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Comparative Analysis of Diagnosis and Behaviour by Gender (Men and Women) in People with Autism Spectrum Disorder

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ABSTRACT: People with Autism Spectrum Disorder (ASD) constitute a specific group, characterized by neurobiological developmental alterations in systemic development at the level of psycho- cerebral information processing, which affects men at a higher prevalence of 3–4 men for every woman, although little research has been conducted into the causes of this difference. This study aims to analyze, where applicable, whether there are possible differences in the gender variable (men and women) in relation to the etiological basis and subsequent behavior or profile. To this end, a total of 116 participants with ASD took part, comprising 87 men and 29 women, who agreed to answer the questions posed in a questionnaire-interview. As can be seen from the results, it can be concluded that, in relation to the determining statistic of the KMO and Bartlett's Test factor analysis, the group of men presents a greater explanatory load with regard to the genetic burden associated with the 15q11-q13 chromosomal region (sig: .02); whilst the 'diseaseway' variable shows a greater explanatory load in the women's group in the coefficients of the linear regression analysis, with a significance level of 0.02 in the determinant statistics.
However, in no case can significant general differences in the diagnostic process be corroborated between the codes; yet, the coping component in relation to the genetic burden and/or early-onset diseases is clearly differential in the codes of the "gender" variable, which may lead to confusion with current analyses of the diagnostic process.

KEYWORDS: Autism Spectrum Disorder, differences in diagnosis between men and women with ASD, behavioral differences related to 'gender', genetics and autism, early-life illnesses and autism, environmental and organic factors within the family.

INTRODUCTION

People with ASD constitute a specific group characterized by neurobiological developmental alterations in systemic development at the level of psycho-cerebral processing of information. Currently, the concept of ASD, according to the American Psychiatric Association's International Classification DSM-V (2013), is characterized by basing the diagnostic process on two basic dimensions: 1) the social dimension, based on social interaction and/or communication, and 2) specific behavior, characterized by the presence of stereotyped and/or restrictive behaviors, according to three levels of intensity, with level 1 being the least severe, whilst level 3 represents the most severe form of ASD across both dimensions.
This conceptual empirical definition has drawn on previous empirical hypotheses, such as the executive dysfunction hypothesis (Ozonoff, Pennington, & Rogers, 1991), the central coherence hypothesis (Frith, 1989) or the empathy-systematization hypothesis (Baron- Cohen, 2009). However, scientific progress in this specific area (Tavares, Fernandes, Antunes, Ferreira, & Prata, 2022) has enabled this conceptual framework to evolve; it has been complemented by the identification of cognitive biomarkers, raising, for the first time, neuroscientific hypotheses supported by clinical neuroimaging studies.
These studies demonstrate the experimental presence of more or less severe alterations in the connectivity of information neuroprocessing in individuals with ASD (Vasa, Mostofsky, & Ewen, 2016). Thus, deficits in connectivity may exist from the very moment information is received through sensory perception, as already indicated by neuroscientific hypotheses regarding neural connectivity and the limitations on the development of connections between incoming information and concepts stored in permanent memory or long-term memory. This is due to the specific way in which people with ASD organize concepts in the brain and synchronize them across specific brain regions, as demonstrated by numerous MRI studies of people with ASD, both in relation to themselves and in comparison, with neurotypical control groups, in connection with studies on the empirical measurement of visual and linguistic tasks (Bressler & Menon, 2010; Shirer, Ryali, Rykhlevskaia, Menon, & Greicius, 2012; Smith et al., 2009).
Based on the conceptual definition of the disorder, it has been observed that it predominantly affects men, at a ratio of approximately

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3–5 men to every 1 woman (Loomes, Hull, L., & Mandy, 2017). Initially, it is difficult to explain this bias, as the processing follows the same cerebral-cerebellar pathway in both genders; however, although this gender ratio is gradually decreasing, the X-chromosome vulnerability model hypothesis suggests that structural and genetic differences between the X and Y sex chromosomes contribute significantly to the higher prevalence of Autism Spectrum Disorder (ASD) in males, which could be an initial, albeit incomplete, explanation (Ferri, 2018; Frazier, Georgiades, Bishop, Hardan, 2014, & Skuse, 2000).

Precisely for this reason, there is currently an experimental interest in attempting to demonstrate possible differences in etiology according to the 'gender' variable, as well as its specific characteristics, including neurocognitive performance; however, these previous studies are limited by neuropsychological measures and their etiology as determined by the current official classification (Pellicano, 2007); as well as by other analyses based on underlying self-referential processes, in accordance with ecological assessment models, regarding comparisons of cognitive limitations between both genders with ASD (Albein- Urios, Youssef, Kirkovski, & Enticott, 2018; Demetriou et al., 2021; Wallace, Kenworthy, Pugliese, Popal, White, Brodsky, & Martin, 2016).

