Comparative Analysis Between The Perceptive-Cognitive Scale And The Evolutive- Behavioural Scales To Autism Diagnosis

PhD. Manuel Ojea Rúa

University of Vigo ORCID: <u>https://orcid.org/0000-0002-9787-2520</u> Mail: moxea@uvigo.es

ABSTRACT

The theoretical- propositional changes of autism spectrum disorder (ASD) from generalized structural deficit conception of development towards a multilateral conceptual basis of neurodevelopmental, it has allowed the evolution of the test and scales to score the items-criteria that make up this disorder from a perspective, not only evolutionary-behavioural, but also perceptual-cognitive and neural-nodal relational, therefore, in this sense, then new scales construction in order to complement evolutive and behavioural analysis with the exhaustive neurocognitive functional study, especially, focused on the synaptic neural processes of the information processing. Right, in this study has been proposed as a general aim to compare the diagnostic conclusions gotten justly the application of the evolutionary- behavioural scales and the integrated complementation of the perceptual-relational-cognitive from the scales based on the analysis of executive processing deepening about. A total of 24 students have participated in this study to, 6 participants with a previous diagnosis who haven't presented ASD' diagnosis from the conventional scales, 9 with diagnosis of ASD-1 level, 6 with diagnosis of ASD-2 level and 3 with diagnosis of ASD-3 according the application of the evolutionary scales. The study complemented with the Revised Perceptual-Cognitive Scale has been analysed throughout non-parametric statistical test by Friedman and Kruskal Wallis. Data found have indicated significant changes over initial diagnostic conclusions, which have been found significant critical differential comparative levels regarding to initial diagnostic group to .05 reliability level.

KEYWORDS

Autism Spectrum Disorder. Diagnosis. Perceptive- Cognitive. Relational Nodes.

Date of Submission: 24-04-2024

Date of acceptance: 02-05-2024

I. INTRODUCTION

The conceptual evolution of ASD from a propositional configuration as a pervasive developmental disorder (American Psychiatric Association [APA], 2013) and World Health Organization [WHO] (1992; 2020) towards a multilateral propositional consideration based on neurodevelopmental conceptualization, in which basically all the perceptual-cognitive factors that make up the neuropsychological executive processing of information are involved, that's generating substantial changes in the comprehensive paradigm of autism as a particular need that decisively influences parallelly way the evolution of the disorder diagnostic process shape.

In relation to the diagnostic process, from these theoretical-applied conceptions, the elaboration of diagnostic tests and scales has followed, according the items-criteria indicated by cited classifications, which, among others, stand out very specially, the Observation of the Diagnosis of Autism Scale, 2nd ed. [ADOS-2] (Lord, Rutter & Le Couteur, 1994; Lord, Rutter, DiLavore & Risi, 1999), the Autism Diagnostic Interview-Revised [ADI-R] (Rutter, Le Couteur & Lord, 2003) and the Gilliam Autism Rating Scale [GARS- 2] (Gilliam, 2001; 2005; 2010), the applied justification for which is based on the concept that have been gotten over the above international classifications criteria.

In this sense, people with ASD are highly deficient in the development of synaptic relational networks, which is influencing along its cognitive-perceptual particularities, also affecting in a particularly special way the emotional and behavioural system. This processing level configures a specific treatment model around which the specific psychosocial and educational development response should be adjusted, as it is precisely through these relational nodes that the feedback of information has developed, both in terms of incoming stimuli with information stored in permanent memory, and between the semantically integrated information itself, its practical application and higher cognitive processes.

The justification for this new paradigm has been developed throughout the current empirical research.

Accordingly, based on the studies of very different authors, both Casanova et al. (2002), like other different authors (Adorjan et al., 2017; Amina et al., 2021; Ariza, Rogers, Hashemi, Noctor & Martinez-Cerdeno, 2018) show that people with autism present specific features determined by the presence of pyramidal neurons, which show a dendritic increase with evident alterations in the system of neuronal connections or interneuronal GABAergic brain system, which form a regulatory network of the activity of the local glutamatergic pyramidal projection. Within these empirical insights, several subtypes based on differences in cell morphology and the particular connectivity between brain circuits have been identified, especially affecting the prefrontal cortex, through which information flows (De Felipe et al., 2013; Hof et al., 1999; Zaitsev, Gonzalez-Burgos, Povysheva, Kroner, Lewis & Krimer, 2005), further characterised by the presence of obvious axonal deficiencies in the nerual transporters of information within the like whole brain system like a whole (Dufour, McBride, Bartley, Juarez & Martínez-Cerdeño, 2023; Wang et al., 2016).

