/s10803-008-0608-0

Keehn, B., Müller, R., & Townsend, J. (2013). Atypical attentional networks and the emergence of autism. *Neuroscience & Biobehavioral Reviews*, *37*(2), 164–183. Retrieved from http://www.sciencedirect.com/science/article/pii/S0149763412002047 doi: 10.1016/j.neubiorev.2012.11.014

Kéïta, L., Mottron, L., & Bertone, A. (2010). Far visual acuity is unremarkable in autism: do we need to focus on crowding? *Autism Research*, *3*(6), 333–341. (PMID: 20928845) doi: 10.1002/aur.164

Kemner, C., Verbaten, M., Cuperus, J., Camfferman, G., & Van Engeland, H. (1995). Auditory event-related brain potentials in autistic children and three different control groups. *Biological Psychiatry*, *38*(0006-3223 (Print)), 150–165. Retrieved from PM:7578658 doi: 10.1016/0006-3223(94)00247-Z

Kenworthy, L., Yerys, B. E., Anthony, L. G., & Wallace, G. L. (2008). Understanding executive control in autism spectrum disorders in the lab and in the real world. *Neuropsychology Review*, *18*(4), 320–338. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2856078/ (PMID: 18956239 PMCID: PMC2856078) doi: 10.1007/s11065-008-9077-7

Kern, J. K., Miller, V. S., Cauller, L., Kendall, R., Mehta, J., & Dodd, M. (2001). Effectiveness of N,N-Dimethylglycine in autism and pervasive developmental disorder. *Journal of Child Neurology*, *16*(3), 169–173. Retrieved from http://jcn.sagepub.com/content/16/3/169 (PMID: 11305684) doi: 10.1177/088307380101600303

Kern, J. K., Trivedi, M., Garver, C., Grannemann, B., Andrews, A., Savla, J., ... Schroeder, J. (2006). The pattern of sensory processing abnormalities in autism. *Autism*, *10*(1362-3613 (Print)), 480–494. Retrieved from PM:16940314 doi: 10.1177/1362361306066564

Khalfa, S., Bruneau, N., Roge, B., Georgieff, N., Veuillet, E., Adrien, J., ... Collet, L. (2004). Increased perception of loudness in autism. *Hearing Research*, *198*(0378-5955 (Print)), 87–92. Retrieved from PM:15617227

Kilner, J. M., Friston, K. J., & Frith, C. D. (2007). Predictive coding: an account of the mirror neuron system. *Cognitive Processing*, *8*(3), 159–166. Retrieved from http://link.springer.com/10.1007/s10339-007-0170-2 doi: 10.1007/s10339-007-0170-2

Kim, J. A., Szatmari, P., Bryson, S. E., Streiner, D. L., & Wilson, F. J. (2000). The prevalence of anxiety and mood problems among children with autism and asperger syndrome. *Autism*, *4*(2), 117–132. Retrieved from http://aut.sagepub.com/content/4/2/117 doi: 10.1177/1362361300004002002

Klin, A. (1991). Young autistic childrens listening preferences in regard to speech - a possible characterization of the symptom of social withdrawal. *Journal of Autism and Developmental Disorders*, *21*(1), 29–42. (WOS:A1991FB78000003) doi: 10.1007/BF02206995

Klin, A. (1992). Listening preferences in regard to speech in 4 children with Developmental-Disabilities. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *33*(4), 763–769. (WOS:A1992HV09400010) doi: 10.1111/j.1469-7610.1992.tb00911.x

Klin, A., Jones, W., Schultz, R., Volkmar, F., & Cohen, D. (2002). Visual fixation patterns during viewing of naturalistic social situations as predictors of social competence in individuals with autism. *Archives of General Psychiatry*, *59*, 809–816.

Klin, A., Lin, D., Gorrindo, P., Ramsay, G., & Jones, W. (2009). Two-year-olds with autism orient to non-social contingencies rather than biological motion. *Nature*, *459*(7244), 257–U142. doi: 10.1038/nature07868

Klinger, L., & Dawson, G. (2001). Prototype formation in autism. *Development and Psychopathology*, *13*(01), 111–124.

Koegel, R. L., & Mentis, M. (1985). Motivation in childhood autism: Can they or won't they? *Journal of Child Psychology and Psychiatry*, *26*(2), 185–191. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/j.1469-7610.1985.tb02259.x/abstract doi: 10.1111/j.1469-7610.1985.tb02259.x

Kohler, E., Keysers, C., Umiltà, M. A., Fogassi, L., Gallese, V., & Rizzolatti, G. (2002). Hearing sounds, understanding actions: Action representation in mirror neurons. *Science*, *297*(5582), 846–848. Retrieved from http://www.sciencemag.org/content/297/5582/846 (PMID: 12161656) doi: 10.1126/science.1070311

Kootz, J. P., & Cohen, D. J. (1981). Modulation of sensory intake in autistic children: cardiovascular and behavioral indices. *Journal of the American Academy of Child Psychiatry*, *20*(4), 692–701. (PMID: 7328247)

Koster-Hale, J., & Saxe, R. (2013). Theory of mind: A neural prediction problem. *Neuron*, *79*(5), 836–848. Retrieved from http://www.sciencedirect.com/science/article/pii/S089662731300754X doi: 10.1016/j.neuron.2013.08.020