For this reason, the rise in such research shows that several scientific groups have investigated potential biases in the practices and measures of current diagnostic assessment processes, which, as they confirm, are closely linked to biased behaviors stemming from the different social expectations associated with the male and female genders (Koening & Tsatsanis, 2005). These authors have proposed several relevant hypotheses regarding this bias, arguing that the items are centered on typical masculine values that underpinned the development of the current International Classification, which are composed of measures of the extreme masculine psychosocial traits (Baron-Cohen, 2002; 2009), constituting the symptomatic criteria of the disorder.

However, the explanations are so varied that Robinson, Lichtenstein, Anckarsäter, Happé, & Ronald (2013) suggest that there may be a protective genetic factor at play in women's coding, meaning that they may require a greater genetic load to exhibit an autism-like symptoms, a finding that has also been corroborated by another group of authors (Gilman, Iossifov, Levy, Ronemus, Wigler, & Vitkup, 2011; Skuse, 2007, *op. cit.*). Other studies suggest that milder cases of ASD in women may even go unnoticed due to social expectations, in which competitiveness is socially attributed to men, who demonstrate it through far more disruptive and competitive behaviors (Dworzynski, Ronald, Bolton, & Happé, 2012). Moreover, men tend to score higher on behavioral problems, creating significant inconsistencies in their lives, which could also lead to confusion during the diagnostic process (Mandy, Chilvers, Chowdhury, Salter, Seigal, & Skuse, 2012; Szatmari, Liu, Goldberg, Zwaigenbaum, Paterson, & Woodbury-Smith, 2012), whilst women score higher on internalizing and externalizing symptoms (Bolte, Duketis, Poustka, & Holtmann, 2011), which has also been corroborated by comparative studies between genders with ASD (Øien et al., 2017; Øien et al., 2018). This suggests that social demands have influenced the criteria and values of these measures, which have focused on diagnostic criteria and subsequent behavior, without taking into account the cerebellar-brain connectivity circuit, which could provide information that would facilitate a more balanced analysis between the genders.

This study, which has many limitations regarding the differential population sample for male and female codes, seeks to provide a more detailed explanation of the gender differences, which are clearly set out in the final section of the study's Conclusions.

OBJECTIVES

Given the evidence of a significantly higher prevalence of ASD in men compared to women, who are substantially fewer in number, with regard to the etiological process and subsequent behavior, the overall aim of this study is to provide an empirical contribution, based on an experimental differential-comparative analysis, regarding the reasons for these differences.

METHOD

Research design

The design of this research is based on an empirical process, drawing on the analysis of a sample obtained via an *ad hoc* questionnaire, comprising the responses to key variables that have demonstrated high statistical reliability regarding the etiology of the disorder and subsequent behavior.

Participants

A total of 116 participants with ASD across the three severity levels (N: 116) took part in the study, of whom 87 were men and 29 were women.

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The study was conducted in two consecutive phases. Initially, a joint experimental study was carried out for the 'gender' variable and, in a second phase, the initial joint database was subdivided according to the 'gender' variable—men and women—in order to specify the differential findings as precisely as possible, should any exist.

Variables

The variables used in the study were: 'gender' (male, female), 'level' (1, 2, 3) in accordance with APA guidelines (2013), 'genesway' (no deficit, 15q11-q13 region, 17q11-q22 region, 7q31-q33 region, SHANK2,3 genes, combination of different genetic alterations), the variable "familyway" (no deficit, severe anxiety or distress, schizophrenia, severe depression, organic neurological disorders, various combined illnesses), in relation to any family member, particularly the closest relatives; and the variable "diseaseway" (no deficit, encephalitis, meningitis, various severe infections, cerebral convulsions, various combined conditions), particularly when these occurred before the age of three.

Data analysis and procedure

The analysis carried out was statistical in nature, based on tests provided by the SPSS 22 program, and was conducted in two phases. Phase I included the empirical analysis of the entire general database (N: 116), based on multiple contingency analysis and the *t-test* for independent samples, performed according to the "gender" variable.

Phase II involved a more specific analysis based on the 'gender' variable, for which the database was subdivided according to this variable (men: n=87 and women: n=29), in order to precisely identify any potential differences separately and to enable a subsequent statistical comparison, based on the calculation of linear regression analysis and factor analysis for the variables selected for each subpopulation.

