Predictive Analysis of the Principal Components that Configure Autistic Spectrum Disorder

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Abstract:

At currently, developments regarding to autism spectrum disorder have enabled its diagnostic group to be defined as a multilateral neurodevelopmental disorder characterised by peculiarities in the procedural functioning of perceptual-cognitive parameters, derived from a characteristic connexional form in the pathway of interconnection between intrinsic information and contextual stimuli. In this characteristic process, neuronal networks are involved a fundamental processing task for working memory to be able to perform the set of executive functions with a certain degree of stability, which is severely limited in people diagnosed with this disorder. In this study, the reports of 403 participants diagnosed with the disorder were analysed with the following basic goals: 1) to analyse the relationship between relational deficits and the elaboration of semantic content, 2) to analyse the etiological attribution of the GABAergic pathway responsible for the limitations in these connections and, consequently, 3) to conclude the main predictive-explanatory level of this disorder. The data have been found by means of different statistical tests, both initial correlational tests, statistical calculation processes, univariate one-factor ANOVA tests and final consequential ordinal multinomial logit regression tests. Data found allow us to delimit that the regression equation of the model fitting information explaining the disorder shows a final logit model chi-square: 217.23, with a significant associated critical level (sig: .00), which are complemented by the significantly positive Pearson and Deviance data significantly related to the logit level (sig: .00), which confirms the importance of the predictive-explanative level of the neuronal and semantic variables derived from the GABAergic limitations in order to be able to be converted into the main propositional components of autism as a highly related neurocognitive systemic process.

Keywords: autism spectrum disorder, etiology, karyotype, neuronal relationships, semantic memory.

Introduction

Conceptually, people with autism spectrum disorders (ASD) shape a diagnostic group characterised by multilateral neurocognitive deficits, which are observable through two behavioural developmental categories, rightly defined by the International Classification of American Psychiatric Association (APA) (2013), which are presented in differences of intensity: 1) structural deficits at the social level, both in social skills and in social or emotional communication complementary to language development, and 2) the presence of restrictive and stereotyped behaviors. In both categories, the affective-emotional symptom groups are intrinsically included, which have thus been focused on by the successive scales for
measuring the disorder diagnosis. However, the differences in intensity of developmental-behavioural are so highly that one person may present these behaviours excessively, while in another they may go almost overlooked, so that the exclusively assessment of these dimensions has led to many errors in the currently diagnostic processes. Indeed, the most updated researches in the field of autism are focusing their efforts on the perceptual-cognitive particularities that occur along the specific component of information processing, which make up main components that have to be a specific objective of the diagnosis to complement the current evolutionary-behavioural assessment.

From the psychoneurological perspective, there are recurrent studies on the etiological components of genetic basis with high hereditary karyotypical components, which Arenella et al. (2022) place in 70-90% of explanation of the expression of autism, which determines a series of specific implications in the conceptual conformation of the disorder that should be exhaustively evaluated. And although the genetic condition is not linear, it is particularly complex, involving a multifactorial genetic mutation process, in which multiple genetic variants may be implicated (Grove et al., 2019) and organic factors produced by diseases, such as encephalitis, meningitis or other severe blood infections, which can cause a neuronal remodelling similar to the consequences derived from the mutation genetic condition, affecting specifically the connexional pathways, which are the facilitators of the necessary interrelation in the information processing, giving specific cognitive particularities that affect especially the cerebral connections located in the cerebral GABAergic pathway, in which multiple neuronal alterations have been found which have been related to the autism specific phenotypical development (Amina et al., 2021), which has likewise been corroborated by the studies of Dufour, McBride, Bartley, Juarez & Martinez-Cerdeño (2023).

Effectively, in the human brain of people with ASD, severe interneuronal alterations have been identified that affect the connective neuropathways, which make up between 20% and 30% of all neocortical neurons, forming a network that regulates all neuronal activity of local glutamatergic-pyramidal projection, with multiple variants and different subtypes that particularly affect the interconnectivity of the circuits through which the information needs to be processed to can be understood and, hence, facilitate psycho-social development about (Ariza et al., 2018; Adorjan et al., 2017; Hashemi et al., 2017; Lawrence, et al., 2010).

