

*Investigation of Executive Functioning in Autism Spectrum Disorder*

Eleni Alexandropoulou, College for Humanistic Science - ICPS, Athens, Greece

---

Autism spectrum disorder (ASD) is a complex brain disorder characterized by impairments in some executive functions. Executive functions play an important role in individuals' daily life, due to the fact that they support social communication and interaction. This paper investigates which executive functions are impacted and how on people who suffer from ASD. In many cases, cognitive theories and research links autism to dysfunction of the prefrontal cortex and more specifically to impairments to "cool" components of EFs, such as inhibition and shifting, while damages to "hot" components of EFs are still not clearly defined. However, studies reveal that neurotransmitters like dopamine seem to have an impact on other brain areas that are involved in autism including the amygdala, cerebellum, and parietal lobe.

---

**Definition of Autism Spectrum Disorder (ASD)**

Autism spectrum disorder (ASD) is a complex neurodevelopmental disorder characterized by impairments in some abilities of the executive functions, including social communication and interaction, as well as restricted interests and repetitive behaviors, across the lifespan (American Psychiatric Association, 2013; Carotenuto et al., 2019; Demetriou, DeMayo & Guastella, 2019). A person can be diagnosed with ASD, at the age of 18 months, but often, most patients are not diagnosed until the age of 5 years (Casanova, 2014). Although once, ASD was considered a rare disorder, research shows that, during the last years, the male to female ratio is close to four to one (Demetriou, et al., 2019; Loomes, Hull & Mandy, 2017). That is the reason why, there is an increasing interest in understanding gender differences and

how the brain functions, in people with ASD but still everything is not very clear. Despite the fact that there are genetic and neurological factors, which may trigger ASD, neurocognitive functions seem to play also an important role (Demetriou et al., 2018). Evidence also shows that, in individuals with ASD, abnormalities in frontal and cortical areas of the brain are reported (Demetriou et al., 2018; Schmitz, Rubia, Daly, Smith, Williams, & Murphy, 2006). The aim of this paper is to explore the executive function performance in ASD, from both cognitive and physiological perspectives.

## **Definition of Executive Functioning (EF) and its importance**

The term “executive” was first mentioned in 1973 by Pribram, when scientists started discussing matters on pre-frontal cortex functioning (Goldstein & Naglieri, 2014). Till nowadays, a lot of higher-level cognitive skills, - such as decision making, evaluative thinking, rule usage, and concept acquisition-, have been included under the umbrella of this term and many attempts have been made to provide a definition of Executive Functioning (EF). In simple terms, executive functions (EF) are really important in daily life, because due to them, individuals are able to communicate with others and to perform tasks. There are several cognitive models and theories, which have tried to shed some light on explaining the observed difficulties in ASD.

## **Cognitive Theories linked to Autism**

One of them is the executive dysfunction model, which links autism disorder to frontal lobe dysfunction and tries to explain behavioral problems in combination with the theory of mind (ToM) deficit hypothesis (Andreou & Skrimpa, 2020; Demetriou, et al., 2019; Hill, 2004). According to Hill (2004), the ToM deficit hypothesis suggests that dysfunction in one of the many components of the social brain may lead to an inability to interpret other aspects of communication. A child’s ToM development depends on the development of EFs. ToM allows individuals to evaluate and interpret the behavior of others, based on their own mental states. Children with ASD usually perform poorly in ToM tasks, such as in the false belief task (FBT), where social understanding is evaluated (Hill, 2004). Failure in ToM tasks reveals impairment in the inhibition of a prepotent response (inhibitory control) and in the working memory (Hill, 2004). Even if some autistic children, during ToM tasks, manage to generate thoughts, beliefs, and intentions, they are not able to execute these skills during social interaction (Andreou & Skrimpa, 2020). Moreover, individuals with ASD tend to repeat routine actions or tasks and show an inability to adapt to new non-routine ones. Difficulties are normally observed in

core EFs, such as planning, set-shifting, inhibition, self-monitoring, and working memory (Carotenuto, et. al, 2019; Demetriou, et al., 2019; Hill, 2004).