PHASE I: STATISTICAL ANALYSIS FOR THE ENTIRE DATABASE

In the first section, Phase I provides an overview of the cross-tabulation for the five variables in the analysis of the entire database (N: 116), which offers a broad and general view of the comparisons or differences in relation to the 'gender' variable, as shown in Table 1.

Indeed, the table shows that, when the analysis focuses on the dynamic variables or factors of the study, the first substantial differences in the diagnostic process in relation to gender are observed in the 'genesway' variable. Thus, across the different levels, boys show the presence of a specific genetic etiology in 46 cases, with various specific etiological conditions, notably 20 participants with abnormalities in the 15q11-q13 chromosomal region (22.98%). Meanwhile, the women's code shows genetic etiological conditions in only 10 participants (34.48%).

However, the male group stands out in the 'diseaseway' variable, to which a total of 37 participants belongs, having suffered various conditions particularly before the age of three (42.52%); compared to the female group, which has 16 cases, correspond to this same "diseaseway" variable, in reference to the three levels of ASD severity (55.72%).

Table 1: Contingency analysis: genesway * diseaseway * sex * level * familyway Crosstabulation (N: 116).

familyway	level	gender	genesway		diseaseway						Total
no deficit					no deficit	encephalitis	meningitis	infections	convulsions	various	no deficit
	level 1	males	genesway	no deficit	13	5	5	5	1	4	33
				15q11-q13	3	0	1	0	0	0	4
				SHANK2,3	0	0	0	2	0	0	2
				various	0	1	0	1	0	1	3
		females	genesway	no deficit	2	1	1			4	8
				15q11-q13	1	0	0			0	1
				17q11-q22	0	0	0			1	1
				7q31-q33	0	0	0			1	1
				various	2	0	0			2	4
	level 2	males	genesway	no deficit	1	1	0	1	1	2	6

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				15q11-q13	1	1	0	0	0	2	4
				7q31-q33	0	0	1	0	0	0	1
		females	genesway	no deficit	1	1		1			3
				15q11-q13	1	0		0			1
				7q31-q33	1	0		0			1
	level 3	males	genesway	no deficit	0	2	0	1			3
				15q11-q13	3	0	0	0			3
				various	0	0	1	0			1
anxiety	level 1	men	genesway	no deficit	3						3
	level 2	males	genesway	15q11-q13	1						1
		females	genesway	no deficit	1						1
				15q11-q13			1				1
schizo.	level 1	males	genesway	no deficit	2			1		1	4
				15q11-q13	0			0		1	1
				various	0			0		1	1
		females	genesway	no deficit	1		1				2
	level 2	males	genesway	15q11-q13	1						1
		females	genesway	no deficit		1					1
	level 3	males	genesway	15q11-q13	1						1
depression	level 1	males	genesway	no deficit		1					1
		females	genesway	15q11-q13	1					1	2
	level 2	males	genesway	no deficit	1					0	1
				15q11-q13	1					0	1
				various	0					1	1
organic	level 1	males	genesway	15q11-q13	2	0					2
				SHANK2,3	1	0					1
				various	0	1					1
		females	genesway	15q11-q13			1				1
	level 2	males	Genesway	15q11-q13	1	1		0			2
				various	0	0		1			1
		females	genesway	15q11-q13	1						1
various	level 1	males	genesway	15q11-q13	1					1	2
	level 2	males	genesway	no deficit		1					1
	level 3	evils	genesway	15q11-q13				1			1

Furthermore, the 'familyway' variable also shows relatively significant differences. Thus, boys account for a total of 23 cases (26.43%), 4 related to anxiety disorders, 6 to schizophrenia, 1 to depression, 1 of various organic etiologies and 4 across various conditions. Conversely, among girls there are 5 cases (17.24%), 2 relating to anxiety disorders, 1 with schizophrenia, 1 with depression and 1 of a diverse organic nature, a figure significantly lower than that of their male counterparts.

Taking into account these differences in the number of cases relating to the 'gender' variable, in the cross-tabulation table for the five variables—where the number of data points for the 'men' code is much higher than for 'women'— the output data do not appear to explain with sufficient clarity the significant differences in relation to this variable between men and women at the etiological level; therefore, to address this limitation, a more empirically robust test has been carried out, based on the comparative *t-test* for independent samples (see Table 2).

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Table 2: Independent Samples Test.

