

# Agency and Reward in Typical Development and Autism

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The ability to perform voluntary actions and make choices is shaped by the motivation from having control over the resulting effects (agency) and positive outcomes (reward). It is proposed that reduced sensitivity to agency and reward in autism spectrum disorder (ASD) may be related to atypical multisensory processes and motor planning, with potential for understanding restricted and repetitive behaviors.

Keywords: agency ; reward ; ASD ; autism ; motor planning

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## 1. Introduction

Our ability to perform actions and make choices is fundamental in our daily interactions with the world of physical and social objects. The link between a given action and its effects in the surrounding environment modifies our behavior and the underlying cognitive and neural processes, with meaningful effects on our acting, thinking, and learning. We can distinguish between the perception of control over the effects of one's actions (agency) and the search for positive outcomes (reward). Understanding how these two different mechanisms give rise to a person's actions and choices allows us to facilitate learning, and also volition and self-determination. This may be crucial for interventions that aim to support learning processes for people with neurodevelopmental conditions whereby agency and reward mechanisms can be affected.

## 2. The Role of Agency and Reward in Shaping Actions

In everyday life, we perform voluntary, goal-oriented actions for which we hold ourselves responsible. Agency can be defined as the perception of control over one's own actions and the external world and can be traced back to the ability to recognize oneself as the cause of an event <sup>[1][2][3]</sup>. Before action execution, the motor system builds a prediction of its sensory consequences. The cortical connectivity between the frontal areas that plan voluntary movements and parietal areas that monitor outcomes is fundamental in retrospectively (i.e., after action execution) assessing the prediction–outcome match <sup>[2]</sup>. In case of alterations in the spatial and temporal contiguity between action and effect, the mismatch between predictions and actual outcomes would push the individual to search for an external cause of the event <sup>[4][5]</sup>. On the other hand, processes in the frontal cortex occurring before the initiation of action prospectively operate and underlie the subjective experience of one's own voluntary action <sup>[2]</sup>. We sometimes consider ourselves authors of an event, even without being directly responsible for it. Whenever an event turns out to be in line with one's intentions, there is a strong tendency to interpret it as self-generated <sup>[1]</sup>. For example, we push the crosswalk light buttons because we expect this to reduce the wait until the green light comes on. When the traffic light turns green, after variable and even prolonged time delays, we attribute this event to our action of pressing the button, even though there is no evidence of our role in turning the light green.

Agency arises from both implicit and explicit processes and can be distinguished into feeling of agency (FoA) and judgement of agency (JoA) <sup>[6]</sup>. FoA comes from implicit sensorimotor representation and can be considered the preconceptual component of agency. Altering the spatial or temporal contiguity between action and effect significantly disrupts this level of agency <sup>[7]</sup>. On the other hand, JoA consists of explicit recognition of oneself as the cause of an event. It is influenced by personal beliefs, contextual and social information <sup>[6]</sup>, and is insensitive to factors of spatial and temporal contiguity between the action and its outcome <sup>[7]</sup>. Different measures have been employed in literature to capture explicit and implicit agency. Self-reported measures of explicit agency come from the person's answer to the question, "Did you do that?" <sup>[2]</sup>. One of the most widely used implicit measures of agency is the intentional binding effect that refers to the tendency of agents to perceive the time interval between a voluntary action and a sensory stimulus as

shorter than it actually is. More specifically, the onset of the voluntary action is reported later in time and awareness of the sensory feedback is temporally anticipated <sup>[8]</sup>.

At the behavioral and motor level, the control-based response selection framework (CBRS) proposed that producing effects that are perceived as self-caused facilitates action selection and execution <sup>[9]</sup>. Indeed, the way we plan and control our movements is affected by several cognitive and sensorimotor features of actions <sup>[10]</sup>. We select response options more frequently and faster when they are associated with a higher probability of producing an effect, compared with no effect; thus, we are motivated and facilitated by having control <sup>[9]</sup>. At an implicit level, agency impacts motor parameters of actions (i.e., reducing reaction times), relies on action–effect temporal contingency (i.e., is disrupted by action–effect delays), and goes beyond individuals' explicit judgements of agency <sup>[9]</sup>. In everyday life, it could be that fluently selecting an action makes it more likely that our intentions will be realized, and expected outcome achieved. Individuals reported greater perceptions of control over a given event when prime stimuli allowed for more fluent and immediate action selection <sup>[11]</sup>. According to this, habitual actions are accompanied by a strong sense of control and could therefore be sustained by agency mechanisms. Intriguingly, the motor system may be insensitive to abstract representations of the valence of an effect (i.e., receiving a positive or neutral effect does not change motor parameters of action) <sup>[12]</sup>.