This condition gives rise to particular cognitive deficits in tasks that require interconnecting information processing for higher executive activities, related to problem-solving tasks or cognitive deductive processes based on the accumulated knowledge relationship within the semantic memory (Hashemi, Ariza, Rogers, Noctor & Martinez-Cerdeno, 2017; Hutsler y Zhang, 2010 & Lawrence Kemper, Bauman & Blatt, 2010). But, besides, its influence is already produced from the first receptive stimulus beginnings, since the initially perceived stimulus presents limited semantic content owing to needs for the immediate execution of neural- nodal relations with the previous information saved in permanent memory, so people with autism need to carry out a second cognitive execution from the initially perceived stimulus, which it will be more local and deeper to complement the initial semantic fault, which involves a mediating cognitive cost that can considerably delay the developmental evolutionary process, both at a social skills level and at a perceptive- cognitive one (Beer et al., 2006), stereotypical and restrictive behaviours (Tanji & Hoshi, 2008), language skills (Hertrich et al., 2021), as well, on educational area applied (Hashemi et al., 2017).

The etiology of this particular process have a genetic basis, in relation to which multiple possible genetic mutations have been identified, as well as environmental processes that, in turn, affect these genetic mutations also, including severe infections or diseases such as encephalitis or meningitis, which can cause neuronal remodelling compatible with this specific genetic mutation with severe consequences observed in the GABAergic pathways, resulting in a significant disequilibrium between cerebral excitation and inhibition processes (Chao et al., Courchesne et al, 2011; 2010; Pizzarelli & Cherubini, 2011; Ploeger, Rojas, Reite, Teale & Rogers, 2010). Likewise, a disruption in the interconnected processing of information is produced, which shapes human particularity, being fundamental for the comprehensive development of sensory input and its procedural linking to higher cognitive- executive tasks. That's to say, the sensory input of isolated stimulus must be duly categorised in relation to their similarities and differences with the incoming stimuli stored to facilitate that working memory can be able of facilitating the access of this information perceived to long-term or permanent memory (semantic memory); but, relational- nodal deficits could keep of or reduce the categorisation of stimulus information, therefore the perceived information simply would be lost immediately. To avoid being lost it is necessary that perceived information be accompanied by the complementary mediating processing, based on the elaboration of correspondent synaptic-neural networks throughout the developmental process (Hadjikhani et al., 2015; Wilson, Rojas, Reite, Teale & Rogers, 2007).

In synthesis, deficits in GABAergic connections in people with ASD have consequences for perceptual-cognitive and neuropsychological process functioning in information processing as a whole, so it is necessary to design specific scales to evaluate these components in order to avoid errors in the ASD' diagnosis jointly to the conventional evolutionary- behavioural analyses indicated by currently international classifications.

Indeed, based on these ongoing studies, Ojea (2023) has developed the global cyclical theory, which configures the particular perceptual-cognitive processing of people with ASD from the initial stimulus reception to the configuration of relational categories in permanent memory. These empirical advances should be accompanied by the development of new diagnostic methods that comprehensively gather the characteristic perceptual-cognitive processing, in order to complement this disorder evolutionary-behavioural process diagnosis.

Well, in this sense, Ojea (2023) has carried out the highly structured design of a specific scale that analyses ten basic dimensions of development, six of which are related to the perceptual-relational-nodal and cognitive components, four of which are related to social interaction and communication abilities and two are related to restrictive and hypersensitive and stereotyped behaviours, which make up the Perceptual-Behavioural Diagnostic Scale (2024a), which was recently revised (Ojea, 2024b).

The **main aim** of this study is precisely to refute, corroborate and higher specify the data of this revised scale, in order to compare the diagnostic conclusions found through perceptive- cognitive scale regarding to traditional scales applied to delimit the disorder diagnostic process.

II. METHOD

Research design

The research design is based on a quantitative comparative measure analysed by means of empirical non-parametric statistical tests for k related tests and for independent tests considering the variable "level" (diagnostic level) as a measure of comparison between the data found by means found of previously diagnostic carried out through the ADOS-2 scale, ADI-R scale or the GARS-2S test of evolutionary- behavioural diagnoses, regarding the possible diagnostic differential found from to complementary data of Perceptual-Cognitive-Revised Scale applied.