Kujala, T., Lepisto, T., Nieminen-von, W. T., Naatanen, P., & Naatanen, R. (2005). Neurophysiological evidence for cortical discrimination impairment of prosody in asperger syndrome. *Neuroscience Letters*, *383*(0304-3940 (Print)), 260–265. Retrieved from PM:15885908 doi: 10.1016/j.neulet.2005.04.048

Lam, K. S., Aman, M. G., & Arnold, L. E. (2006). Neuro-

chemical correlates of autistic disorder: A review of the literature. *Research in Developmental Disabilities*, *27*(3), 254–289. Retrieved from http://www.sciencedirect.com/science/article/pii/S0891422205000557 doi: 10.1016/j.ridd.2005.03.003

Legare, C. H. (2012). Exploring explanation: Explaining inconsistent evidence informs exploratory, Hypothesis-Testing behavior in young children. *Child Development*, *83*(1), 173–185. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/j.1467-8624.2011.01691.x/abstract doi: 10.1111/j.1467-8624.2011.01691.x

Lepisto, T., Kujala, T., Vanhala, R., Alku, P., Huotilainen, M., & Naatanen, R. (2005). The discrimination of and orienting to speech and non-speech sounds in children with autism. *Brain Research*, *1066*(0006-8993 (Print)), 147–157. Retrieved from PM:16325159 doi: 10.1016/j.brainres.2005.10.052

Lepisto, T., Silokallio, S., Nieminen-von, W. T., Alku, P., Naatanen, R., & Kujala, T. (2006). Auditory perception and attention as reflected by the brain event-related potentials in children with asperger syndrome. *Clinical Neurophysiology*, *117*(1388-2457 (Print)), 2161–2171. Retrieved from PM:16890012 doi: 10.1016/j.clinph.2006.06.709

Limongi, R., Sutherland, S. C., Zhu, J., Young, M. E., & Habib, R. (2013). Temporal prediction errors modulate cingulate–insular coupling. *NeuroImage*, *71*, 147–157. Retrieved from http://www.sciencedirect.com/science/article/pii/S105381191300044X doi: 10.1016/j.neuroimage.2012.12.078

Lopez, B., Donnelly, N., Hadwin, J. A., & Leekam, S. R. (2004). Face processing in high-functioning adolescents with autism: Evidence for weak central coherence. *Visual Cognition*, *11*(6), 673–688. (WOS:000223476600001) doi: 10.1080/13506280344000437

Lopez, B. R., Lincoln, A. J., Ozonoff, S., & Lai, Z. (2005). Examining the relationship between executive functions and restricted, repetitive symptoms of autistic disorder. *Journal of Autism and Developmental Disorders*, *35*(4), 445–460. Retrieved from http://link.springer.com/article/10.1007/s10803-005-5035-x doi: 10.1007/s10803-005-5035-x

Loth, E., Happe, F., & Gomez, J. C. (2010). Variety is not the spice of life for people with autism spectrum disorders: Frequency ratings of central, variable and inappropriate aspects of common real-life events. *Journal of Autism and Developmental Disorders*, *40*(6), 730–742. (WOS:000277372000008) doi: 10.1007/s10803-009-0929-7

Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, *55*(1), 3–9. (PMID: 3571656)

Lovaas, O. I., Koegel, R. L., & Schreibman, L. (1979). Stimulus overselectivity in autism: a review of research. *Psychological Bulletin*, *86*(6), 1236–1254. (PMID: 515280)

Loveland, K., Tunalikotoski, B., Chen, R., Brelsford, K., Ortegon, J., & Pearson, D. (1995). Intermodal perception of affect in persons with autism or down-Syndrome. *Development and Psychopathology*, *7*(3), 409–418. (WOS:A1995RL78400002)

Mackinlay, R., Charman, T., & Karmiloff-Smith, A. (2006). High functioning children with autism spectrum disorder: A novel test of multitasking. *Brain and Cognition*, *61*(1), 14–24. Retrieved from http://www.sciencedirect.com/science/article/pii/S0278262605001855 doi: 10.1016/j.bandc.2005.12.006

Markram, K., & Markram, H. (2010). The intense world theory - a unifying theory of the neurobiology of autism. *Frontiers in Human Neuroscience*, *4*, 224. (PMID: 21191475) doi: 10.3389/fnhum.2010.00224

Mathys, C., Daunizeau, J., Friston, K. J., & Stephan, K. E. (2011). A bayesian foundation for individual learning under uncertainty. *Frontiers in Human Neuroscience*, *5*. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3096853/ (PMID: 21629826 PMCID: PMC3096853) doi: 10.3389/fnhum.2011.00039

Matson, J. L., & Nebel-Schwalm, M. S. (2007). Comorbid psychopathology with autism spectrum disorder in children: an overview. *Research in developmental disabilities*, *28*(4), 341–352. (PMID: 16765022) doi: 10.1016/j.ridd.2005.12.004

Matson, M. L., Matson, J. L., & Beighley, J. S. (2011). Comorbidity of physical and motor problems in children with autism. *Research in Developmental Disabilities*, *32*(6), 2304–2308. Retrieved from http://www.sciencedirect.com/science/article/pii/S0891422211002939 doi: 10.1016/j.ridd.2011.07.036

May, P., & Tiitinen, H. (2010). Mismatch negativity (MMN), the deviance-elicited auditory deflection, explained. *Psychophysiology*, *47*(1540-5958 (Electronic)), 66–122. Retrieved from PM:19686538 doi: 10.1111/j.1469-8986.2009.00856.x