According to the analysis of Lisman & Buzsaki (2008) and also Pizzarelli & Cherubini (2011) specificities in the connexional scope are owing to the presence of disequilibrium between excitation and inhibition, which present a dysregulation with gamma oscillations, which are generated by GABergic neurons and are responsible for the stimuli sensory reception, as well-being and, above all, for the capacity to perform high-level executive functions, when the relations between semantic categories are absolutely needed. But, as currently studies by Hadjikhani et al. (2015) have shown, these particularities are already observable in the early phases of sensory perception, observable in deficits over contextual and emotional recognition of incoming stimuli, owing precisely to automatic limitations in the development of the relationships needed between incoming stimulus and the meaning comprehension laid semantically.

Therefore, from the reception of the stimulus through the sensory-perceptual memory, cognitive processing is initiated through the working memory, which must categorise this new stimulus with understanding in order to be able to facilitate its access to the permanent memory suitably categorised, where it'll be stored unlimitedly until it's required for mnesic according to contextual or interoceptive requests.

Consequently, the basic focus of psycho-cognitive processing is the long-term storage of information in semantic terms in semantic-permanent memory. However, for this cognitive processing to be effective and functionally executed, it is required that working memory performs two main executive functions, firstly
the development of relationships of neuronal nodes relating the new stimulus to previously knowledgeable information, in order to facilitate its conceptual understanding and then, encoding the information in a categorical and sub-categorical level, which again involves the development of relationships and neuronal nodes between all the relational semantic components. This procedure is required owing to spatial limitation of permanent memory, which is unable to register all the individual stimuli that happen every day in the human life being. It follows that semantic memory and the elaboration of neuronal networks and relational nodes are absolutely essential for working memory to perform the principal function of mediating between initial perception and content that have been store-keeping in long-term memory. In this sense, the basic starting hypothesis is that both main components of neuropsychological information processing, based on the neuronal networks and semantic memory processing must be significantly related to encourage human cognitive and cognitive development.

An empirical relational study, analysed using Pearson's correlation (r), between both principal components was carried out to N: 403 participants with ASD of the disorder different levels according to the current norms of the international classification (APA, 2013), and significant relationships were found (sig: .00), with an r: .40 to .01 significant level.

Thus, given that both of these componential variables of cognitive functioning are correlated, it is possible to codify both variables statistically calculated as a single referential executive element to give an explanatory insight into perceptual-cognitive functioning within the brain matter and to encourage procedural development. In this same study, neuronal relations ("neuronal") and semantic memory ("semantic") have been configured as a single dimension of analysis, which has been found by means of statistical calculation, and has been designated "MEMORY", it has been hypothesised that executive process will function more or less effectively in relation to the brain parameters that may influence this interactive connexional. The most up-to-date studies that have been analysed the influence of psycho-organic parameters that decisively influence the interaction of the "MEMORY" dimension is the connexional pathway of the cerebral GABAergic system fluidity, which is determined by the cellular action involved in the synaptic-brain processes responsible for the substance that facilitates the interconnected fluence of the information, which is determined by multiple variables, among which, the neuro-cerebral characteristics are highlighted.

When these activities or connective actions present some alteration way, derived from an organic impairment or the detection of a genetic karyotype can influence directly or by a neuronal remodelling on the connexional synaptic pathway, so that, as a result, the cognitive circuit can be altered and shape temporary or permanent perturbations, which disrupt, limit or impair of the information semantic configuration development of to some degree, as a function of the multiple variables that are affecting it.

Hence, if the potential causes involved in the particular deficits in the elaboration of interrelated synapses at brain level in the group of participants with ASD are analysed for the sample of this study, then the initial explanatory hypotheses regarding the particular cognitive-executive interactions of the group belonging to this disorder could be corroborated. In effect, when we proceeded to undertake an explanatory univariate statistical analysis of different explanatory genetic-organic factors of the statistically calculated dimension "MEMORY" for the total of N: 403, it has been observed that the presence of the main influence of the 15q11-q13 gene has been found in 109 cases (27.04%), the SHANK2-3 gene in 14 cases (3.47%), the 7q11-q33 gene in 13 cases (3.22%), likewise, the influence of several genes and other organic alterations has been found in 51 cases (12.65%). In all the situations found, a direct or indirect relationship is established in the decisive influence on the fluidity of the synaptic substance that facilitates inter-informative relations, both from the outside towards the
permanent memory, and in the ambit of the retrieval of information fluidity from the inside towards the attention-perception that requests such semantic information.

