The Aberrant Contribution of the Mirror Neuron System in Autism Spectrum Disorder

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The mirror neuron system (MNS) is a recently discovered system, existing largely in the inferior frontal gyrus and inferior parietal lobe, which is activated when one either perceives or performs an action, and thought to be important for social interaction. Some have linked atypical MNS activation with autism spectrum disorder (ASD) (e.g., Rizzolatti and Fabbri-Destro, 2010). This literature review was conducted to attempt to determine if deficits in the MNS contribute to the social and communicative deficits seen in ASD. Many electroencephalogram (EEG), functional magnetic resonance imaging (fMRI), and behavioral studies indicate that individuals with ASD improperly engage their MNS in social interaction, which results in social and communicative deficits. Other fMRI, EEG, and behavioral studies indicate that individuals with ASD have a functional MNS. Recent experimental work has also shown that the functioning of the MNS is heavily subject to top-down influences, and that individuals with ASD have abnormal top-down processing. This review therefore concludes that individuals with ASD have a functional MNS that is irregularly inactive during social interaction due to abnormalities in the connectivity of their automatic top-down processing systems, and this inactivity contributes to the social deficits seen in ASD.


Keywords: ASD; MNS; Top-Down Processing; fMRI; EEG

Introduction

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder with the essential features of impaired or notably abnormal development in social interaction and communication, and a noticeably reduced range of activities (American Psychiatric Association, 2000). ASD is complex in that it involves deficits in both cognitive and social processes, and these deficits may be present in a variety of degrees. These features lead to broad impairment and can have severely debilitating consequences. While outcomes for individuals with ASD have improved in recent years, most adults with ASD cannot live alone, do not have jobs, have few close friends, and are notably impaired in daily life (Howlin et al., 2004). Moreover, the rate of individuals with ASD per 1,000 individuals is growing substantially (Keyes et al., 2011). The societal impact of the growing rate of ASD is therefore quite large, and finding efficacious interventions for ASD is thus important.

Because of the complexity of ASD, developing a holistic intervention is difficult. Currently, the most effective form of treatment for ASD is an optimized medication regime that allows educational interventions to have their greatest influence (Myers et al., 2007).
However, medicinal regimes are often tailored to symptoms; there is no single medication that improves the functioning of the core disabilities of ASD (Myers et al., 2007). Scientists are hopeful that ascertaining the neural basis of the social and communicative deficits in ASD will lead to novel treatments for the core disabilities of the disorder. Moreover, determining these neural correlates could provide an objective way to diagnose the disorder, which might allow the possibility of intervening earlier in the disorder. Early intervention is desirable in ASD; the younger brain is more plastic, and brain plasticity influences how much an individual can respond to a treatment. Early intervention thus produces better outcomes (Dawson and Zanoli, 2008). The prospect of determining the neural basis of the social and communicative deficits in ASD brings with it the hope of finding an effective intervention for this otherwise challenging disorder. The purpose of this literature review is therefore to offer a theory about the neural basis of the social and communicative deficits of ASD based upon a survey of the relevant research, and thereby contribute to the possibility of developing an effective intervention for ASD.

Background

In the past, some researchers believed that the neural basis of ASD was a dysfunctional mirror neuron system (MNS) (e.g., Williams et al., 2001). Subsequent research has led to an abandonment of that belief because it cannot account for basic perceptual or neural abnormalities present in ASD that are entirely uncorrelated with the MNS (Behrmann et al., 2006; Martino et al., 2009). Nevertheless, much evidence indicates that the MNS of individuals with ASD is atypically dormant during social interaction, and many believe that abnormal engagement of the MNS in ASD is responsible for social and communicative deficits seen in ASD (e.g., Perkins et al., 2010; Rizzolatti and Fabbri-Destro, 2010; Gallese et al., 2011; Zhu et al., 2011; Enticott et al., 2012; Oberman et al., 2012).

An explanation of the MNS itself is necessary before examining the potential contribution of the MNS to the dysfunctions seen in ASD. While the existence of the MNS in humans has only recently been confirmed, researchers have presumed its existence since its discovery in Macaque monkeys over twenty years ago (Pellegrino et al., 1992; Mukamel et al., 2010). Although the MNS is primarily found in the inferior parietal lobe and inferior frontal gyrus (IFG), its presence and activity in humans seems to be quiet diffuse (Schippers and Keysers, 2011).