### **3. A Developmental Journey**

The mechanisms underlying and associated with agency and reward are subject to specialization and tuning throughout child development and may undergo atypical trajectories under specific neurodevelopmental conditions. Decades of research demonstrated that infants learn through embodied sensorimotor contingencies, thus using their bodies to produce effects in the external world <sup>[13]</sup>, with behavioral and neural markers of action–effect binding at around 3 months of age. After disrupting the action–effect contingency of infants' movements, some of them showed EEG mismatch negativity and a reduction in their movement behavior, which respectively underpin violation of expectation and behavioral extinction, potentially related to reduced agency <sup>[14]</sup>. Moreover, infants at around 9 months of age are aware of the association between actions and effects, thus responding faster to events that they had previously actively produced than to action-independent events <sup>[15]</sup>. Other authors question the appropriateness of these methods for studying the sense of agency in preverbal children, and point out that the mere association between stimulus and response is not sufficient to constitute a minimal sense of agency, which should be distinguished from reinforced learning <sup>[16]</sup>. Additional studies have investigated implicit agency in school-aged children, who showed reduced temporal binding than adults <sup>[17][18]</sup>. Other authors found adult-levels of intentional binding in children from 6 years of age <sup>[19]</sup>.

Notably, the threshold for detecting temporal biases between action and consequence may change during development. From the age of 4 to 15, there is a progressive decrease in the minimum temporal delay necessary for a person to be aware of the action-effect alteration <sup>[20]</sup>. Overall, the temporal interval within which multisensory stimuli are likely to be perceptually bound (namely, multisensory temporal binding window) gradually decreases up to adolescence <sup>[21]</sup>. The time window for intentional binding seems to be associated with manual dexterity, and is extended in children with developmental coordination disorder (DCD) <sup>[5]</sup>. In this population, reduced implicit agency was associated with depressive tendencies, thus contributing to the children's well-being <sup>[5]</sup>. Contradictory findings come from adolescence, whereby researchers have found both reduced implicit agency compared with children and adults <sup>[22]</sup>, and greater experience of implicit agency during mid-adolescence, which was mediated by a neural oversuppression of action outcomes (sensory attenuation) and over-reliance on motor preparation (late readiness potential) <sup>[23]</sup>. It can be concluded that different sensitivities in detecting temporal biases could contribute to differences in implicit mechanisms of agency and impact the broader dimensions of child development and well-being.

As for the explicit judgment of agency, school-aged children and adults seem to be equally accurate in estimating their control over an event as a function of action–outcome congruency <sup>[24]</sup>. However, top-down processes, such as metacognition, change across the lifespan and affect children's explicit agency up to later childhood. In particular, the outcome valence influences our causal attributions. A self-attribution bias that over-attributes positive outcomes to oneself and negative outcomes to external factors is pervasive in the general population but greater in children than adults <sup>[24]</sup>. For instance, children from 8 to 10 years old accurately judged a negative outcome as not self-caused, but believed they were responsible for positive outcomes that they did not actually cause <sup>[25]</sup>. Overall, school-aged children are happier when allowed to make choices among options, rather than being given only one option, thus being motivated by explicit agency <sup>[26]</sup>. However, in cases of a negative outcome, children's emotions may worsen after self-determined choices compared with having no choice <sup>[26]</sup>. Moreover, children's academic success is positively associated with their judgment of control, or explicit agency (i.e., believing that they know how to influence outcomes of success and failure in their academic life) <sup>[27]</sup>. Crucially, explicit agency is built on high-level cognitive processes (e.g., expectations, beliefs, and attitudes), which may be affected by some neurodevelopmental disorders. For instance, people with attention deficit and

hyperactivity disorder (ADHD) show reduced self-attribution bias [28], which plays a fundamental role in their well-being [29]. Moreover, children with ADHD may be more sensitive to their action outcome valence, with an enhanced sensitivity to positive and negative outcomes and underlying atypicalities in neural reward circuits [30][31][32].

The nature of rewards may constitute a different degree of motivation depending on the context and the individual characteristics and age of the actor. Toddlers more frequently orient their attention toward social stimuli compared with non-social stimuli that respond to their gaze [33]. Later in childhood, monetary incentives may have stronger reinforcing value compared with social incentives when children perform cognitive tasks [34]. Finally, adolescence may be a critical period whereby social rewards are particularly valued [35]. However, different personality traits seem to mediate the extent to which a child benefits from different types of rewards, with higher reward-seeking tendencies and social skills being respectively related to higher benefits from monetary or social rewards [34].

In conclusion, both the feeling of control arising from agency and the positive valence of outcomes drive children's actions. However, these mechanisms undergo developmental trajectories and specialize with age, potentially playing a role in atypical development.