McPartland, J., Dawson, G., Webb, S. J., Panagiotides, H., & Carver, L. J. (2004). Event-related brain potentials reveal anomalies in temporal processing of faces in autism spectrum disorder. *Journal of Child Psychology and Psychiatry*, *45*(7), 1235–1245. (WOS:000223940300007) doi: 10.1111/j.1469-7610.2004.00318.x

Metereau, E., & Dreher, J. (2013). Cerebral correlates of salient prediction error for different rewards and punishments. *Cerebral Cortex*, *23*(2), 477–487. Retrieved from http://cercor.oxfordjournals.org/content/23/2/477 (PMID: 22368086) doi: 10.1093/cercor/bhs037

Miller, S. (1981). Predictability and human stress: Toward a clarification of evidence and theory. *Advances in Experimental Social Psychology*, *14*, 203–255.

Milne, E., Swettenham, J., Hansen, P., Campbell, R., Jeffries, H., & Plaisted, K. (2002). High motion coherence thresholds in children with autism. *Journal of Child Psychology and Psychiatry*, *43*(2), 255–263. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/1469-7610.00018/abstract doi: 10.1111/1469-7610.00018

Mitchell, P., Mottron, L., Soulieres, I., & Ropar, D. (2010).

Susceptibility to the shepard illusion in participants with autism: Reduced top-down influences within perception? *Autism Research*, *3*(3), 113–119. doi: 10.1002/aur.130

Mongillo, E. A., Irwin, J. R., Whalen, D. H., Klaiman, C., Carter, A. S., & Schultz, R. T. (2008). Audiovisual processing in children with and without autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *38*(7), 1349–1358. (WOS:000257935100015) doi: 10.1007/s10803-007-0521-y

Moran, R. J., Campo, P., Symmonds, M., Stephan, K. E., Dolan, R. J., & Friston, K. J. (2013). Free energy, precision and learning: The role of cholinergic neuromodulation. *The Journal of Neuroscience*, *33*(19), 8227–8236. Retrieved from http://www.jneurosci.org/content/33/19/8227 (PMID: 23658161) doi: 10.1523/JNEUROSCI.4255-12.2013

Mottron, L., Bouvet, L., Bonnel, A., Samson, F., Burack, J. A., Dawson, M., & Heaton, P. (2013). Veridical mapping in the development of exceptional autistic abilities. *Neuroscience & Biobehavioral Reviews*, *37*(2), 209–228. (PMID: 23219745) doi: 10.1016/j.neubiorev.2012.11.016

Mottron, L., & Burack, J. A. (2001). Enhanced perceptual functioning in the development of autism. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research* (pp. 131–148). Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers.

Mottron, L., Dawson, M., Soulières, I., Hubert, B., & Burack, J. (2006). Enhanced perceptual functioning in autism: An update, and eight principles of autistic perception. *Journal of Autism and Developmental Disorders*, *36*(1), 27–43.

Mottron, L., Peretz, I., & Menard, E. (2000). Local and global processing of music in high-functioning persons with autism: beyond central coherence? 74. *Journal of Child Psychology and Psychiatry*, *41*(0021-9630 (Print)), 1057–1065. Retrieved from PM:11099122

Nassar, M. R., Rumsey, K. M., Wilson, R. C., Parikh, K., Heasly, B., & Gold, J. I. (2012). Rational regulation of learning dynamics by pupil-linked arousal systems. *Nature Neuroscience*, *15*(7), 1040–1046. (PMID: 22660479) doi: 10.1038/nn.3130

Nemeth, D., Janacsek, K., Balogh, V., Londe, Z., Mingesz, R., Fazekas, M., ... Vetro, A. (2010). Learning in autism: implicitly superb. *PLOS ONE*, *5*(7), e11731. (PMID: 20661300) doi: 10.1371/journal.pone.0011731

Odom, S. L., Brown, W. H., Frey, T., Karasu, N., Smith-Canter, L. L., & Strain, P. S. (2003). Evidence-Based practices for young children with autism contributions for Single-Subject design research. *Focus on Autism and Other Developmental Disabilities*, *18*(3), 166–175. Retrieved from http://foa.sagepub.com/content/18/3/166 doi: 10.1177/10883576030180030401

O'Reilly, J. X. (2013). Making predictions in a changing world—inference, uncertainty, and learning. *Frontiers in Decision Neuroscience*, *7*, 105. Retrieved from http://www.frontiersin.org/Decision_Neuroscience/10.3389/fnins.2013.00105/full doi: 10.3389/fnins.2013.00105

O'Riordan, M., Plaisted, K. C., Driver, J., & Baron-Cohen, S. (2001). Superior visual search in autism. *Journal of Experimental Psychology: Human Perception and Performance*, *27*(3), 719–730. (PMID: 11424657)

Ornitz, D. E. M. (1974). The modulation of sensory input and motor output in autistic children. *Journal of Autism and Childhood Schizophrenia*, *4*(3), 197–215. Retrieved from http://link.springer.com/article/10.1007/BF02115226 doi: 10.1007/BF02115226

O'Connor, K. (2012). Auditory processing in autism spectrum disorder: A review. *Neuroscience & Biobehavioral Reviews*, *36*(2), 836–854. Retrieved from http://www.sciencedirect.com/science/article/pii/S0149763411002065 doi: 10.1016/j.neubiorev.2011.11.008