Mirror neurons have the unique property of firing both when one performs a given action and when one merely observes another individual performing that same action (Cattaneo and Rizzolatti, 2009). Mirror neurons appear to be the basis of imitation learning, as one area of the MNS combines disparate activities from various parts of the MNS and conveys that information to the prefrontal cortex,—believed to be the location of executive function (Rizzolatti and Craighero, 2004). Most researchers understand mirror neurons to allow an understanding of the actions of others and the intentions behind those actions (Cattaneo and Rizzolatti, 2009). Because mirror neurons fire when one merely observes an action, some researchers have claimed that mirror neurons are necessary for one’s theory of mind (ToM) — that is, the understanding that other individuals have beliefs and desires, and that others’ beliefs and desires may differ from one’s own (Williams et al., 2001).

The MNS is critically involved in typical social interactions. Experiments have shown that the MNS is necessary — though possibly not sufficient — for empathy, intention understanding, and ToM (Schulte-Rüther et al., 2007; Schippers et al., 2010; Spunt et al., 2011). The MNS is also responsible for the synchronization of brains during social interaction (Dumas et al., 2010). Defective activation of the MNS, then, should result in social deficits.

Because of the aforementioned properties of the MNS, modulation of the MNS has potential clinical applications. Since mirror neurons fire when an individual merely observes an action, researchers have noted that mirror neurons have promising restorative clinical applicability in the use of video therapy rehabilitation for individuals who have suffered
a stroke (Nedelko et al., 2010). Additionally, some have proposed hypothetical treatments for ASD based upon research showing atypical MNS activation in ASD (Zhu et al., 2011).

This literature review surveys research concerning the MNS and its contributions to the social and communicative deficits of ASD. A review of the literature indicates that while the MNS is typically dysfunctional in ASD, certain experimental conditions indicate that individuals with ASD have a functional MNS. To explain this paradox, it is noted that the MNS is subject to top-down processing, and that automatic top-down processing is impaired in ASD. This impairment seems to lead to a reduced involvement of the MNS in social interaction, thus producing the social and communicative deficits seen in ASD.

**Review**

**MNS Improperly Employed in Social Interactions of ASD Individuals**

There is a prodigious amount of research concerning the MNS and ASD, with much research appearing to confirm the idea that in ASD the MNS is atypically recruited in social interaction. Martineau and colleagues (2008) presented a variety of stimuli thought or known to involve the MNS to 14 children with ASD and 14 age- and gender-matched, healthy individuals. The study examined the participants’ electroencephalogram (EEG) activity — a measure of the electrical activity of the neocortex — while the study participants observed the experimental stimuli. The researchers found a desynchronisation in the EEG of healthy individuals during the observation of the movements of others — mainly in the theta 1 frequency band around the left central, parietal, and posterior temporal regions, which are involved in the MNS. By contrast, individuals with ASD did not display this same desynchronisation when they watched the same movements. Oberman et al. (2005), in another EEG study, examined mu wave suppression in healthy individuals and individuals with ASD. In healthy individuals, mu wave suppression occurs when one performs an action and when one watches another perform that action, and because of this, many consider mu wave suppression a valid indicator of MNS function (Muthukumaraswamy et al., 2004). Confirming this, functional magnetic resonance imaging (fMRI) and repetitive transcranial magnetic stimulation (rTMS) have linked mu wave suppression to areas of the brain related to the MNS (Arnstein et al., 2011; Keuken et al., 2011). In Oberman and colleagues’ study (2005), healthy individuals showed the expected mu wave suppression when watching someone perform an action and performing that action themselves, but individuals with ASD showed no mu wave suppression. Bernier and colleagues (2007) replicated the overall findings of Oberman and colleagues’ study.

Oberman et al. (2012) recently conducted an analytical study in which they incorporated data from four studies that used EEG to measure mu suppression and used a nearly identical methodology. Their study attempted to ascertain whether MNS functioning improved with age for individuals with ASD, as some studies have reported (e.g., Bastiaansen et al., 2011). Oberman and colleagues’ study was the first to compare the developmental trajectory of mu suppression in both healthy individuals and individuals with ASD. They found that while mu suppression increases with age for both individuals with ASD and healthy individuals, the MNS of individuals with ASD is consistently underactive in comparison to individuals without ASD. Oberman et al. (2005) therefore showed that MNS functioning does not improve with age for individuals with ASD, and the findings of previous studies reflected a diagnosis-independent, age-related change in mu suppression. There is therefore much evidence from EEG studies that supports the idea that individuals with ASD do not recruit their MNS in a typical manner during social interaction.