## **4. Agency and Reward in Autism**

The perception–action processes on which the sense of self is rooted are particularly affected by autism spectrum disorder (ASD). This neurodevelopmental condition is diagnosed from the very first years of a child's life based on persistent and pervasive deficits in social communication and social interaction, as well as restricted and repetitive patterns of behaviors, interests, or activities [36]. Restrictive and repetitive behaviors may come along with atypical action selection processes, among which agency and reward play a crucial role. Understanding these mechanisms in ASD may shed light on how to promote learning, volition, and self-determination.

Using implicit measurements of agency, some researchers found differences in the autistic adult population. Participants were asked to press the spacebar whenever they wanted. Sensory feedback was presented after a variable temporal delay (i.e., 250, 450, or 650 ms), and participants were required to estimate the delay. Despite being accurate overall in their time perception, autistic adults showed reduced intentional binding compared with controls [37]. Another study on explicit agency in ASD showed that high-functioning autistic and neurotypical adults were equally able to judge whether a visual effect was self-caused or not [38]. Participants were asked to move a joystick and its cursor representation on a screen. The authors manipulated the degree of correspondence between participants' actual movement and the visual feedback (i.e., the cursor movement). Half of the trials delivered synchronous visual feedback of participants' real movement. The other half of the trials showed pre-recorded cursor movements from a randomly selected previous trial performed by the same participant. When analyzing explicit measures of agency by asking the question, "Did you perform the action on the monitor?" no significant differences emerged between the two groups [38]. These findings suggest a dissociation between explicit and implicit agency in people with ASD [39]. Although these considerations are based on very few studies and further investigation is needed, it can be hypothesized that people with ASD experience a reduced sense of implicit agency, thus being less motivated by the sense of control that accompanies voluntary actions and self-caused events. To the best of the researchers' knowledge, there are no previous studies investigating agency in children with ASD, thus preventing us from understanding the developmental trajectory leading to any atypicalities can be found in adult populations.

To understand agency in ASD despite the limited research on ASD populations, we can take a hint from studies on other clinical populations that have atypicalities in common with ASD. For instance, developmental coordination disorder (DCD) entails early-emerging persistent difficulties in the acquisition and execution of coordinated movements [36]. Motor coordination difficulties seem to be negatively associated with socio-affective abilities, thus being a potential bridge between DCD and ASD [40]. The sensory processes underlying explicit agency have been found to be different in children with DCD compared with neurotypical ones. Children were asked to make an action that would cause an effect after a random temporal delay, and to judge whether the effect was self-caused. The time window for agency was extended in children with DCD, negatively associated with manual dexterity and positively related to depressive symptoms [41]. As multisensory temporal binding windows are enlarged in ASD [42], this could also impact the emergence of implicit agency. Looking at the cognitive mechanisms of agency, some interesting insights come from attention deficit hyperactivity disorder (ADHD) research. The cognitive mechanisms underlying the inattentiveness and impulsive symptoms that characterize ADHD may also affect agency. For example, a self-attribution bias that over-attributes positive outcomes to oneself and negative outcomes to external factors is pervasive in the general population, but greater in children than adults, and reduced in ADHD [28]. However, no difference in self-attribution bias was found in ASD [43], suggesting that higher-order cognitive mechanisms of explicit agency may be unaffected.

Extensive literature has investigated the motivation from reward in people with ASD. Neuroimaging evidence showed that when anticipating monetary reward, NAc activity was reduced compared with neurotypical individuals, whereas when perceiving the actual reward, hyperactivation of VMPFC was observed, suggesting reduced motivation from rewards [44] [45]. Additional evidence suggests that higher autistic traits are associated with enhanced neural activity related to reward anticipation, but do not modulate reward consumption [46]. Reduced motivation from rewards has been particularly found with respect to social rewards. Among children with ASD, researchers found reduced neural responses of VS to social rewards [47], an attenuated vmPFC response to a presentation of favorite faces [48], and reduced activation of frontostriatal networks during socially rewarded learning [49]. However, there was contradictory data on this, leaving it an open debate. Some studies have found decreased amygdala activation in children with ASD [45], whereas others have reported increased amygdala activation during social reward anticipation in adults with ASD [50]. These results suggest an atypical developmental trajectory in amygdala reactivity to social incentives [51]. Overall, the social motivation account of ASD that hypothesizes reduced motivation from social rewards seems to be supported by just over half of the studies in the literature [52], which leaves many open questions about individual differences and heterogeneity. Moreover, it is interesting to note that atypicalities in the reward system are also present in other neurodevelopmental conditions or psychopathologies, and may constitute a trans-diagnostic feature [53].

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