Oudeyer, P., Baranes, A., & Kaplan, F. (2010). Intrinsically motivated exploration for developmental and active sensorimotor learning. In O. Sigaud & J. Peters (Eds.), *From motor learning to interaction learning in robots* (pp. 107–146). Springer Berlin Heidelberg. Retrieved from http://link.springer.com/chapter/10.1007/978-3-642-05181-4_6

Oudeyer, P., Kaplan, F., & Hafner, V. (2007). Intrinsic motivation systems for autonomous mental development. *IEEE Transactions on Evolutionary Computation*, *11*(2), 265–286. doi: 10.1109/TEVC.2006.890271

Ozonoff, S., Macari, S., Young, G. S., Goldring, S., Thompson, M., & Rogers, S. J. (2008). Atypical object exploration at 12 months of age is associated with autism in a prospective sample. *Autism*, *12*(5), 457–472. Retrieved from http://aut.sagepub.com/content/12/5/457 doi: 10.1177/1362361308096402

Palmer, C. J., Paton, B., Hohwy, J., & Enticott, P. G. (2013). Movement under uncertainty: The effects of the rubber-hand illusion vary along the nonclinical autism spectrum. *Neuropsychologia*. Retrieved from http://www.sciencedirect.com/science/article/pii/S0028393213002091 doi: 10.1016/j.neuropsychologia.2013.06.020

Paton, B., Hohwy, J., & Enticott, P. G. (2012). The rubber hand illusion reveals proprioceptive and sensorimotor differences in autism spectrum disorders. *Journal of Autism and Developmental Disorders*, *42*(9), 1870–1883. Retrieved from http://link.springer.com/article/10.1007/s10803-011-1430-7 doi: 10.1007/s10803-011-1430-7

Payzan-LeNestour, E., Dunne, S., Bossaerts, P., & O'Doherty, J. P. (2013). The neural representation of unexpected uncertainty during Value-Based decision making. *Neuron*, *79*(1), 191–201. Retrieved from http://www.sciencedirect.com/science/article/pii/S0896627313003681 doi: 10.1016/j.neuron.2013.04.037

Pellicano, E., & Burr, D. (2012). When the world becomes 'too real': a bayesian explanation of autistic perception. *Trends in Cognitive Sciences*, *16*(10), 504–510. (PMID: 22959875) doi: 10.1016/j.tics.2012.08.009

Pellicano, E., Gibson, L., Maybery, M., Durkin, K., & Badcock,

D. (2005). Abnormal global processing along the dorsal visual pathway in autism: A possible mechanism for weak visuospatial coherence? *Neuropsychologia*, *43*(7), 1044–1053. doi: 10.1016/j.neuropsychologia.2004.10.003

Pellicano, E., Jeffery, L., Burr, D., & Rhodes, G. (2007). Abnormal adaptive face-coding mechanisms in children with autism spectrum disorder. *Current Biology*, *17*(17), 1508–1512. (WOS:000249386600033) doi: 10.1016/j.cub.2007.07.065

Perry, E. K., Lee, M. L., Martin-Ruiz, C. M., Court, J. A., Volsen, S. G., Merrit, J., ... Wenk, G. L. (2001). Cholinergic activity in autism: Abnormalities in the cerebral cortex and basal forebrain. *American Journal of Psychiatry*, *158*(7), 1058–1066. Retrieved from http://dx.doi.org/10.1176/appi.ajp.158.7.1058 doi: 10.1176/appi.ajp.158.7.1058

Pierce, K., Glad, K. S., & Schreibman, L. (1997). Social perception in children with autism: an attentional deficit? *Journal of Autism and Developmental Disorders*, *27*(3), 265–282. (PMID: 9229258)

Pinborough-Zimmerman, J., Bakian, A. V., Fombonne, E., Bilder, D., Taylor, J., & McMahon, W. M. (2012). Changes in the administrative prevalence of autism spectrum disorders: Contribution of special education and health from 2002-2008. *Journal of Autism and Developmental Disorders*, *42*(4), 521–530. (WOS:000301571300005) doi: 10.1007/s10803-011-1265-2

Plaisted, K. C. (2001). Reduced generalization in autism: An alternative to weak central coherence. In J. A. Burack, T. Charman, N. Yirmiya, & P. R. Zelazo (Eds.), *The development of autism: Perspectives from theory and research* (pp. 149–169). Mahwah, NJ, US: Lawrence Erlbaum Associates Publishers.

Plaisted, K. C., O'Riordan, M., & Baron-Cohen, S. (1998). Enhanced discrimination of novel, highly similar stimuli by adults with autism during a perceptual learning task. *Journal of Child Psychology and Psychiatry*, *39*(5), 765–775. doi: 10.1111/1469-7610.00375

Poljac, E., & Bekkering, H. (2012). A review of intentional and cognitive control in autism. *Frontiers in Psychology*, *3:436*. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3481002/ (PMID: 23112781 PMCID: PMC3481002) doi: 10.3389/fpsyg.2012.00436

Premack, D., & Woodruff, G. (1978). Does the chimpanzee have a theory of mind? *Behavioral and Brain Sciences*, *1*(04), 515–526. doi: 10.1017/S0140525X00076512

Preuschoff, K., & Bossaerts, P. (2007). Adding prediction risk to the theory of reward learning. *Annals of the New York Academy of Sciences*, *1104*, 135–146. (PMID: 17344526) doi: 10.1196/annals.1390.005