Participants

A total of 24 participants agreed to collaborate in this study, to whom the Perceptual-Cognitive- Revised Scale was applied as a complementary measure to the initial diagnostic process, six of whom did not have a specific diagnosis of ASD, six participants had a level 1 diagnosis, six with a level 2 diagnosis and three with ASD[′] level 3 (see Table 1).

		14810 11		<u>j 101 an ee</u>				= :)•	
				z_1	Z ₂	p_1	p_2	evolutive	cognitive
level	no deficit	1		00	+.01	<50	>51	1.50	3.00
		2		00	00	<50	<50	1.50	1.50
		3		+.01	+.01	>51	>51	1.50	3.00
		4		00	+.01	<50	>51	3.00	3.00
		5		+.01	+.10	>51	>75	4.50	3.00
		6		00	00	<50	<50	4.50	3.00
		Total	Ν	6	6	6	6	6	6
	level 1	1		+.01	00	>51	<50	4.50	1.50
		2		+.01	00	>51	<50	3.00	1.50
		3		+.01	+.10	>51	>75	3.00	1.50
		4		+.01	+.01	>51	>51	3.00	3.00
		5		+.01	+.10	>51	>75	3.00	1.50
		6		+.01	00	>51	<50	3.00	1.50
		7		+.01	00	>51	<50	3.00	3.00
		8		+.01	00	>51	<50	3.00	1.50
		9		+.01	+.01	>51	>51	4.50	1.50
		Total	Ν	9	9	9	9	9	9
	level 2	1		+.01	+.10	>51	>75	4.50	3.00
		2		+.01	+.01	>51	>51	4.50	3.00
		3		+.01	00	>51	<50	3.00	1.50
		4		+.01	+.10	>51	>75	3.00	3.00
		5		+.10	+.01	>75	>51	4.50	3.00
		6		+.01	+.10	>51	>75	4.50	4.50
		Total	Ν	6	6	6	6	6	6
	level 3	1		+.10	+.10	>75	>75	4.50	4.50
		2		+.01	+.01	>51	>51	4.50	4.50
		3		+.10	+.10	>75	>75	4.50	4.50
		Total	Ν	3	3	3	3	3	3
	Total	Ν		24	24	24	24	24	24

Table 1: summary for all cases according to ASD' level (N: 24).

Variables

In order to carry out this study, a total of seven variables have been operationalised, five study variables and two others that have been calculated from the isolated variables.

The five isolated variables are the following:

- 1) "level" (diagnostic level): 0 "no deficit", 1 "low level", 2 "half level", 3 "high level" (APA, 2013).
- 2) z_1 : typical score related to the developmental-behavioural study.
- 3) " z_2 ": typical score further extended with the perceptual-cognitive study.
- 4) " p_1 ": percentile relating to the developmental-behavioural study.
- 5) " p_2 ": percentile found with complemented perceptual-cognitive study.

The two statistical calculated variables are the following:

6) "evolutive" variable, which is formed by the mean (μ) of the sum of the evolutionary-behavioural variables: z_1 and p_1 , being "z" typical score regarding the developmental-behavioural study and "p" the associated percentile.

7) "cognitive" variable, calculated based on μ of z_2 and p_2 , being "z" typical score regarding the perceptive- cognitive study and "p" the associated percentile.

Procedure

The participants who participated in the study (N: 24) had a diagnosis based on the Behavioural-Evolutionary Scales, belonging to personal database from a total of 390 archived cases. The 24 participants who have accessed the study, were administered the Perceptual-Cognitive-Revised Scale and the data found have been compared with the previously existing data, which correspond to the indicated operational variables, analysed by means of non-parametric tests owing a relatively small sample.

III. RESULTS

Comparative analysis for relating samples

The comparative study of the measurement variables, regardless of the disorder level, both for individual variables and for statistically calculated grouped variables, has been developed through the Friedman statistical test.

Firstly, from the attribution of the specific ranks, which can be seen in Table 2, the comparative test is deducted.

