Precise Minds in Uncertain Worlds: Predictive Coding in Autism

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There have been numerous attempts to explain the enigma of autism, but existing neurocognitive theories often provide merely a refined description of 1 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 disorder 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 autism spectrum disorder. The complex, fluctuating nature of regularities in the world and the stochastic and noisy biological system through which people experience it require that, in the real world, people not only learn from their errors but 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, people 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

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Autism spectrum disorder (ASD) refers to a group of neurodevelopmental conditions with an early onset and characterized by sociocommunicative impairments and stereotyped, restricted behavior patterns and interests (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., attention-deficit/hyperactivity disorder, 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 into two groups, depending on which symptoms are considered 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). It focuses on the social problems and argues that the core deficit lies in the understanding of the behavior of others in terms of one's own 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) focus on the perceptual pecu-

liarities in ASD and argue for a locally (as opposed to globally) oriented processing style in individuals with ASD. Accounts that, 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 has been highly influential in shaping the fieldand has shifted the research and clinical focus from an exclusively descriptive behavioral approach toward an enhanced desire to understand the atypical neurocognitive mechanisms in ASD. Nevertheless, serious limitations with these frameworks have become evident over the years. First, although local processing styles, theory-of-mind difficulties, and executive problems are common in ASD, they are neither specific to the disorder nor apparent in all cases. Second, although they are called neurocognitive, these mechanisms 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, although 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 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 that, 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 (The Anticipating Brain) 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 (Predictive Coding in ASD) we propose a specific etiological mechanism for ASD, which is then 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 (Development and Exploration). Next, we discuss how perceptual and cognitive alterations in ASD can originate from our theory (Cognitive Functioning). In the subsequent sections, sensorimotor and affective consequences are covered (Sensorimotor Abilities and a Sense of Self and Chronic Unpredictability and Its Affective Consequences). In the Social Functioning section, 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 (Neurobiological Underpinnings). Before reaching our conclusions, we cover a few related accounts of ASD to discuss commonalities and indicate the added value of our approach (Related Approaches).

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 the environment. Some of this structure is readily available, and 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 through the development of predictive coding models (Clark, 2013b). This computational scheme is heavily inspired by perception-as-inference (von 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. It slso stipulates 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, although 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 extraclassical 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 downward, 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. The information immediately becomes input relative to the organism, its models of the world, and its current state. This approach also emphasizes that processing does not start with the onset of a stimulus. Preexisting, intrinsic activity of the brain is considered formative, as it reflects the continuous predictive activity of the proactive 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, & de 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) described 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 but 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 uncertainties 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 in order 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 the Neurobiological Underpinnings section).

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.

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 ±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 yet fully learned. 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 that 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 and, 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, deriving our model 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 demo-

tivating effect on particularly noisy interactions, such as those involved in social situations (see the Social Functioning section).

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 are 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; that is, 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 that in typically developing individuals, if not superior (see the Perceptual Processing section).

Of importance, 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) from having an effect on perception and learning. Aberrant neuromodulatory mechanisms of precision, as discussed in the Neurobiological Underpinnings section, 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 the Neurobiological Underpinnings section.

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 that 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 from being 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 the Social Functioning section).

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, more complex 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 discernible 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). The study investigated how TD children explain evidence violating their predictions and illustrated 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 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), and 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 interesting, however, the kind of explanation children gave predicted the way they played with the objects later on. Although 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 into two categories based on what had happened when they first placed the objects on the box. This sorting behavior was a less sophisticated form of exploration and was 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 is considered a special case unlike previous instances. The latter is what HIPPEA proposes to be the case in ASD. Though Legare's (2012) study included only TD children, the sorting behavior found in spontaneous play for the subset of children that gave noncausal 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 in which they will engage. 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, 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 involves not only learned mental models and perceptual predictionsbut 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, p. 213). Caregivers of children with ASD often describe them as detached from the world, as if they are living in their own walled world. This is not because they are unhappy or are 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, para. 15). Hence, it seems that children with ASD, because they (initially) cannot predict more complex environments, are perfectly content to stick to the confined space and motion they know.

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 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, are covered in the Social Functioning section.

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 by 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 for a target 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 facili-

tates 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 which features in a task are relevant). 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 children with ASD, TD children, and mentally disabled children were presented with video fragments of social interactions containing one or more cues, children with ASD 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) noted 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 the larger reaction times for diverse tasks that are 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, perseveration and overselectivity may often 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 report clear deficits, reflected by a higher number of perseverative responses when a rule switch should occur, and more controlled task-switching paradigms generally fail to find cognitive flexibility problems in ASD (Geurts, Corbett, & Solomon, 2009; Van Eylen et al., 2011).