Preuschoff, K., Quartz, S. R., & Bossaerts, P. (2008). Human insula activation reflects risk prediction errors as well as risk. *The Journal of Neuroscience*, *28*(11), 2745–2752. Retrieved from http://www.jneurosci.org/content/28/11/2745 (PMID: 18337404) doi: 10.1523/JNEUROSCI.4286-07.2008

Proulx, T., Inzlicht, M., & Harmon-Jones, E. (2012). Understanding all inconsistency compensation as a palliative response to violated expectations. *Trends in Cognitive Sciences*, *16*(5), 285–291. Retrieved from http://www.sciencedirect.com/science/article/pii/S1364661312000800 doi: 10.1016/j.tics.2012.04.002

Pruett, J., John R, LaMacchia, A., Hoertel, S., Squire, E., McVey, K., Todd, R. D., ... Petersen, S. E. (2011). Social and non-social cueing of visuospatial attention in autism and typical development. *Journal of Autism and Developmental Disorders*, *41*(6), 715–731. (PMID: 20809377) doi: 10.1007/s10803-010-1090-z

Rajkowski, J., Kubiak, P., & Aston-Jones, G. (1993). Correlations between locus coeruleus (LC) neural activity, pupil diameter and behavior in monkey support a role of LC in attention. In *Society for neuroscience abstracts* (Vol. 19, p. 974).

Rao, R. P. N., & Ballard, D. H. (1999). Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. *Nat Neurosci*, *2*(1), 79–87. Retrieved from http://dx.doi.org/10.1038/4580 doi: 10.1038/4580

Reynhout, G., & Carter, M. (2011). Evaluation of the efficacy of social stories (TM) using three single subject metrics. *Research in Autism Spectrum Disorders*, *5*(2), 885–900. doi: 10.1016/j.rasd.2010.10.003

Rice, K., Moriuchi, J. M., Jones, W., & Klin, A. (2012). Parsing heterogeneity in autism spectrum disorders: Visual scanning of dynamic social scenes in school-aged children. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*(3), 238–248. (WOS:000301031200003) doi: 10.1016/j.jaac.2011.12.017

Richler, J., Huerta, M., Bishop, S. L., & Lord, C. (2010). Developmental trajectories of restricted and repetitive behaviors and interests in children with autism spectrum disorders. *Development and Psychopathology*, *22*(01), 55. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/pmc2893549/ doi: 10.1017/S0954579409990265

Robertson, C. E., Kravitz, D. J., Freyberg, J., Baron-Cohen, S., & Baker, C. I. (2013). Slower rate of binocular rivalry in autism. *The Journal of Neuroscience*, *33*(43), 16983–16991. Retrieved from http://www.jneurosci.org/content/33/43/16983 (PMID: 24155303) doi: 10.1523/JNEUROSCI.0448-13.2013

Robertson, C. E., Martin, A., Baker, C. I., & Baron-Cohen, S. (2012). Atypical integration of motion signals in autism spectrum conditions. *PLoS ONE*, *7*(11), e48173. Retrieved from http://dx.doi.org/10.1371/journal.pone.0048173 doi: 10.1371/journal.pone.0048173

Rogers, S. J., & Vismara, L. A. (2008). Evidence-Based comprehensive treatments for early autism. *Journal of clinical child and adolescent psychology*, *37*(1), 8–38. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2943764/ (PMID: 18444052 PMCID: PMC2943764) doi: 10.1080/15374410701817808

Rommelse, N. N., Geurts, H. M., Franke, B., Buitelaar, J. K., & Hartman, C. A. (2011). A review on cognitive and brain endophenotypes that may be common in autism

spectrum disorder and attention-deficit/hyperactivity disorder and facilitate the search for pleiotropic genes. *Neuroscience & Biobehavioral Reviews*, *35*(6), 1363–1396. Retrieved from http://www.sciencedirect.com/science/article/pii/S0149763411000340 doi: 10.1016/j.neubiorev.2011.02.015

Ropar, D., & Mitchell, P. (2001). Susceptibility to illusions and performance on visuospatial tasks in individuals with autism. *Journal of Child Psychology and Psychiatry*, *42*(4), 539–549. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/1469-7610.00748/abstract doi: 10.1111/1469-7610.00748

Ropar, D., & Mitchell, P. (2002). Shape constancy in autism: the role of prior knowledge and perspective cues. *Journal of Child Psychology and Psychiatry*, *43*(5), 647–653. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/1469-7610.00053/abstract doi: 10.1111/1469-7610.00053

Ropar, D., Mitchell, P., & Ackroyd, K. (2003). Do children with autism find it difficult to offer alternative interpretations to ambiguous figures? *British Journal of Developmental Psychology*, *21*(3), 387–395. Retrieved from http://onlinelibrary.wiley.com/doi/10.1348/026151003322277766/abstract doi: 10.1348/026151003322277766

Ropar, D., & Peebles, D. (2007). Sorting preference in children with autism: The dominance of concrete features. *Journal of Autism and Developmental Disorders*, *37*(2), 270–280. doi: 10.1007/s10803-006-0166-2

Rouse, H., Donnelly, N., Hadwin, J., & Brown, T. (2004). Do children with autism perceive second-order relational features? the case of the thatcher illusion. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *45*(7), 1246–1257. doi: 10.1111/j.1469-7610.2004.00317.x