$\begin{tabular}{|c|c|c|c|c|} \hline { Table 2: ranks to Friedman Test.} \\ \hline μ Rank \\ \hline z_1 & 2.85 \\ \hline z_2 & 2.88 \\ p_1 & 2.85 \\ p_2 & 2.88 \\ \hline \end{tabular}$

5.35

4.19

evolutive

cognitive

Indeed, the ranks distribution is significant differential, being slightly higher in the developmental-behavioural variables regarding to the perceptual-cognitive variables ($\neq 1.16$), which allows us to obtain a rightly differential critical level between both criteria in Friedman's test for five degrees of freedom (df.) (see Table 3).

Table 3: Friedman test.			
Ν	24		
Chi-Square	46.26.		
df.	5		
Asymp. Sig.	.000.		

Indeed, a significantly differential critical level has been observed (sig: .00), hence, a significantly differential explanatory score was found between the developmental and perceptive- cognitive data, as reflected in the chisquare (x^2 : 46.265). This subject allows us to conclude that there are significant differences according to the diagnosis whether it is carried out by means of developmental-behavioural tests or perceptive- cognitive side, which allows deduce higher significant differential results on final diagnostic group.

Comparative analysis by disorder level

In this section, it has been tried to analyse whether the differential components according to diagnostic level found for k independent samples compared by Kruskal-Wallis, being the variable "level" used like grouping variable of the evolutive and cognitive components of this study.

In effect, the ranks found from which the comparative test for independent samples measured as a function of "level" variable are significant differential between the developmental-behavioural variables and the perceptual-cognitive variables (see Table 4).

	Table 4: ranks to Kruskal Wallis test.					
	level	Ν	μ Rank			
Z1	no	6	6.00			
	deficit					
	level 1	9	13.00			
	level 2	6	14.67			
	level 3	3	19.67			
Z_2	no	6	11.17			
	deficit					
	level 1	9	9.83			
	level 2	6	15.17			
	level 3	3	17.83			
p_1	no	6	6.00			
-	deficit					
	level 1	9	13.00			
	level 2	6	14.67			

	level 3	3	19.67
p ₂	no	6	11.17
-	deficit		
	level 1	9	9.83
	level 2	6	15.17
	level 3	3	17.83
evolutive	no	6	8.75
	deficit		
	level 1	9	10.83
	level 2	6	15.50
	level 3	3	19.00
cognitive	no	6	13.33
•	deficit		
	level 1	9	7.22
	level 2	6	14.58
	level 3	3	22.50
	Total	24	

As can be seen, the differences found are generally within code 0 ("no deficit") to "level" variable (\neq -4.58), while for behavioural and cognitive variables the differences in the ranks are for level- 1 (\neq 3.61), to level- 2 (\neq .92) and to level- 3 (\neq -3.5).

The specific Kruskal- Wallis test indicates, therefore, significant differential critical levels in the disorder diagnostic conclusions as a function of the developmental-behavioural variables regarding to the perceptual-cognitive variables (see Table 5).

Table 5: Kruskall Wallis test.						
	z1	z2	p1	p2	evolutive	cognitive
Chi-Square	13.70	4.55	13.70	4.55	6.98	13.70
df.	3	3	3	3	3	3
Asymp. Sig.	.00	.20	.00	.20	.07	.00

In fact, the categorical differential statistical value between the mean of the behavioural and cognitive variables is (\neq -6.72) for three degrees of freedom (df.: 3).

Non- parametric analysis to differential diagnostic groups.

If it is justly considered the cases in which differences have been found in the diagnostic process as a consequence of the groups of variables used side, it can be observed that, of the 24 cases analysed, 6 (25%) that were initially diagnosed the autism disorder, has been lost this diagnostic category with the application of the Perceptual- Cognitive Scale (see Table 6).

				evolutive	cognitive
level	level 1	1		4.50	1.50
		2		3.00	1.50
		3		3.00	1.50
		4		3.00	3.00
		5		3.00	1.50
		Total	Ν	5	5
	level 2	1		3.00	1.50
		Total	Ν	1	1
	Total	Ν		6	6

Table 6: summary	of t	he disor	der diag	gnosis lo	st.
------------------	------	----------	----------	-----------	-----

As can be seen, five participants with ASD' level 1, who had a positive diagnosis of this disorder, it has lost this conception with the complementation of the perceptual-cognitive scale applied, with a differential: 7.5; while, one participant with ASD' level 2 diagnosis moved away from this diagnostic group of disorder with a differential score: 1.50 as a consequence of the new perceptual-cognitive scales applied along this study. Likewise, three participants (8.33%) who were not initially in the diagnostic group with the Behavioural-

Evolutionary Scales, with the addition of the Perceptual-Cognitive Scale, have been jointed to this disorder diagnostic (see Table 7).