This evidence is not alone. Enticott and colleagues (2012) recently carried out a study that supports the idea that abnormal MNS involvement correlates positively with social impairment. Enticott and colleagues used rTMS to examine the functioning of the MNS in individuals with ASD by measuring corticospinal excitability after applying rTMS, as...
nominally un invasive way to disrupt an area thought to contribute to the function that is under investigation (Fadiga et al., 2005). Enticott et al. (2012) applied rTMS to the left primary motor cortex and used an electromyogram (EMG) to record the activity of index finger and thumb muscles while individuals with and without ASD observed a short series of video clips displaying a static hand, pantomimed movements, or transitive hand movements. In this study, individuals without ASD displayed MNS activity solely during the observation of transitive hand movements. However, individuals with ASD failed to achieve significant MNS activity as measured by reduced motor corticospinal excitability. More importantly, evidence of MNS activity correlated inversely with social functioning in individuals with ASD.

Cook and Bird (2011), in a recent behavioral study, also provided evidence that the MNS is atypically employed by individuals with ASD in social interaction. They showed that individuals with ASD do not process social cues in the modulation of imitation as individuals without ASD do. Other behavioral measures indicate abnormal employment of the MNS in individuals with ASD. Boria and colleagues (2009) sought to determine whether individuals with ASD could understand the goals and intentions of the motor acts of another. They conducted two experiments on individuals with and without ASD. In both experiments, individuals with ASD had no difficulty in determining the goal of an observed motor act. However, individuals with ASD showed a significantly higher error rate in correctly stating the intentions of the agent — given the context — that executed the observed motor act in only one of the two experiments. Specifically, individuals with ASD showed no behavioral deficiency in naming the intentions of agents performing motor acts if there was functional information involved — that is, an object’s typical use was apparent. However, if there was no functional information present, only motor information, individuals with ASD showed a significantly higher error rate in their responses. This shows that individuals with ASD do not translate individual motor actions into a single global action without additional information, indicating disconnection between their MNS and other brain regions.

EMG studies also seem to support the concept that in ASD the MNS is not typically employed in social interaction. Cattaneo and colleagues (2007) performed a study that seems to confirm this idea. The researchers examined the activity of the mylohyoid muscle of individuals with and without ASD during the observation and execution — thus assessing activity of the MNS — of reaching for food and eating it or reaching for food and putting it into a container placed on his own shoulder. Cattaneo and colleagues (2007) also performed an experiment with hands and feet that used similar conditions to show that any effect they found was not due to specific details of the mouth. This study found that for individuals without ASD, the mylohyoid muscle activated when they observed or executed the actions of reaching for, grasping, and bringing the food to the mouth. However, Cattaneo et al. (2007) found that the mylohyoid muscle in individuals with ASD activated solely when these individuals observed or executed the movement of bringing the food to the mouth. It should be noted, however, that a recent study performed by Pascolo and Cattarnussi (2012) they were unable to replicate the aforementioned findings of one of the experiments conducted in Cattaneo and colleagues’ 2007 study. Pascolo and Cattarnussi (2012) identified two major methodological issues in the Cattaneo et al. (2007) study that possibly influenced their results, namely, the presence of several experimenters during the tests and the distance between the subjects and the food. When controlling for these methodological issues, Pascolo and Cattarnussi (2012) found that individuals both with and without ASD first grasped the food and then opened their mouths. While methodological issues may have skewed Cattaneo et al.’s (2007) results in one of their three experiments, the other two experiments reported in their study still indicate that in individuals with ASD the MNS is activated abnormally in social interaction.

Many fMRI studies support the notion that individuals with ASD have an abnormally functioning MNS. Dapretto and colleagues (2005) examined individuals both with and
without ASD using fMRI while they imitated and observed emotional expression. Dapretto et al. (2005) found that, while individuals with and without ASD performed these tasks equally well, individuals with ASD did not show the activity in the IFG — reflective of mirror neuron activity — that individuals without ASD did. Importantly, Dapretto et al. (2005) found that symptom severity in ASD correlates inversely with the level of activation of the IFG in individuals with ASD.