Rushworth, M., Behrens, T., Rudebeck, P., & Walton, M. (2007). Contrasting roles for cingulate and orbitofrontal cortex in decisions and social behaviour. *Trends in Cognitive Sciences*, *11*(4), 168–176. Retrieved from http://linkinghub.elsevier.com/retrieve/pii/S1364661307000538 doi: 10.1016/j.tics.2007.01.004

Rutherford, M. D., Troubridge, E. K., & Walsh, J. (2012). Visual afterimages of emotional faces in high functioning autism. *Journal of Autism and Developmental Disorders*, *42*(2), 221–229. (WOS:000299526100008) doi: 10.1007/s10803-011-1233-x

Said, C. P., Egan, R. D., Minshew, N. J., Behrmann, M., & Heeger, D. J. (2013). Normal binocular rivalry in autism: Implications for the excitation/inhibition imbalance hypothesis. *Vision Research*, *77*, 59–66. Retrieved from http://www.sciencedirect.com/science/article/pii/S0042698912003628 doi: 10.1016/j.visres.2012.11.002

Sasson, N. J., Elison, J. T., Turner-Brown, L. M., Dichter, G. S., & Bodfish, J. W. (2011). Brief report: Circumscribed attention in young children with autism. *Journal of Autism and Developmental Disorders*, *41*(2), 242–247. Retrieved from http://link.springer.com/article/10.1007/s10803-010-1038-3 doi: 10.1007/s10803-010-1038-3

Sasson, N. J., Turner-Brown, L. M., Holtzclaw, T. N., Lam, K. S., & Bodfish, J. W. (2008). Children with autism demonstrate circumscribed attention during passive viewing of complex social and nonsocial picture arrays. *Autism Research*, *1*(1), 31–42. Retrieved from http://onlinelibrary.wiley.com/doi/10.1002/aur.4/abstract doi: 10.1002/aur.4

Saygin, A., Cook, J., & Blakemore, S. (2010). Unaffected perceptual thresholds for biological and non-biological form-from-motion perception in autism spectrum conditions. *PLOS ONE*, *5*(10), e13491. doi: 10.1371/journal.pone.0013491

Schiffer, A., Ahlheim, C., Wurm, M. F., & Schubotz, R. I. (2012). Surprised at all the entropy: Hippocampal, caudate and midbrain contributions to learning from prediction errors. *PLOS ONE*, *7*(5), e36445. Retrieved from http://dx.plos.org/10.1371/journal.pone.0036445 doi: 10.1371/journal.pone.0036445

Schiffer, A., & Schubotz, R. I. (2011). Caudate nucleus signals for breaches of expectation in a movement observation paradigm. *Frontiers in Human Neuroscience*, *5*. Retrieved from http://www.frontiersin.org/Human_Neuroscience/10.3389/fnhum.2011.00038/abstract doi: 10.3389/fnhum.2011.00038

Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science*, *275*(5306), 1593–1599. Retrieved from http://www.sciencemag.org/content/275/5306/1593 (PMID: 9054347) doi: 10.1126/science.275.5306.1593

Serretti, A., & Fabbri, C. (2013). Shared genetics among major psychiatric disorders. *Lancet*, *381*(9875), 1339–1341. (PMID: 23453886) doi: 10.1016/S0140-6736(13)60223-8

Seth, A. K., Suzuki, K., & Critchley, H. D. (2012). An interoceptive predictive coding model of conscious presence. *Frontiers in Psychology*, *2*. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3254200/ (PMID: 22291673 PMCID: PMC3254200) doi: 10.3389/fpsyg.2011.00395

Shah, A., & Frith, U. (1993). Why do autistic individuals show superior performance on the block design task? *Journal of Child Psychology and Psychiatry*, *34*(8), 1351–1364. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/j.1469-7610.1993.tb02095.x/abstract doi: 10.1111/j.1469-7610.1993.tb02095.x

Silverstein, S. M. (2013). Schizophrenia-related phenomena that challenge prediction error as the basis of cognitive functioning. *Behavioral and Brain Sciences*, *36*(03), 229–230. doi: 10.1017/S0140525X12002221

Simmons, D. R., Robertson, A. E., McKay, L. S., Toal, E., McAleer, P., & Pollick, F. E. (2009). Vision in autism spectrum disorders. *Vision Research*, *49*(22), 2705–2739. Retrieved from http://www.sciencedirect.com/science/article/pii/S0042698909003563 doi: 10.1016/j.visres.2009.08.005

Smith, E. G., & Bennetto, L. (2007). Audiovisual speech

integration and lipreading in autism. *Journal of Child Psychology and Psychiatry*, *48*(8), 813–821. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/j.1469-7610.2007.01766.x/abstract doi: 10.1111/j.1469-7610.2007.01766.x

Smith, J. D. (2009). The study of animal metacognition. *Trends in Cognitive Sciences*, *13*(9), 389–396. Retrieved from http://www.sciencedirect.com/science/article/pii/S136466130900148X doi: 10.1016/j.tics.2009.06.009

Smith, J. D., Coutinho, M. V. C., Church, B. A., & Beran, M. J. (2013). Executive-attentional uncertainty responses by rhesus macaques (Macaca mulatta). *Journal of Experimental Psychology: General*, *142*(2), 458–475. Retrieved from http://doi.apa.org/getdoi.cfm?doi=10.1037/a0029601 doi: 10.1037/a0029601