However, it is necessary affirm that in 215 cases there were no specific genetic alterations (53.35%), therefore also other explanatory hypotheses owing to organic processes, such as infectious blood processes or diseases, such as encephalitis or meningitis, which can produce a neuronal remodelling observable through the frequent processes of involution in specific developmental-behavioural and cognitive areas need to be analysed. In this sense, it is necessary to assert that highly hostile contextual environmental processes may also be responsible for these multivariate causes, especially when they happen in critical evolutionary phases, especially from first 3 years of childhood, which may lead to a specific structural effect on the inhibitory interactive pathways of the inter-connective process, which may remodel the neuronal process that affects to connective areas fluidity, which may be the main aim of future research work.

However, in this specific research, the general goal was designed to determine whether the data indicated as explanatory of the connexional fluency, that affects the cognitive components of the disorder, are empirically significant and, as a consequence, to form the disorder diagnostic group, for which it has proceeded to perform this research process.

**Methods**

**Research Design**

This study was an experimental analysis based on data analysis of the registers related to the processes of diagnosis of the autism speciality developed at provincial scale, through the data analysis archived in a fully anonymous and confidential way.

**Participants**

A total of 403 diagnoses of participants with specific diagnosis of ASD have been analysed throughout the study, of which 256 indicate diagnosis of ASD level-1, 119 of level-2 and 28 of level-3, which have been extracted completely anonymously from the dossiers related the specific diagnosis reports of the autism speciality provincial service.

**Variables**

Variables related to the main explicative components of this disorder:

- "neuronal": the ability to establish frequent and autonomous nodal relationships between information from the outside and existing contents in permanent memory, found from the Wisconsin Card Sorting Test (Heaton, 1981). Likewise, relational activities have been developed based on previous knowledge, e.g., if in the previous learning content, the student knows how to sum and knows the use of "x" value, then, if "x": 2+3 (5), if it’s added 7 to the previously sum, what is now the value of "x"?

- “semantic”: the capacity to assign meaningful content to external stimuli and information, as well as the capacity for conceptual categorisation, which is the basic condition for working memory to be able to facilitate access to information to permanent memory and its subsequent retrieval, as measured by the My Test (Memory Test) (Yuste, 2005). It also proceeded to synthesising summaries and abstracts of different stories to students.

- "disorder", that related to the currently three existing levels of the disorder, that have been found through, among other tests, the Autism diagnostic observation schedule, second edition (ADOS-2) (Lord, Luyster, Gotham & Guthrie, 2012) and the Autism diagnostic interview revised (ADI-R) (Rutter, Le Couteur & Lord, 2003).

- "karyotype", it corresponds to the genetic starting hypothesis as it is the fundamental main component to determine the process of "MEMORY" calculated, that found from the corresponding clinical health analysis.

Variables "neuronal" and "semantic" have been unified in a statistical process of calculating their averages to provide a new coded variable, which will become the fundamental explanatory factor
to disorder way: "MEMORY" (neuronal + semantic).

**Procedure**

Diagnostic analyses have been developed throughout 12 years at the provincial level of a specific service for students with ASD. The diagnoses analysis has been carried out with absolute confidentiality of the data, limiting its collection to the study specific aims.

**Data Analysis**

The influence of the variable "karyotype" in relation to the variable "disorder" was found by means of an ANOVA analysis for a univariate factor.

In the same way, the possible significance of the incidence of the variable "karyotype" on the calculated variable "MEMORY" was analysed through the same univariate parametric test.

If significant analyses of these variances are confirmed, it is possible to advance in the field of the interrelation between the variables that shape the calculated variable "MEMORY" with the variable "karyotype" to determine the type of disorder "disorder" and, consequently, to advance in the explanatory hypotheses of ASD.

Finally, a predictive-explanatory analysis of the analysis variables "MEMORY" and "karyotype" considered as factors to delimit the variance explanatory quantity in the conceptual variable of ASD is performed by means of an ordinal multinomial logit regression analysis.

**Community Involvement**

The provincial service of diagnostic to people with autism has participated in the study, through the diagnosis dossiers with the guarantee of anonymity and confidentiality. Likewise, the Social Institute of Scientific Research in the Area of People with Autism Spectrum Disorder (ID: G44568509) has also participated in the study.

**Results**

Results are classified in two sections, firstly, the explanatory analysis of variable variance "karyotype" regarding to disorder: "disorder" variable, which is considered as a dependent variable (DV), with the aim of analysing if the expected explicative hypothesis is achieved, which has been analysed by one-factor ANOVA univariate analysis test. The overall data can be seen in Table 1.