Martineau and colleagues (2010) used fMRI to examine seven right-handed high-functioning male individuals with ASD and eight age-matched, education-matched, healthy control subjects while they observed and executed hand movements, and this study compared these results to the resting states of the individuals. This study likewise found atypical activation in the IFG in individuals with ASD. However, in contrast to Dapretto and colleagues (2005), who found no activity in the IFG of children with ASD, Martineau and colleagues (2010) found that, when observing a task, there was greater activation in the IFG of individuals with ASD than there was in controls. To explain the differential activation in the IFG, one notes that the tasks involved in these experiments differ — Dapretto et al. (2005) had individuals with ASD observe and imitate emotions, whereas Martineau et al. (2010) had individuals with ASD observe and imitate hand movements only. Individuals with ASD may have deemed the task of the Martineau et al. (2006) experiment relevant, while failing to deem the task of Dapretto et al. (2005) relevant, and neural activity would therefore differ.

Williams and colleagues (2006) examined 16 right-handed adolescent or adult males with ASD and 15 healthy, control individuals. Williams and colleagues presented the participants in this experiment with three types of stimuli previously shown to elicit a response from the MNS and then asked them to perform an action in response to these stimuli while examining them with fMRI. The results of this study were that individuals with ASD failed to show the same activation in the anterior intraparietal sulcus — a region previously thought to be part of the MNS — that individuals without ASD showed. Because the MNS is crucial for social interaction and individuals with ASD do not show activation of their MNS during social interaction, this suggests that abnormal involvement of the MNS produces the social and communicative deficits found in ASD.

**Individuals with ASD Have a Functional MNS**

While much evidence shows that the MNS is not typically recruited in individuals with ASD during social interaction, much evidence also seems to indicate that individuals with ASD have a functional MNS. Hamilton and colleagues (2007) conducted a behavioral study on imitation and action in ASD. This study found that children with ASD were not impaired in performing imitative and gesture recognition tasks known to involve the MNS; individuals with ASD were, however, impaired on the ToM tasks. This study therefore seems to dissociate ToM from the MNS, and seems to show that individuals with ASD have a functional MNS.

EEG studies also provide evidence that individuals with ASD have a functional MNS. Fan and colleagues (2010) found that experimental conditions affected mu wave suppression more than ASD. This study used a similar paradigm as the previously mentioned EEG studies — twenty individuals with ASD and twenty individuals without ASD observed and executed hand movements while recorded with EEG — but found very different results. In particular, they found similar mu wave suppression in individuals both with and without ASD. In interpreting these findings by comparison to previously cited EEG studies, note that the studies that found a lack of mu suppression in individuals with ASD involved abnormal representations of intransitive movements, while this study featured normal representations of transitive movements. Fan and colleagues also found that while individuals with ASD showed evidence of normal MNS function, they failed to imitate the observed actions. This study therefore indicates that the imitative deficits seen in ASD are not due to a dysfunctional MNS.

Many fMRI studies also provide evidence that individuals with ASD have a functional MNS. Dinstein and colleagues (2010)
sought to ascertain whether the MNS of individuals with ASD showed the same repetition suppression (movement-selection adaptation) found in normal activity of the MNS of individuals without ASD. In this study, the researchers found normal MNS responses in the observation and execution of hand movements in individuals with ASD as well as normal movement-selection adaptation. Movement-selection adaptation in this case refers to the reduced responses of mirror neurons in the observation of repeated movements, which provides selectivity in distinguishing between different movements. Because movement selectivity is a defining characteristic of mirror neurons (Fogassi et al., 2005), the findings of Dinstein et al. (2010) indicate that individuals with ASD have a functional MNS.