Soulières, I., Mottron, L., Giguère, G., & Larochelle, S. (2011). Category induction in autism: Slower, perhaps different, but certainly possible. *The Quarterly Journal of Experimental Psychology*, *64*(2), 311–327. doi: 10.1080/17470218.2010.492994

Spencer, J., O'Brien, J., Riggs, K., Braddick, O., Atkinson, J., & Wattam-Bell, J. (2000). Motion processing in autism: Evidence for a dorsal stream deficiency. *Neuroreport*, *11*(12), 2765–2767. (PMID: 10976959)

Spengler, S., Bird, G., & Brass, M. (2010). Hyperimitation of actions is related to reduced understanding of others' minds in autism spectrum conditions. *Biological Psychiatry*, *68*(12), 1148–1155. (WOS:000285389000012) doi: 10.1016/j.biopsych.2010.09.017

Stoet, G., & López, B. (2011). Task-switching abilities in children with autism spectrum disorder. *European Journal of Developmental Psychology*, *8*(2), 244–260. Retrieved from http://www.tandfonline.com/doi/abs/10.1080/17405629.2010.492000 doi: 10.1080/17405629.2010.492000

Summerfield, C., Monti, J. M., Trittschuh, E. H., Mesulam, M., & Egner, T. (2008). Neural repetition suppression reflects fulfilled perceptual expectations. *Nature Neuroscience*, *11*(9), 1004–1006. (PMID: 19160497 PMCID: 2747248)

Taylor, N., Isaac, C., & Milne, E. (2010). A comparison of the development of audiovisual integration in children with autism spectrum disorders and typically developing children. *Journal of Autism and Developmental Disorders*, *40*(11), 1403–1411. (PMID: 20354776) doi: 10.1007/s10803-010-1000-4

Teufel, C., Subramaniam, N., & Fletcher, P. C. (2013). The role of priors in bayesian models of perception. *Frontiers in Computational Neuroscience*, 25. Retrieved from http://www.frontiersin.org/Computational_Neuroscience/10.3389/fncom.2013.00025/full doi: 10.3389/fncom.2013.00025

Todorovic, A., & de Lange, F. P. (2012). Repetition suppression and expectation suppression are dissociable in time in early auditory evoked fields. *Journal of Neuroscience*, *32*(39), 13389–13395. Retrieved from http://www.jneurosci.org/content/32/39/13389 (PMID: 23015429) doi: 10.1523/JNEUROSCI.2227-12.2012

Todorovic, A., van Ede, F., Maris, E., & de Lange, F. (2011). Prior expectation mediates neural adaptation to repeated sounds in the auditory cortex: an MEG study. *Journal of Neuroscience*, *31*(1529-2401 (Electronic)), 9118–9123. Retrieved from PM:21697363 doi: 10.1523/JNEUROSCI.1425-11.2011

Tomchek, S., & Dunn, W. (2007). Sensory processing in children with and without autism: a comparative study using the short sensory profile. *American Journal of Occupational Therapy*, *61*(0272-9490 (Print)), 190–200. Retrieved from PM:17436841

Treffert, D. A. (2009). The savant syndrome: an extraordinary condition. a synopsis: past, present, future. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *364*(1522), 1351–1357. Retrieved from http://rstb.royalsocietypublishing.org/content/364/1522/1351 doi: 10.1098/rstb.2008.0326

Turner, M. (1999). Annotation: Repetitive behaviour in autism: A review of psychological research. *Journal of Child Psychology and Psychiatry*, *40*(6), 839–849. Retrieved from http://onlinelibrary.wiley.com/doi/10.1111/1469-7610.00502/abstract doi: 10.1111/1469-7610.00502

Uddin, L. Q., & Menon, V. (2009). The anterior insula in autism: Under-connected and under-examined. *Neuroscience & Biobehavioral Reviews*, *33*(8), 1198–1203. Retrieved from http://linkinghub.elsevier.com/retrieve/pii/S0149763409000815 doi: 10.1016/j.neubiorev.2009.06.002

Uljarevic, M., & Hamilton, A. (2013). Recognition of emotions in autism: A formal meta-analysis. *Journal of Autism and Developmental Disorders*, 1–10. Retrieved from http://link.springer.com/article/10.1007/s10803-012-1695-5 doi: 10.1007/s10803-012-1695-5

Valentine, T. (1991). A unified account of the effects of distinctiveness, inversion, and race. *Quarterly Journal of Experimental Psychology Section a-Human Experimental Psychology*, *43*(2), 161–204. (WOS:A1991FM42200001)

Van de Cruys, S., de-Wit, L., Evers, K., Boets, B., & Wagemans, J. (2013). Weak priors versus overfitting of predictions in autism: Reply to pellicano and burr (TICS, 2012). *i-Perception*, *4*(2), 95–97. Retrieved from http://i-perception.perceptionweb.com/journal/I/article/i0580ic

Van de Cruys, S., & Wagemans, J. (2011). Putting reward in art: A tentative prediction error account of visual art. *i-Perception*, *2*(9), 1035–1062. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3485793/ (PMID: 23145260 PMCID: PMC3485793) doi: 10.1068/i0466aap

van Boxtel, J. J. A., & Lu, H. (2013). A predictive coding perspective on autism spectrum disorders. *Frontiers in Psychology*, *4*. Retrieved from http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3556598/ (PMID: 23372559 PMCID: PMC3556598) doi: 10.3389/fpsyg.2013.00019