**Table 1. Univariate Test**

<table>
<thead>
<tr>
<th></th>
<th>Sum of Squares</th>
<th>df</th>
<th>µ Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between Groups</td>
<td>40.90</td>
<td>5</td>
<td>8.18</td>
<td>28.46</td>
<td>.00</td>
</tr>
<tr>
<td>Within Groups</td>
<td>114.10</td>
<td>397</td>
<td>.28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>155.00</td>
<td>402</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** DV: disorder; Levene (sig: .00); Factor: "karyotype"

Indeed, as indicated, with the Levene homogeneity statistic being significant (sig: .00): 17.38, it can be observed that univariate explanatory analysis of "disorder" variable by the variable "karyotype", shows a significant critical level (sig: .00), being F value: 28.46, whose sum of squares between groups is: 40.90 and within group: 114.10, which allows to conclude that explanatory variance of the variable "karyotype" is significantly influential on the different levels of the ASD diagnostic group.

Graph 1 indicating the residuals of the explanatory variance, shows that expected hypothetical incidence of "karyotype" variable (lower horizontal axe) is equivalently adjusted to the frequencies observed in the data corresponding to "disorder" variable, represented on the left vertical axe.
In the second section of the results, the calculated variable "MEMORY" has been operationalised as DV, in an attempt to observe to what degree, the "karyotype" factor is significantly explanatory on the main components included in "MEMORY" dimensional variable.

Data found using the between-subjects effects test of the univariate ANOVA test can be seen in Table 2.

### Table 2. Tests of Between-Subjects Effects to “MEMORY”

<table>
<thead>
<tr>
<th>Source</th>
<th>Type III Sum of Squares</th>
<th>df</th>
<th>η Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corrected Model</td>
<td>145.73(a)</td>
<td>2</td>
<td>72.86</td>
<td>70.25</td>
<td>.00</td>
</tr>
<tr>
<td>Intercept</td>
<td>8444.42</td>
<td>1</td>
<td>8444.42</td>
<td>8141.65</td>
<td>.00</td>
</tr>
<tr>
<td>karyotype</td>
<td>145.73</td>
<td>2</td>
<td>72.86</td>
<td>70.25</td>
<td>.00</td>
</tr>
<tr>
<td>Error</td>
<td>414.87</td>
<td>400</td>
<td>1.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>16620.00</td>
<td>403</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corrected Total</td>
<td>560.60</td>
<td>402</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Note:** R Squared = .260 (Adjusted R Squared = .256); Factor: “karyotype”.
As can be seen, the specific critical level of the factor-variable "karyotype" is significant in explaining its incidence over interaction of the variables "neuronal" and "semantic", which have been coded as "MEMORY" (sig: .00), whose $F$: 70.25, being the sum of squares: 145.73 and mean square: 70.25. Likewise, both the corrected model and the intersection analysis indicate significant critical levels of explanatory factor variance on the operationalised DV: "MEMORY".

This conclusion is very important, as it allows the hypothetical study to be focused that "karyotype" variable influences the dimensional variable that gives the cognitive-perceptive ("neuronal" and "semantic" structure analysis of the "disorder" variable as a whole systemic interactive propositional to.

In Graph 2 of the residuals of the explanatory variance, it can be seen graphically that the observed data are clearly related to the expected data in this hypothetical statistical relationship between the variable "karyotype" (lower horizontal axe), with the interactive explanatory variable "MEMORY" expressed on the left vertical axe.

Hypothetically, it is expected to corroborate that the variable "disorder" is empirically explained by the dimensional variable "MEMORY" and "karyotype" variable, which has been analysed through the specific ordinal multinomial logit regression test to give the adjusted information of the influence of the factors on "disorder" variable, it has considered as DV. These analyses have been observed with -2 log likelihood of model fitting information (see Table 3).
Table 3. Model fitting Information (DV: “disorder”)

<table>
<thead>
<tr>
<th>Model</th>
<th>-2 Log Likelihood</th>
<th>Chi-Square</th>
<th>df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept only</td>
<td>361.90</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Final</td>
<td>144.67</td>
<td>217.23</td>
<td>14</td>
<td>.00</td>
</tr>
</tbody>
</table>

**Note:** Link function: Logit.

In the ordinal multinomial regression model, it can be observed that the final critical level is significant (sig: .00), with -2 logit: 144.67 of predictive explanatory level of the variables-factors: "karyotype" and "MEMORY" on the DV: "disorder", chi-square: 217.236, being the p-values of the chi-square of the goodness of fit and deviation also significant and with correct values to verify the previous data (see Table 4).