Table 7: summary	of	disorder	diagnosis	gotten.

no	1		evolutive	cognitive
no	1		1 50	
	1		1.50	3.00
deficit	2		3.00	3.00
	Total	Ν	2	2
	deficit	deficit 2 Total	deficit 2 Total N	deficit 2 3.00 Total N 2

Regarding to participants who change the diagnostic criteria positively, two belonging to students who no longer had a differential diagnostic conclusion, whose statistical difference is (\neq -1.5), while one participant who had been included in the ASD' level 1 diagnostic group classified with the Behavioural-Evolutionary Scales, has given up this diagnostic status with the complementing of the perceptual-cognitive study.

IV. CONCLUSIONS

Although this study has widespread limitations due to small sample of participants, some considerations can be clearly deduced to be and can be refuted in other higher sample size analyses.

As can be observed, in general terms, 33.3% of the cases analysed for a total of N: 24 present changes in the initial diagnostic process carried out on from the traditional evolutionary scales, six of which, who had been left out of the diagnostic group are included in the disorder symptomatic condition with the new perceptive- cognitive scale applied, while three of them, who had been considered within the disorder symptomatic group, were left out of the initial diagnostic group with the new scale now.

Indeed, when the disorder diagnosis is limited exclusively to the developmental-behavioural components, the observable behaviours in their different levels of intensity could to lead to wrong definitive diagnostic conclusions, owing, in mild situations or situations of lesser need, related to ASD' level 1, the social-behavioural manifestations could evolve favourably if there aren't sensible deficits in the GABAergic synaptic connections that make up the perceptual- relational- cognitive processing. In these situations, therefore it could be concluded with evident improvements in the presence of the behavioural deficits observed throughout the evolutive development in the short-term and medium-term, which in this research study, corresponds to 25% of the cases analysed.

On the other hand, there are cases that show very mild behavioural manifestations, either presenting a series of behavioural acts in one dimension but not in others, which from direct observation should place them outside the diagnostic group of the disorder, but their level of procedural executive functioning presents limitations in the interrelational pathways of information connection that make these behavioural patterns recurrent over time or significantly worse in the short and medium term, which in this study corresponds to 8.33% of the cases studied.

Besides, significant changes in scores are highlighted, although they haven't led changes of disorder diagnosis. Thus, in case 5 of "no deficit", the score found is significantly modified since +.01 to +.10 in typical score. Likewise, at "level" 1, cases 3 and 5 move up the typical level from +.01 to +.10 of the analysis gaussian curve. The same is true for cases 1, 4 and 6 corresponding to the comparative analysis of "level" 2. Finally, it should be highlighted that, in "level" 3 of the case studies compared, the scores remain constant and invariable throughout the course of the further study with the Perceptual-Cognitive Scale regarding the initial evolutionary-behavioural data found.

Therefore, connective processes analysis that systemically influence the global functioning of the perceptual-cognitive process is a fundamental and highly determinant issue in the specific differential diagnosis of this disorder, hence the ASD['] diagnosis should include the clearly specified perceptual-cognitive variables to complete the evolutionary-behavioural analysis indicated by the currently classifications of ASD, otherwise, the basic error in the diagnostic processes of this disorder will be recurring.

For this reason, it is a priority subject matter to revise the currently criteria for this specific disorder in order to incorporate the relational perceptive- cognitive dimensions as intrinsic variables within the current existing dimensions and as own new dimensions, which comprehensively analyse the human perceptual-cognitive executive functioning that is the final diagnostic goal.

Hence, the addition of these dimensions is absolutely necessary, not just to avoid initial diagnostic errors, but, above all, because the executive processual analysis of information brain connectivity provides a more specific criterial basis from the differential perspective of this disorder than observable social-behavioural dimensions currently isolated.