## **Supervisory Attentional System (SAS) & Executive Dysfunction Model**

The executive dysfunction model including diminished ToM in ASD seems to be in alignment not only with Luria's theory (1973), but also with the Supervisory Attentional System (SAS) model, a higher-order system located in the frontal lobe, which works together with other brain processes (Van der Linden, 2001). Luria was the first one, who suggested that damage to the frontal lobes and more specifically, in the prefrontal cortex is expected to cause problems in regulating or controlling mental activity and behavior such as motor coordination, disinhibition, and inability to follow specific instructions (Chan, Shum, Touloupoulou & Chen, 2008; Demetriou et al., 2019). At first, Norman & Shallice (1986) extended Luria's theory by introducing the SAS model. A few years later, the SAS model was also extended by Burgess and his colleagues. According to this cognitive model, there are two systems involved in daily tasks and behaviors. One system is responsible for handling and prioritizing the routine operation and the other one, for regulating the non-routine operation (Carotenuto et al., 2019; Chan, et al., 2008). Studies show that in patients, where there is a lesion in the frontal lobe, as in individuals who suffer from ASD, there is a loss in supervisory control, attention deteriorates, reaction time in shifting worsens and behavior is impacted (Chan, et al., 2008).

## **Study's Findings in children with Autism related to Executive Functioning**

The aforementioned findings seem also to be compatible with the outcome of a study, conducted in preschoolers with ASD, where dysfunction of the prefrontal cortex was detected (Carotenuto et al., 2019). The aim of the study was to evaluate EFs, between twenty-five children diagnosed with autism and twenty-five typically developed children, using the BRIEF-P tool (an ecological screening tool for monitoring EF at a very early age). Results showed that children with autism were facing difficulties in two EFs: inhibition and shift; autistic children were not able to inhibit a prepotent response and to change easily from one situation or task or behavior to another. Both of these EFs, regarded also as "cool" components of EFs, depend on the frontal lobe and particularly on the prefrontal cortex (Carotenuto et al., 2019; Demetriou et al., 2019).

## Cool & Hot EF

Neuroimaging techniques have lately increased their interest in the localization of the EF processes and on the role of the neurotransmitters involved. Studies show that “cool” EFs are located in the dorsolateral prefrontal cortex and “hot” EFs in the orbitofrontal prefrontal cortex (Demetriou et al., 2019). The classification between “cool” and “hot” EFs, has been made according to their functionality. The EFs, such as inhibitory control and shifting, which tend to malfunction in case of autism, are considered as “cool”, due to the fact that they do not involve any emotional control, while the EFs that involve emotional and personal interpretation, is defined as “hot” EFs (Chan, et al., 2008; Demetriou et al., 2019). The impairments monitored in ASD, are monitored as well, in individuals with lesions in the dorsolateral prefrontal cortex (“cool” EFs), who tend to suffer from lack of concentration, attention deficits and problems in shifting. On the other hand, individuals with lesions in the orbitofrontal prefrontal cortex (“hot” EFs), suffer from behavioral disinhibition and emotional lability (Bonelli & Cummings, 2007; Chan, et al., 2008). Damasio (1995) was the first one, who introduced the role of “hot” EFs in emotion and social behavior and their impact on “cool” EFs (Chan, et al., 2008). According to his somatic marker hypothesis, people will not be able to regulate their behaviors, if they cannot link those to an emotional-related somatic signal. Damasio noticed that this difficulty was happening to individuals with damage to the ventromedial frontal cortex, whilst some researchers nowadays, attribute this deficiency specifically to damage to the orbitofrontal prefrontal cortex. Unfortunately, research on “hot” components of EFs in autism, is still quite limited, therefore their impact is unclear.

## Which Neurotransmitters influence EF processes

As far as the involvement of neurotransmitters’ is concerned, dopamine (DA) seems to influence “cool” EF processes (inhibition & shifting) and to diminish “hot” EF constructs (Demetriou et al., 2019). Several PET studies, have reported abnormal DA levels in autism cases and seem to relate this neurotransmitter with several impairments such as motor problems, repetitive and stereotyped behaviors, seizures, attention deficits, and executive dysfunction (Kriete & Noelle, 2015). Researchers conclude that dopamine tends to have an impact on all the brain areas involved in ASD, including the prefrontal cortex, amygdala, cerebellum, and parietal lobe. Serotonin, on the other hand, seems to modify response inhibition in the orbitofrontal prefrontal cortex, which was also visible in PET studies performed in autistic boys, where abnormalities in the prefrontal cortex were detected (Chugani et al., 1997, as cited in Hill, 2004; Demetriou et al., 2019). It is worth mentioning that according to research, elevated whole blood sero-

tonin is reported in some autistic children, and there seems to be a link between serotonin transporter gene and autism risk, in boys (Muller, Anacker & Veenstra-VanderWeele, 2016). Last but not least, noradrenaline (NA) seems to affect arousal and attention (Demetriou et al., 2019), but a recent PET study, performed between adults with ASD and neurotypical controls, showed that in the ASD group, there was no association of noradrenaline transporter (NAT) binding with empathy and EFs. Researchers concluded that this outcome could be due to the heterogeneity of ASD and that noradrenaline (NA) may impact the clinical characteristics of autism (Kubota et al., 2020).