Marsh and Hamilton (2011) investigated the relationship between mentalizing — that is, assessing intentions through ToM — and mirroring in individuals with ASD. To test this, they examined individuals with and without ASD using fMRI while these individuals observed rational actions (defined as actions that achieve their goals efficiently given the constraints of their environment), irrational actions (actions that are inefficient), and simple moving shapes with no biological form. Rational actions activate the MNS, whereas irrational actions activate mentalizing networks without prompt. When watching rational actions in contrast to moving, non-biological objects, Marsh and Hamilton (2011) found activation in individuals with and without ASD in the parietal lobe, indicative of MNS function, and they found repetition suppression in both groups with no group differences in this respect. However, Marsh and Hamilton found that while individuals with and without ASD both showed activation in the anterior intraparietal sulcus when observing irrational actions, when individuals without ASD watched irrational actions they exhibited a differential activation in the medial prefrontal cortex that was absent in individuals with ASD who watched irrational hand movements. The findings of this study thus indicate that, while individuals with ASD are deficient in ToM, they have a functional MNS.

Spengler and colleagues (2010) queried the neural correlates of the symptoms of echopraxia in ASD, which involves excessively imitating behaviors and disregarding the meaning of the things imitated, and whether this behavior correlated with mentalizing ability. To study this, Spengler et al. (2010) used a complex, imitation-inhibition task that created an interference effect. In particular, they instructed participants to react immediately to a number that appeared after a blank screen, with instructions to lift a certain finger given for each number, and the screen displayed concurrent hand actions that could be either congruent or incongruent with the number — and thus instructions for action — given. Spengler et al. (2010) assessed mentalizing through the comprehension of strange stories as well as by asking for explanations of what occurred during animations shown while individuals were examined with fMRI. The researchers found that individuals with ASD were significantly less able to inhibit imitation during incongruous tasks than the control group. Moreover, difficulties in inhibition positively correlated with deficits in mentalizing and its correlated brain regions — namely, the medial prefrontal cortex and temporoparietal junction. These data therefore indicate individuals with ASD do not have a deficit in the MNS itself but instead have a deficit in an automatic mentalizing ability, which results in improper use of the MNS.

Schulte-Rüther and colleagues (2011) sought to ascertain whether there are any behavioral or neurological differences in empathizing for individuals with ASD. To test this, they used fMRI to examine fourteen adults with ASD and fourteen adult controls. The researchers explicitly instructed the participants to empathize with the images of persons on the screen, either by “feeling into” the emotional state of the person on the screen (other-task) or by explaining the emotions they themselves feel after seeing the emotional state of the person on the screen (self-task). Behaviorally, the groups showed no significant difference in their ability to empathize as measured by reports of emotions given on the other-task and the self-task. Moreover, fMRI scans showed activation of both the MNS and mentalizing brain regions for both groups, and there was no significant functional difference between groups for either the other-task or the self-task. This study thus
provides evidence that individuals with ASD are able to use their MNS, empathize, and mentalize when they intentionally attempt to. In summary, many data from EEG, EMG, fMRI, and behavioral studies show that individuals with ASD have a functional MNS.

Top-Down Processing Influences the MNS

Presented were various conflicting data concerning the contribution of the MNS to the social and communicative deficits seen in individuals with ASD. These data are explicable by the notion that individuals with ASD have a functional MNS that is not subjugated to automatic top-down processing — as it is typically — resulting from abnormal connectivity between the MNS and higher brain centers. This lack of control of the MNS by automatic top-down processing would result in the lack of use of the MNS, which would lead to the social and communicative deficits seen in ASD.

Contrary to intuition, activation of the MNS does not require observations of a living organism; the movements of artificial objects can activate the MNS as well. Engel and colleagues (2008) used fMRI to show that nonbiological moving objects could activate the MNS. However, they showed that the activation of the MNS from these movements is strongly dependent upon an individual judging whether human hands could perform the movement pattern. This therefore indicates that bottom-up processing alone is not sufficient to activate the MNS. Instead, these data show that top-down processing plays an integral role in the activation of the MNS — especially that individuals consider actions as performable by other humans. The findings of Engel et al. (2008) are complemented by fMRI data collected by Schippers and Keysers (2011). The latter found that significant modulation of the activity of the MNS occurs when individuals are instructed either to passively observe actions or to actively guess the intentions of those actions. Spunt and colleagues (2011) further complemented these data by showing that attention modulates the activity of the MNS. Spunt et al. (2011) showed that, while the MNS is active when individuals watch an action and are asked to describe why the individual is doing that action, activity in the MNS increases when individuals are asked to describe what the individual is doing and increases further when they are asked to describe how the individual does an action. These fMRI data indicate that the MNS is a dynamic system influenced by both bottom-up and top-down processing.