		Levene's Test for Equality of Variances		t-test for equality of means					% confidence interval	
		F	Sig.	*t*	Df	Sig. (2-tailed)	Mean difference ce	Stan dard error	Lower	Upper
level	Equal variances assumed	5.47	.02	.93	114	.35	.12	.13	-.14	.39
	Equal variances not assumed			1.1 1	69.18	.27	.12	.11	-.10	.35
diseaseway	Equal variances assumed	3.92	.05	-.94	11	.34	-.40	.42	-1.24	.44
	Equal variances not assumed			-.86	41.98	.39	-.40	.46	-1.33	.53
genesway	Equal variances assumed	1.31	.25	-.89	114	.37	-.31	.34	-.99	.37
	Equal variances not assumed			-.84	43.66	.40	-.31	.36	-1.05	.43
familyway	Equal variances assumed	1.42	.23	.49	114	.61	.16	.32	-.47	.79
	Equal variances not assumed			.54	57.21	.58	.16	.29	-.42	.74

In this regard, the *t-test* is not as clear-cut as the cross-tabulation of data from the contingency analysis, as it shows only two significant results, which raise doubts regarding possible differences in relation to the 'gender' variable: as can be seen in the output data for the 'level' variable (sig: .02), which clearly differs between the male and female codes, and the 'diseaseway' variable, whose level of significance lies on the critical threshold of significance (sig: .05). For this reason, the descriptive data are not as definitive as they might appear, perhaps due to the small sample size or the difference in the number of participants for the 'gender' variable, which is much higher for the male code than for the female code. The other variables, both the genetic variable "genesway" and the issues related to the "familyway" variable, do not show significant differences when the analysis is carried out for the entire data set.

II) PHASE: SPECIFIC ANALYSIS ACCORDING TO THE "GENDER"

Regression analysis for the male code of the "gender" variable

In the summary of the predictive regression model (see Table 3), the variables "familyway", "genesway" and "diseaseway" are combined into a single factor, which explains only 11% of the differences found in the "level" variable with respect to the men's code for the "gender" variable.

Table 3: Model Summary.

Model	R	R Square	Adjusted R Square	Standard error of the Estimate
1	,11(a)	,01	–0.01	,68

a) Predictors: (Constant), familyway, disesaway, genesway and diseaseway.

Meanwhile, the ANOVA analysis of the regression analysis (see Table 4) reveals a close relationship between the factors in the analysis and the dependent variable (DV), where the significance level indicates that the R value is not zero (0.55), with a mean R^2 value of 0.18, suggesting that these variables are moderately correlated (sig:
.75).

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Table 4: ANOVA(b).

Model		Sum of Squares	df	Mean Square	F	Sig.
1	Regression	.55	3	,18	,39	,75(a)
	Residual	38.84	83	.46		
	Total	39.40	86			

a) Predictors: (Constant), familyway, genesway and diseaseway.
b) VD: level.

In the meantime, the coefficients from the regression analysis indicate that the constant of the standardized factors indicates a differential level (sig: .00), which is not subsequently reflected in each factor analyzed separately (see Table 5).

Table 5: Coefficients

.Model		Standardised coefficients	t	Sig.
		Beta		
1	(Constant)		12.57	.00
	diseaseway	-.041	-.36	,71
	genesway	.080	.69	,49
	familyway	.067	.59	.55

a) Dependent variable: “level”.

In the following histogram, referring to the regression analysis for the ‘men’ group of the ‘gender’ variable, the frequencies found by levels for the DV: ‘level’, in relation to the ‘men’ code, can be seen visually (see Figure 1).

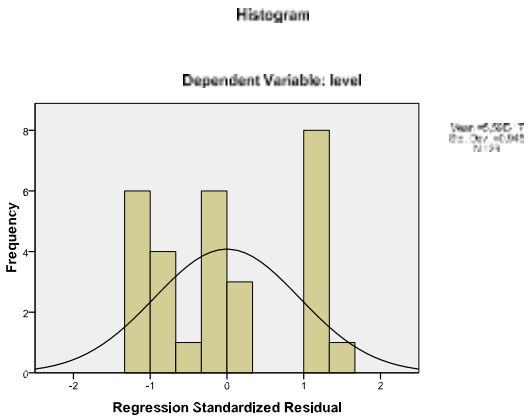

Figure 1: Histogram for the ‘men’ code.

These same data found for the ‘men’ code of the ‘gender’ variable can be viewed more clearly in the residual plot in Figure 2, which shows very little dispersion and a high degree of clustering according to the ‘gender’ variable in line with the three levels of ASD severity.

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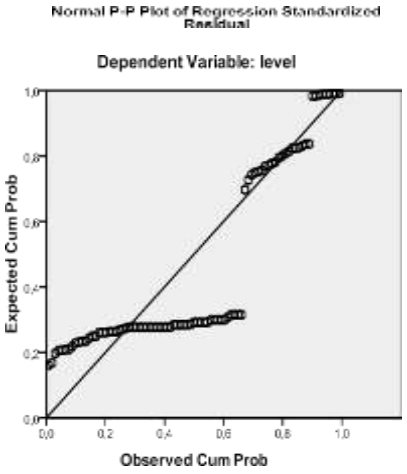

Figure 2: Normal P-P plot of regression. Standardised residual. Dependent variable: level

Regression analysis for the 'female' code of the 'gender' variable

The regression analysis model captures the overall systematic nature of the three grouped explanatory factors in the analysis in a single factor (see Table 6).