Several researchers recently 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 an indication that a rule switch will occur (as in the Wisconsin Card Scoring Task), 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 has 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 but 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 childrens books (Ropar & Peebles, 2007), children with ASD relied less on category labels (games vs. sports) and more on purely perceptual features (color and size) than did TD children. More one-dimensional sorting was found in free sorting of shapes by children with ASD, especially in more complex stimulus sets (D. J. Edwards, Perlman, & Reed, 2012). In a 20-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 flexible switching of 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 these 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 nonreinforcement. 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 cuing task as developed previously (Vossel et al., 2014; Yu & Dayan, 2005) could contribute to this. In the typical Posner cuing task, a simple cue (a briefly presented flash) indicates the actual (valid) location of the target to children in only a certain percentage of trials (e.g., 75%). Participants typically 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 (e.g., when probability structure has 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) precedes 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., 2014), pin-pointing exactly whether and how the learning style of individuals with ASD 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 those with 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. Whereas 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 are motivated to do well in them.

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), and 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. This idea is consistent with EPF, but we can better specify how and why perception is enhanced. The detectable size of prediction errors is not smaller; 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, which will have limited applicability, will be formed. 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, although 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 toward 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 upward, 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). Also relevant in this context is the increased prevalence of absolute pitch and musical savants in the ASD population (e.g., Heaton, Williams, Cummins, & Happé, 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 this finding 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 perceptual gain control.

Local versus global perception. A common paradigm to study a more locally focused processing style in ASD is to examine susceptibility to visual illusions. Overall, these studies yielded mixed to positive effects. Although 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), and participants with ASD reported a more elliptic shape (closer to the proximal stimulus, not discounting the slant).

The global–local processing issue is usually studied with 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 lessens 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 the use of 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, because availability of top-down templates evidently also influences rivalry. For example, the first study uses gratings and the second uses familiar objects. The less familiar (or semantically high-level) the stimulus, the better it is for use in studies of ASD, 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 is 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 those with ASD than in controls.

Face and speech perception. Face and speech perception are crucial for smooth and successful social interactions and are therefore prominent targets of ASD research. Deep difficulties here can go a long way in explaining communication problems so central in ASD. Of interest, face and speech perception are also prime examples of the hierarchical "analysis by synthesis" approach inherent to predictive coding. This would normally 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, suppressing 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. Not only does the brain 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) and can only be extracted by integrating and interpolating information, a process that 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 those with ASD we see increased access to fine-grained acoustic features of complex sounds (e.g., disembedding tones from musical chords; Heaton, 2003; Mottron, Peretz, & Ménard, 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 impede 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 those with 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 intraclass similarity, with small and rather subtle differences among many dimensions distinguishing two human faces from each other. Countless transformations of an individual face among 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 extrafacial 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 nonfunctional 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 were 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 are 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). Forming a face prototype typically 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. Plaisted et al. (1998) consistently found that high-functioning adults with ASD learned to discriminate between configurations of colored disks to higher levels of accuracy than controls; when tested with slightly different exemplars of the same overall configurations, normal controls showed transfer from the learned exemplars to the novel ones, and 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 spontaneous in 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 (e.g., Pellicano, Jeffery, Burr, & Rhodes, 2007; Rutherford, Troubridge, & Walsh, 2012). 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. Although 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 informa-

tive. Here, presentation of an unexpected oddball stimulus within a sequence of repeated predictable stimuli, elicits a novelty response in the event-related potential. The MMN was originally 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). With regard to the MMN, a number of studies observed larger amplitudes and/or earlier latencies to infrequent pitch changes in tones and vowels in those with ASD relative to TD controls, thus suggesting 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). Of interest, 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 and thus suggestive of impaired recognition of the more global phonetic characteristics of speech (e.g., Kujala, Lepisto, Nieminen-von Wendt, 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 those with 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 (enhanced and earlier MMN sensory responses toward simple stimuli) but have difficulty picking up changes in the broader frontoparietal predictive system, which is tuned toward 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.

Savant Skills

Autistic savants are individuals with ASD with co-occurring excellence in an isolated skill (i.e., an "island of genius" that 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 should remain a peak in performance.

Several scholars have 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 increased 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 argued that people with ASD prefer either no change or systems that 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 this is 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 those with 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). Although 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.

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 M. J. Edwards, Adams, Brown, Pareés, and

Friston (2012), movement is defined by "what we want to see (or feel), rather than what we want to do" (p. 3498). 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. Unpredictable surroundings in particular may be expected to elicit this kind of behavior, with the aim of reestablishing predictability and reducing stress (see the Chronic Unpredictability and Its Affective Consequences section). Ornitz (1974) observed 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" (p. 204). This latter part is significant because it indicates that although 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 aim only to reduce that part of the perceptual input that cannot be predicted and moreover actively attempt to create predictability to compensate. 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 a perfectly controlled (i.e., predictable) act 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 extracorporeal 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 overprecision 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 those with 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.

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 hypersensitivity 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. the Development and Exploration section). 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 have 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 hyperactivity (Ben-Sasson et al., 2009; Tomchek & Dunn, 2007). Social interactions might suffer most from this lack of motivation (Chevallier, Grezes, Molesworth, Berthoz, & Happé, 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 to be 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).

Social Functioning

Social interaction problems are among the first described symptoms of ASD (Asperger, 1944; Kanner, 1943) and are crucial pieces in *the Diagnostic and Statistical Manual of Mental Disorders* 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 nonsocial 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.