Top-down processing influences the activity of the MNS in actual social interaction, as shown by Dumas and colleagues (2010). In their studies, they instructed two participants to imitate each other at will while measuring the participants’ EEG. The researchers found that the MNS played a crucial role in synchronizing the brains of individuals as they interacted with one another. The study found no synchronization between the brains of participants when they were not told to imitate each other. Moreover, the researchers found that during this imitative synchrony, higher frequency bands were asynchronous, reflecting who was leading the imitative gesture and who was following it. This study therefore shows, with high ecological validity, that top-down processing influences the MNS in that the perception of being a leader or a follower regulates MNS activity.

Naem and colleagues (2012) recently showed that top-down processing influences the MNS in coordinated social interaction. To show this, they instructed individuals either to intentionally maintain their own rhythmic movements regardless of the other’s rhythm or to adapt to each other. In the interactions of these participants, the researchers found that top-down processing modulates mu suppression directly, and that there is a nearly constant top-down modulation of mu suppression in coordinated social interaction. There is thus much persuasive evidence that top-down processing influences the MNS.

Individuals with ASD Have Deficits in Top-Down Processing

The evidence in this paper indicates that individuals with ASD have deficits in top-down processing, and the literature supports this indication (Neumann et al., 2006; Loth et al., 2010; Loth et al., 2011). This might seem to conform to the view that the social and
communicative deficits seen in ASD are due to impairments in top-down processing as a whole (Spengler et al., 2010). Unfortunately, the findings of Schulte-Rüther et al. (2011) — which showed that individuals with ASD can modulate MNS activity if they intentionally attempt to do so — do not support this conclusion. However, there are two types of top-down processing (Balluch and Itti, 2011). One type of top-down processing is automatic, and it modifies perceptions, is pervasive, and develops through experience-dependent plasticity (Balluch and Itti, 2011).

Individuals with ASD seem to have a compromised system of automatic top-down processing due to abnormalities in the mechanisms of experience-dependent plasticity that would result in the abnormalities commonly observed in their MNS due to its relative disuse. Bolstering this notion, individuals with ASD have reduced functional connectivity within typical neural networks, which reflects faulty synaptic stabilization and axon myelination, but increased functional connectivity outside of typical neural networks, which reflects diminished pruning (Müller et al., 2011). This concurs with a study done by Hadjikhani and colleagues (2006). Hadjikhani et al. (2006) found gray matter decreases in individuals with ASD in areas of the brain that are involved in the MNS, and also found that the greater the decrease in gray matter in these areas, the greater the severity of the disorder. Further buttressing this idea, research has found resting-state abnormalities in functional connectivity within areas of the brain related to the MNS (Paakki et al., 2010) as well as diminished frontal-parietal functional and anatomical connectivity (Schipul et al., 2011). Notably, symptom severity in ASD correlates with diminished connectivity between these disparate brain regions (Cheung, 2009).

Together, these data indicate that individuals with ASD have deficits in automatic top-down processing. This deficit results in an underused and underdeveloped MNS, which dominoes to difficulties in social interaction. This idea can explain the data that show that individuals with ASD have a functional MNS that is not activated automatically (Oberman et al., 2009) the fact that familiarity modulates MNS function in individuals with ASD (Oberman et al., 2008), and the findings of previously cited studies showing MNS activity and those that did not find the activity under the same conditions. By intentionally deeming some actions as socially important — for example, by being instructed to imitate normal representations movements usually deemed important — individuals with ASD can override their automatic top-down processing system and activate their MNS. However, if individuals with ASD do not intentionally deem stimuli as relevant, their abnormal automatic top-down processing continues and this results in either an atypical or a negligible recruitment of the MNS. Nonetheless, these experiments still provide evidence that individuals with ASD atypically employ the MNS in social interaction, which produces social deficits.

**Conclusion**

Individuals with ASD appear to have a functional MNS that is abnormally unemployed in social interactions due to connectivity abnormalities in automatic top-down processing systems. Because the MNS is integral in social interactions, this atypical use of the MNS in ASD produces social impairments. Whether interventions that encourage functioning of the MNS in individuals with ASD will prove beneficial therefore remains open, and this avenue of research may prove valuable, especially if this research addresses the functional connectivity of the MNS in automatic top-down processing.

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