REFERENCES

- [1]. Adorjan, I., Ahmed, B., Feher, V., Torso, M., Krug, K., Esiri, M., ... & Szele, F. G. (2017). Calretinin interneuron density in the caudate nucleus is lower in autism spectrum disorder. Brain, 140(7), 2028-2040. https://doi.org/10.1093/brain/awx131
- [2]. American Psychiatric Association (APA) (2013). Diagnostic and Statistical Manual of Mental Disorders (DSM-5). Arlington, VA. https://psychiatry.org/psychiatrists/practice/dsm
- [3]. Amina, S., Falcone, C., Hong, T., Wolf-Ochoa, M. W., Vakilzadeh, G., Allen, E., ... & Martinez- Cerdeno, V. (2021). Chandelier cartridge density is reduced in the prefrontal cortex in autism. Cerebral Cortex, 31(6), 2944-2951. <u>https://doi.org/10.1093/cercor/bhaa402</u>
- [4]. Ariza, J., Rogers, H., Hashemi, E., Noctor, S. C., & Martinez- Cerdeno, V. (2018). The number of chandelier and basket cells are differentially decreased in prefrontal cortex in autism. Cerebral Cortex, 28(2), 411- 420. <u>https://doi.org/10.1093/cercor/bhw349</u>
- [5]. Dufour, B. D., McBride, E., Bartley, T., Juarez, P., & Martínez- Cerdeño, V. (2023). Distinct patterns of GABAergic interneuron pathology in autism are associated with intellectual impairment and stereotypic behaviours. Autism, 27(6) 1730- 1745. DOI: 10.1177/13623613231154053.