Vandenbroucke, M. W. G., Scholte, H. S., van Engeland, H., Lamme, V. A. F., & Kemner, C. (2008). Coherent ver-

sus component motion perception in autism spectrum disorder. *Journal of Autism and Developmental Disorders*, *38*(5), 941–949. (PMID: 17952582) doi: 10.1007/s10803-007-0467-0

van der Geest, J. N., Kemner, C., Verbaten, M. N., & van Engeland, H. (2002). Gaze behavior of children with pervasive developmental disorder toward human faces: a fixation time study. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *43*(5), 669–678. (WOS:000176357400011) doi: 10.1111/1469-7610.00055

Van der Hallen, R., Evers, K., Brewaeys, K., Van den Noortgate, W., & Wagemans, J. (2014). Global processing takes time: A Meta-Analysis on Local-Global visual processing in ASD.
(under review)

Van Eylen, L., Boets, B., Steyaert, J., Evers, K., Wagemans, J., & Noens, I. (2011). Cognitive flexibility in autism spectrum disorder: Explaining the inconsistencies? *Research in Autism Spectrum Disorders*, *5*(4), 1390–1401. Retrieved from http://www.sciencedirect.com/science/article/pii/S1750946711000316 doi: 10.1016/j.rasd.2011.01.025

Vladusich, T., Olu-Lafe, O., Kim, D., Tager-Flusberg, H., & Grossberg, S. (2010). Prototypical category learning in high-functioning autism. *Autism Research*, *3*(5), 226–236. doi: 10.1002/aur.148

Volkmar, F., Chawarska, K., & Klin, A. (2005). Autism in infancy and early childhood. *Annual Review of Psychology*, *56*, 315–336. Retrieved from ://000227503800013 doi: 10.1146/annurev.psych.56.091103.070159

Vossel, S., Mathys, C., Daunizeau, J., Bauer, M., Driver, J., Friston, K. J., & Stephan, K. E. (2013). Spatial attention, precision, and bayesian inference: A study of saccadic response speed. *Cerebral Cortex*. Retrieved from http://cercor.oxfordjournals.org/content/early/2013/01/14/cercor.bhs418 (PMID: 23322402) doi: 10.1093/cercor/bhs418

Wacongne, C., Changeux, J., & Dehaene, S. (2012). A neuronal model of predictive coding accounting for the mismatch negativity. *The Journal of Neuroscience*, *32*(11), 3665–3678. Retrieved from http://www.jneurosci.org/content/32/11/3665 (PMID: 22423089) doi: 10.1523/JNEUROSCI.5003-11.2012

Wacongne, C., Labyt, E., Wassenhove, V. v., Bekinschtein, T., Naccache, L., & Dehaene, S. (2011). Evidence for a hierarchy of predictions and prediction errors in human cortex. *Proceedings of the National Academy of Sciences*, *108*(51), 20754–20759. Retrieved from http://www.pnas.org/content/108/51/20754 doi: 10.1073/pnas.1117807108

Webb, S. J., Merkle, K., Murias, M., Richards, T., Aylward, E., & Dawson, G. (2012). ERP responses differentiate inverted but not upright face processing in adults with ASD. *Social Cognitive and Affective Neuroscience*, *7*(5), 578–587. (WOS:000305409900010) doi: 10.1093/scan/nsp002

Weigelt, S., Koldewyn, K., & Kanwisher, N. (2012). Face identity recognition in autism spectrum disorders: A review of behavioral studies. *Neuroscience & Biobehavioral Reviews*, *36*(3), 1060–1084. Retrieved from http://www.sciencedirect.com/science/article/pii/S0149763411002156 doi: 10.1016/j.neubiorev.2011.12.008

White, S. J. (2013). The triple i hypothesis: Taking another('s) perspective on executive dysfunction in autism. *Journal of Autism and Developmental Disorders*, *43*(1), 114–121. Retrieved from http://link.springer.com/article/10.1007/s10803-012-1550-8 doi: 10.1007/s10803-012-1550-8

White, S. J., Burgess, P. W., & Hill, E. L. (2009). Impairments on "open-ended" executive function tests in autism. *Autism Research*, *2*(3), 138–147. Retrieved from http://onlinelibrary.wiley.com/doi/10.1002/aur.78/abstract doi: 10.1002/aur.78

Winkler, I., Denham, S. L., & Nelken, I. (2009). Modeling the auditory scene: predictive regularity representations and perceptual objects. *Trends in Cognitive Sciences*, *13*(12), 532–540. Retrieved from http://www.sciencedirect.com/science/article/pii/S1364661309002095 doi: 10.1016/j.tics.2009.09.003

Yu, A. J., & Dayan, P. (2005). Uncertainty, neuromodulation, and attention. *Neuron*, *46*(4), 681–692. (PMID: 15944135) doi: 10.1016/j.neuron.2005.04.026

Zaki, J. (2013). Cue integration a common framework for social cognition and physical perception. *Perspectives on Psychological Science*, *8*(3), 296–312. Retrieved from http://pps.sagepub.com/content/8/3/296 doi: 10.1177/1745691613475454

Zwaigenbaum, L., Bryson, S., Rogers, T., Roberts, W., Brian, J., & Szatmari, P. (2005). Behavioral manifestations of autism in the first year of life. *International Journal of Developmental Neuroscience*, *23*(2–3), 143–152. Retrieved from http://www.sciencedirect.com/science/article/pii/S0736574804000553 doi: 10.1016/j.ijdevneu.2004.05.001