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, and so on (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 diffi-cult 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). Sub-cultures (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 prop-erties 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 nonsocial stimuli. This deficit is most clearly illustrated by eye-movement studies. Individuals with ASD show a reduced attention to faces but more attention toward bodies and objects in the background of a social scene (e.g., Klin, Jones, Schultz, Volk-mar, & 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 toward 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 nonsocial phys-ical 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 stim-ulus material (Chevallier et al., 2012; Harms et al., 2010) also speaks for our hypothesis. Although the distinction between rele-vant and irrelevant information may be rather clear-cut in simple social stimuli (e.g., isolated, well-controlled, and reused 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 orga-nized 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, Happé, & Gomez, 2010). It is easy to see that indiscriminate precision of social and nonsocial cues results in narrow and specific social scripts (e.g., making friends when I'm playing soccer), wrought with spurious, concrete features. Inter-ventions that try to remedy social script deficits select and describe the relevant cues for a given script, linking it with possible appro-priate responses (for a meta-analysis, see Reynhout & Carter, 2011).

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 circum-stances. Again we note that uncertainty of the different sources has to be taken into account in order to determine which information should have more say in the eventual social judgment. Indeed, this can be formalized with Bayes' theorem (Zaki, 2013), which is already widely used in (nonsocial) perceptual cue integration stud-ies. 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 intermodal 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 spa-tiotemporal 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 mis-match 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 be similarly explained (Mongillo et al., 2008; Taylor, Isaac, & Milne, 2010).

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 implic-it—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 (sometimes described as "mindblind-ness"; e.g., Baron-Cohen, 2001; Frith & Frith, 2003). The discov-ery of mirror neurons (in monkeys) that are active both during the action observation or imagination (offline processing) and 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 friend's 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. Of importance, in predictive coding this hierarchical model is used for forward action generation and 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 be explained away only 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, but 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 Sensorimotor Abilities and a Sense of Self). 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 from transcending 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, hyperimitation 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 the Neurobiological Underpinnings section), 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 built (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 recognizing emotion 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 literature review and a meta-analysis, respectively) 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 prevent adequate emotional inferences (Seth et al., 2012).

Neurobiological Underpinnings

In this 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 (see, e.g., D. G. Amaral, Schumann, & Nordahl, 2008; Bauman & Kemper, 2005). 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 into 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 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) have long been 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 have reported abnormalities (Lam, Aman, & Arnold, 2006), including in the main source of ACh, the basal forebrain (Bauman & Kemper, 1994; Perry et al., 2001). Raised NE signaling 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 Attention and Executive Functioning). 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 meta-plastic 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 should 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 the Development and Exploration section 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 oddball 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 participants, 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, de 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.

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 address the question whether a unified account is possible at all, in view of the heterogeneous nature of ASD. We close this section with a discussion of a recent theory of schizophrenia, which is closely related to our theory of ASD.

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). Pellicano and Burr 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 the Perceptual Processing section). In other words it would, according to Pellicano and Burr, 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 Boxtel & Lu, 2013; Van de Cruys et al., 2013), this stance has been criticized on theoretical and empirical grounds. Teufel et al. (2013) reminded 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) noted that perception (the posterior) can move closer to perceptual input (likelihood) for two different reasons: either, as Pellicano and Burr (2012) argued, 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 the Attention and Executive Functioning section). 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 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). Although 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, nonrepeating noise. We believe that this view is more readily compatible with the broad range of behavioral peculiarities in ASD.

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 & Ronald, 2008). Finally and most important, phenotypic variability is notorious in ASD (Rommelse, Geurts, Franke, Buitelaar, & Hartman, 2011). This causes but may also be caused by difficulties in diagnosing ASD. Bringing the view of ASD as singular entity even further into question is the fact that "virtually every symptom characteristic of ASD can be observed in children who do not fit this diagnostic category" (Bishop, 1989, The Borderlands of Autism section, para. 2). 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 Ronald (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 do subclinical signs become clinical symptoms meriting a diagnosis.

Naturally, we agree with Happé and Ronald (2008) that a better characterization of the subcomponents of ASD is much needed, but an intrinsic coherence of the components may shine through only when the appropriate level of description has been found. As we progress toward 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, although executive functioning and attentional deficits may not be specific to ASD (cf. attention-deficit/hyperactivity disorder), 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 such as 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 toward the syndrome does not preclude the possibility that one main cognitive mechanism is impaired. There is little reason to expect a one-to-one mapping from cognitive processing to neurobiology. The previous section provided possible ways HIPPEA links up with neurobiological evidence.

ASD in Relation to Schizophrenia

Increasing evidence suggests that 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, Frith, & 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 worldviews and delusions.

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).

Although 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 overaccommodating 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. More work is clearly needed in this respect.

Conclusions

Although 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 because 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 conclusively demonstrated in only 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 psychoeducation for ASD that focus on early learning (such as applied behavioral analysis, Lovaas, 1987; Rogers & Vismara, 2008) could take inspiration from HIPPEA, which 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 on exploration (Development and 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 counterintuitively, reducing intense concentration on learning experiences, thus preventing individuals from trying to match all details ("early stopping"), has also been proposed to be beneficial (Bakouie et al., 2009).

Although we consider HIPPEA to be 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 nonfalsifiability (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.

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