- [6]. Casanova, M. F., Buxhoeveden, D. P., Switala, A. E., & Roy, E. (2002). Minicolumnar pathology in autism. Neurology, 58(3), 428-432. <u>https://doi.org/10.1212/wnl.58.3.428</u>
- [7]. Chao, H. T., Chen, H., Samaco, R. C., Xue, M, Chahrour, M., Yoo, J, ... & Zoghbi, H. (2010). Dysfunction in GABA signalling mediates autism-like stereotypies and Rett syndrome phenotypes. Nature, 468, 263-269. <u>https://pubmed.ncbi.nlm.nih.gov/21068835/</u>
- [8]. Courchesne, E., Mouton, P. R., Calhoun, M. E., Semendeferi, K., Ahrens- Barbeau, C., Hallet, M. J., ... & Pierce, K. (2011). Neuron number and size in prefrontal cortex of children with autism. JAMA: The Journal of the American Medical Association, 306, 2001-2010. <u>https://pubmed.ncbi.nlm.nih.gov/22068992/</u>
- [9]. De Felipe, J., Lopez- Cruz, P. L., Benavides- Piccione, R., Bielza, C., Larranaga, P., Anderson, S., . . . & Ascoli, G. A. (2013). New insights into the classification and nomenclature of cortical GABAergic interneurons. Nature Reviews Neuroscience, 14(3), 202-216. <u>https://doi.org/10.1038/nrn3444</u>
- [10]. Gilliam, J. E. (2001). Gilliam Asperger's Disorder Scale. Austin, TX: Pro-Ed. <u>https://www.txautism.net/evaluations/gilliam-aspergers-disorder-scale</u>
- [11]. Gilliam, J. E. (2005). Gilliam Autism Rating Scale GARS-2 (2ª ed.). México: PROED. www.proedlatinoamerica.com
- [12]. Gilliam, J. E. (2010). Escala de Evaluación del Autismo de Gilliam. México: Rústica. ISBN: 13: 9789707293656
- [13]. Hadjikhani, N., Zürcher, N. R., Rogier, O., Ruest, T., Hippolyte, L., Yehezkel Ben- Ari, Y., ... & Lemonnier, E. (2015). Improving emotional face perception in autism with diuretic bumetanide: A proof-of-concept behavioural and functional brain imaging pilot study. Autism, 19(2) 149- 157. DOI: 10.1177/1362361313514141
- [14]. Hashemi, E., Ariza, J., Rogers, H., Noctor, S. C., & Martinez- Cerdeno, V. (2017). The number of parvalbumin-expressing interneurons is decreased in the prefrontal cortex in autism. Cerebral Cortex, 27(3), 1931-1943. <u>https://doi.org/10.1093/cercor/bhw021</u>
- [15]. Hof, P. R., Glezer, I. I., Conde, F., Flagg, R. A., Rubin, M. B., Nimchinsky, E. A., ... & Vogt- Weisenhorn, D. M. (1999). Cellular distribution of the calcium-binding proteins parvalbumin, calbindin, and calretinin in the neocortex of mammals: Phylogenetic and developmental patterns. Journal of Chemical Neuroanatomy, 16(2), 77- 116. <u>https://doi.org/10.1016/s0891-0618(98)00065-9</u>
- [16]. Hutsler, J. J., & Zhang, H. (2010). Increased dendritic spine densities on cortical projection neurons in autism spectrum disorders. Brain Research, 1309, 83-94. <u>https://doi.org/10.1016/j.brainres.2009.09.120</u>
- [17]. Lawrence, Y. A., Kemper, T. L., Bauman, M. L., & Blatt, G. J. (2010). Parvalbumin-, calbindin-, and calretinin-immunoreactive hippocampal interneuron density in autism. Acta Neurologica Scandinavica, 121(2), 99- 108. <u>https://doi.org/10.1111/j.1600-0404.2009.01234.x</u>
- [18]. Lord, C., Rutter, M., & Le Couteur, A. (1994). Autism Diagnosis Interview–Revised: a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. Journal of Autism and Developmental Disorders 24, 659-685. DOI: <u>10.1007/BF02172145</u>
- [19]. Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (1999). Autism Diagnostic Observation Schedule: Manual. Los Angeles, CA: Western Psychological Services. https://link.springer.com/article/10.1023/A:1005592401947
- [20]. Ojea, M. (2023a). Autism: new conceptual propositional hypothesis. European Journal of Theoretical and Applied Sciences, 1(6), 115-124. <u>https://ejtas.com/index.php/journal/article/view/437</u>
- [21]. Ojea, M. (2024a). Autism Perceptual- Behavioural Precision Scale. European Journal of Theoretical and Applied Sciences, 2(1), 18-45. DOI: 10.59324/wjtas.2024.2(1).02. <u>https://ejtas.com/index.php/journal/article/view/555</u>
- [22]. Ojea, M. (2024b). Escala perceptivo- cognitiva de diagnóstico del trastorno del espectro autista revisada (EPPC-TEA-R). RIP: REGAGE23e00084280417. ISBN: 978-84-09-59160-2. Editions Kindle Direct Publishing. <u>https://www.amazon.es/dp/B0CW1L8BTH</u>
- [23]. Pizzarelli, R., & Cherubini, E. (2011) Alterations of GABAergic signalling in autism spectrum disorders. Neural Plasticity, 297153 (12 pp.). <u>https://pubmed.ncbi.nlm.nih.gov/21766041/</u>
- [24]. Ploeger, A., Raijmakers, M. E., van der Maas, H. L., & Galis, F. (2010) The association between autism and errors in early embryogenesis: what is the causal mechanism? Biological Psychiatry, 67, 602- 607. <u>https://pubmed.ncbi.nlm.nih.gov/19932467/</u>
- [25]. Rutter, M., Le Couteur, A., & Lord, C. (2003). Autism diagnostic interview revised (ADI-R). Los Angeles, CA: Western Psychological Services. DOI: https://doi.org/10.1007/978-1-4419-1698-3_894
- [26]. Wang, Y., Zhang, P., & Wyskiel, D. R. (2016). Chandelier cells in functional and dysfunctional neural circuits. Frontiers in Neural Circuits, 10, 33. <u>https://doi.org/10.3389/fncir.2016.00033</u>
- [27]. Wilson, T. W, Rojas, D. C., Reite, M. L., Teale, P. D., & Rogers, S. J. (2007). Children and adolescents with autism exhibit reduced MEG steady-state gamma responses. Biological Psychiatry, 62, 192-197. <u>https://pubmed.ncbi.nlm.nih.gov/16950225/</u>
- [28]. World Health Organization. (1992). ICD 10: Mental and behavioural disorders: Clinical descriptions and guidelines for follow-up. World Health Organization. United Nations Organization (UN). https://www.who.int/publications/i/item/9241544228
- [29]. World Health Organization. (2020). ICD 11-R: Mental and behavioural disorders: clinical descriptions and guidelines for follow-up. World Health Organization. United Nations Organization (UN). https://icd.who.int/en
- [30]. Zaitsev, A. V., Gonzalez-Burgos, G., Povysheva, N. V., Kroner, S., Lewis, D. A., & Krimer, L. S. (2005). Localization of calciumbinding proteins in physiologically and morphologically characterized interneurons of monkey dorsolateral prefrontal cortex. Cerebral Cortex, 15(8), 1178-1186. <u>https://doi.org/10.1093/cercor/bhh218</u>