Table 4. Goodness-of-Fit

<table>
<thead>
<tr>
<th></th>
<th>Chi-Square</th>
<th>df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pearson</td>
<td>92.95</td>
<td>42</td>
<td>.00</td>
</tr>
<tr>
<td>Deviance</td>
<td>101.89</td>
<td>42</td>
<td>.00</td>
</tr>
</tbody>
</table>

**Note:** Link function: Logit.

Indeed, the Pearson and Deviance values also showed significant critical levels, involving an interactive process of the factor variance, which decisively influence the subject of "disorder" variable. Likewise, the Cox and Snell logit function values (Pseudo-R$^2$: .417) are rather agreeable for the explanatory-predictive function of the factor variables, which are slightly higher for the Nagelkerke statistic (R$^2$: .514) and lower for the MacFaden test (R$^2$: .32), nevertheless, it is possible to confirm with relative precision the goodness of fitting of the significant Pearson and Deviance data found in table 4.

**Discussion**

Despite the evidence of the relationship of karyotype-genetic specificities in the presence of the ASD diagnostic group, both in the diagnostic processes in the real sense, there is very limited content related to the presence of a karyotypic study at the individual genetic level and in the hereditary processes in the successive generations on diagnostic processes. Thus, from reports of this study N: 403, they justly represent 44.6% of the total number of all the available cases (N: 902) in the dossiers, however than autism diagnosis is indicated, they did not include a specific karyotypical study.

Nevertheless, from a highly predictive perspective, the performance of previous karyotypic analyses should be possible predictors of the presence of a possible diagnosis, which would lead to a positive prediction of possible clinical and educational actions as early as possible, as has been shown in the studies performed by The deepening in the knowledge of the main aetiological components that can influence in the presence of autism constitutes, therefore, a fundamental matter that requires a further study in the currently research.

This research should include the widest and earliest possible social setting, in ordered to educate families in all significant parameters according to the current studies, thus enabling them to be able to adopt preventive measures, since the performance of karyotypic studies can anticipate clinical prophylaxis measures already during the pre- and neonatal process that will avoid or, at least, limit the intensity of the dysregulation processes analysed. But, above all,
it would allow the most effective attention to be given as efficiently as possible to the consequences of the disorder, both in the developmental-behavioural area, and, above all, in the strengthening of relational connections and connective regulation that affect cognitive growth as a whole in people with ASD (Kreiman & Boles, 2020; Schaefer, Mendelsohn & Professional Practice Guidelines Committee, 2013).

In this respect, explanatory training measures can already be found through different training programmes with initiatives from the governmental scope, such as explanatory leaflets, official guides, internet procedures, advance courses, permanent seminars, congresses and conferences, adjusted to the proximity of the general-social people. In this direction, satisfactory results are already being produced by the approximation of these conceptual training components to society in general, as indicated by authors like Hellquist & Tammimies (2022) and Srivastava et al. (2019), which are getting a very positive response in Swedish society by the families to whom the training is addressed, however, a specific deepening is required in the specific training of these basic assumptions in the family and social ambit (The Swedish Neuropediatric Section of The Society for Swedish Pediatricians, 2019).

But, above all, this training should be prioritised in the factors that are directly related to the psycho-social care of people with ASD from the earliest age, so it should be initiated in the socio-health services responsible for diagnosis, educational services and public and private associations that provide psycho-social and educational services for people with ASD.

Of course, this training-information must be supported by the provision of highly specialised health and educational services and resources that are able to respond to the demands produced by this information in undertaking the necessary karyotypic clinic analysis, as well as the early medical and educational measures that would be necessary for each particular need.