## Conclusion

As it seems, autism is an extremely heterogeneous developmental disorder with varied behavior and that is the reason why studies often report controversial findings. ToM is considered the most important disabling feature in ASD since as a multidimensional approach, it incorporates a great variety of cognitive and neurobiological aspects. Studies focus mainly on difficulties in inhibitory control and shifting, the so-called “cool” constructs of executive functions, which are located in the dorsolateral prefrontal cortex. As far as the “hot” components are concerned, there is still little research; therefore, there is no much evidence to share. Current literature reveals a plethora of studies in ASD, that involve dysfunction in the frontal lobe and prefrontal cortex, which is in alignment with many cognitive models and theories. But when neurotransmitters are involved, other brain regions such as the cerebellum and parietal lobe seem to play also a role in behavioral changes in ASD. Especially for dopamine, studies reveal its involvement in several impairments. However, despite the fact that studies in autism are conducted constantly during the last years, trying to investigate the cognitive differences, what still remains a mystery, is the relationship between biology and behavior.

## References

- [1]. Andreou, M., & Skrimpa, V. (2020). Theory of mind deficits and neurophysiological operations in autism spectrum disorders: A review. *Brain Sciences*, 10(6), 393.
- [2]. Bonelli, R. M., & Cummings, J. L. (2007). Frontal-subcortical circuitry and behavior. *Dialogues in clinical neuroscience*, 9(2), 141.
- [3]. Carotenuto, M., Ruberto, M., Fontana, M. L., Catania, A., Misuraca, E., Precenzano, F.,...& Smirni, D. (2019). Executive functioning in autism spectrum disorders: A case-control study in preschool children. *Curr Pediatric Res*, 23, 112-6.

- [4]. Casanova, M. F. (2014). The neuropathology of autism. *Handbook of Autism and Pervasive Developmental Disorders, Fourth Edition*.
- [5]. Chan, R. C., Shum, D., Touloupoulou, T., & Chen, E. Y. (2008). Assessment of executive functions: Review of instruments and identification of critical issues. *Archives of clinical neuropsychology, 23*(2), 201-216.
- [6]. Demetriou, E. A., DeMayo, M. M., & Guastella, A. J. (2019). Executive function in autism spectrum disorder: history, theoretical models, empirical findings, and potential as an endophenotype. *Frontiers in psychiatry, 10*, 753.
- [7]. Demetriou, E. A., Lampit, A., Quintana, D. S., Naismith, S. L., Song, Y. J. C., Pye, J. E.,... & Guastella, A. J. (2018). Autism spectrum disorders: a meta-analysis of executive function. *Molecular psychiatry, 23*(5), 1198-1204.
- [8]. Goldstein, S., & Naglieri, J. A. (2014). *Handbook of executive functioning*. Springer.
- [9]. Hamilton, A. F. D. C. (2013). Reflecting on the mirror neuron system in autism: a systematic review of current theories. *Developmental cognitive neuroscience, 3*, 91-105.
- [10]. Hill, E. L. (2004). Evaluating the theory of executive dysfunction in autism. *Developmental review, 24*(2), 189-233.
- [11]. Hill, E. L., & Frith, U. (2003). Understanding autism: insights from mind and brain. *Philosophical Transaction*
- [12]. Kriete, T., & Noelle, D. C. (2015). Dopamine and the development of executive dysfunction in autism spectrum disorders. *PloS one, 10*(3), e0121605.
- [13]. Kubota, M., Fujino, J., Tei, S., Takahata, K., Matsuoka, K., Tagai, K.,... & Higuchi, M. (2020). Binding of dopamine D1 receptor and noradrenaline transporter in individuals with autism spectrum disorder: A PET Study. *Cerebral Cortex, 30*(12), 6458-6468.
- [14]. Loomes, R., Hull, L., & Mandy, W. P. L. (2017). What is the male-to-female ratio in autism spectrum disorder? A systematic review and meta-analysis. *Journal of the American Academy of Child & Adolescent Psychiatry, 56*(6), 466-474.
- [15]. Muller, C. L., Anacker, A. M., & Veenstra-VanderWeele, J. (2016). The serotonin system in autism spectrum disorder: from biomarker to animal models. *Neuroscience, 321*, 24-41.

- [16].Schmitz, N., Rubia, K., Daly, E., Smith, A., Williams, S., & Murphy, D. G. (2006). Neural correlates of executive function in autistic spectrum disorders. *Biological psychiatry*, 59(1), 7-16.
- [17].Van der Linden, P. M. A. (2001). Supervisory attentional system in patients with focal frontal lesions. *Journal of Clinical and Experimental Neuropsychology*, 23(2), 225-239.