Ojea (2019) has precisely proposed a specific training plan in the socio-familial field with the aim of empowering families in the specific knowledge of all the variables that intervene regarding autism. In this comprehensive approach it involves the individual, school and family level, through a specific comprehensive plan to facilitate a strategy for joint action in regard to the particular needs of ASD. Indeed, the benefits of this combined action are widely observed when they include the global social area, as has been empirically corroborated by authors of recognised prestige, such as Haven, Manangan, Sparrow & Wilson (2013), Ingersoll & Dvortcsak (2009), Interagency Autism Coordinating Committee (2009), Suppo & Mayton (2012) and Schultz, Schmidt & Stichter (2011). These studies highlight, indeed, the training benefits in the scope of specific improvement, however, this training should also involve the global social level in order to inform society as a whole about the main components that configure the etiology and executive processing of the disorder regarding with the scientific advances, than have been currently corroborated.

**Conclusion**

The logit predictive function analysed has indeed shown that systemic functioning of the study variables, which have been surveyed for this analysis, significantly explained a great part of the explanatory empirically variance found over ASD’ diagnosis ("disorder"). Obviously, the components set that interact on cognitive-perceptive level had a systemic function around these operative variables, in the same way, other organic factors may be involved around the "karyotype" variable value, which may remodulate the neuronal process leading to the same consequences as the "karyotypical" process variable in itself, Thus, this whole explanatory framework just confirm the importance of perceptual-cognitive factors in the ASD’ diagnostic identification.

Once the diagnosis in the strict sense has been configured, the developmental-behavioural components are evidence of greater or lesser intensity in the presence of the diagnostic group, but the developmental-behavioural components
will be the determinants of the diagnostic group. In this sense, highly evident criterion behaviours can be observed, but also totally imperceptible behaviours can occur, however, in the two situations, an exhaustive evaluation of the functional neurocognitive components is necessary for the specific determination of the setting of this diagnostic group.

This process is fundamental for diagnostic means, but also, consequently, to outline the conceptual didactic approach to indicators to design the methodological and educational action in accordance with the specific needs of the disorder particular features.

From this perspective, the educational intervention should be designed on the basis of the elaboration of basic instrumental mediators facilitating the cognitive synaptic connectionist pathways between the informational processes, which will first be more guided, but, progressively, facilitating a greater autonomy in their development, which will be an own specific function to the degree of intensity of the diagnosed structural deficits and also to the complexity of the learning contexts. This mediation process means to facilitate from the outside the networking of information enabling connections between contents coming from the outside and concepts previously stored in the semantic memory, with as many relational mediators, with as much functionality and as deep as may be necessary, in aim to enable the relational meaningfulness that is difficulted to perform autonomously of the diagnosed person.

In so doing, the approach to new learning should be extremely proximal to the already learned concept by configuring a conceptual category or subcategory in permanent memory (see Figure 1).

Figure 1. Approximal Mediation Process

Therefore, from the presence of the new stimulus in the sensory memory, the working memory should be encouraged to create so relational relationships as possible, which should be functional and highly significant in accordance with the information previously categorised in the permanent (long-term) memory and, when, for whatever reason, it is difficult to make functionally the relational knowledge available, then it is necessary to promote the repetition of the process on as many occasions as necessary for achieving an automated cerebral process and, in this hypothesis, it will be very useful to change the learning contexts progressively, but slightly, to encourage the repetition of the process. The resulting facilitating processes of self-categorical elaboration, higher executive actions and higher semantic mnesic capacity enhancement will depend on a multivariate whole of factors, which are related to intrinsic and extrinsic features and comorbidity processes that may evolve surrounding the disorder.

But what do it mean by creating mediated relationships?

The answer can be seen clearly with an example, if I have an actor in my mind, but I don't remember his name, I try to access some film in which he has participated, if I don't remember it, hence, I try to access some social action in which he has participated, but, even so I can't
remember him either, then, a external mediator should help me to progressively facilitate those remembering, so that I can gradually can find myself the right way to reach the relationship that allows me to access the actor name; for this to be possible, I must have previously assimilated the semantic content related to the fact of having seen a film or social action previously related to that actor, otherwise, it is not possible to execute a content meaningful level, even if external mediation has properly happened. For this reason, during the teaching-learning process, it is essential to design a continuous feedback of previous knowledge systematic assessment it has been categorised in the semantic memory in to be able to use the most adjusted mediators to the needs of each contextual situation.

Limitations of the Study
The group is large and numerous in scope to facilitate the generalisation of the data, however, the main limitation is determined by the limitation to a single provincial level, as only imports relating to this area of work have been analysed.

Declaration of Conflicting Interest
The author declares that there is no conflict of interest, as the author himself has been in charge of diagnostic analysis in the provincial team for 10 years in the official public service of the Galicia Government.

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