Table 6: Regression Analysis Model Summary for the 'women' code.

Variables included/excluded[a]			
Model	Variables included	Variables removed	Method
1	familyway, diseaseway, genesway[b]	.	Introduction

a. Dependent variable: "level".

b. All requested variables entered.

As with the 'men' code, the R value is not zero, although it is higher than that for the 'women' code. Meanwhile, the R^2 value indicates that 21.6% of the variance is explained by this variable in determining the etiology of ASD across its three levels of severity (see Table 7).

Table 7: Model Summary for the women's code.

Model	R	R-squared	Adjusted R Square	Standard error of the Estimate
1	,46(a)	,21	,12	,44

a) Predictors: (Constant), familyway, diseaseway, genesway.

Furthermore, the ANOVA analysis, which complements the regression analysis by summarizing the values of variability in relation to the 'gender' variable and allowing for the examination of estimates and contrasts for the parameters captured by the regression model, indicates that it cannot be concluded that the factors are related (F: 2.29), with a significance level of .10 (see Table 8).

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Table 8: ANOVA.

Model		Sum of squares	df	Mean square	F	Sig.
1	Regression	1.33	3	.44	2.29	.10[a]
	Residual	4.86	25	.19		
	Total	6.20	28			

a. Predictor variables: (Constant), familyway, diseaseway, genesway.
b. DV: level.

The coefficients shown in Table 9 below, which present the model estimates, confirm significant levels for the 'constant' (sig: .00), but more importantly, the 'diseaseway' variable (sig: .02) is also positive (sig: .00), in contrast with the analyses relating to their male counterparts.

Table 9: Coefficients (a).

Model		Unstandardised Coefficients		Standardised Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	1.550	,140		11,043	,00
	diseaseway	,090	,038	-422	-2.333	,02
	genesway	-,044	,048	-0.165	-,912	,37
	familyway	-0.013	,066	-,036	-,197	,84

a) Dependent variable: "level".

The histogram shows a more normalized curve in relation to the regression analysis data (see Figure 3). There is a degree of homogeneity in relation to greater variability, with a much less homogeneous dispersion (see Figure 4).

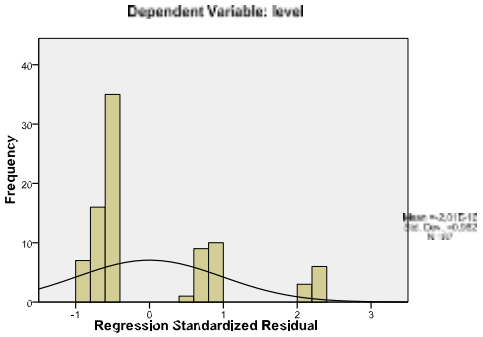

Figure 3: Histogram

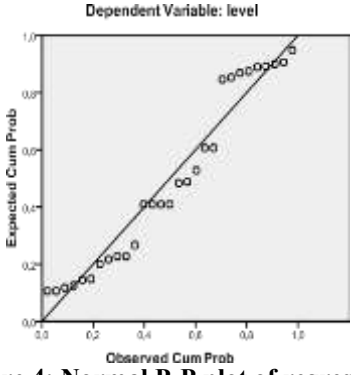

Figure 4: Normal P-P plot of regression.

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Factor analysis for the 'men' code of the 'gender' variable

To explore the similarities and/or differences in relation to the "gender" variable, a factor analysis was carried out for the male and female codes, using two separate subsets of data. Regarding the 'men' code, firstly, the covariance matrix is observed: 18.38, indicating a medium-low score, with a medium KMO statistic (.43) and a very small p-value in the Barlet test, which confirms a highly significant sample adequacy of the data (sig: .02), as well as the values of the total explanatory variance (see Table 10). This confirms a relatively good sample adequacy of the data for conducting the factor analysis.

Table 10: Correlation matrix.

Table: KMO and Bartlett's Test

Kaiser-Meyer-Olkin measure of sample adequacy.		.43
Bartlett's test of sphericity	Approximate chi-square	9.62
	df	3
	Sig.	,02

a. Determinant = 18.38

In a second step, the amount of variability in each variable explained by the factors can be analyzed; therefore, it is desirable for the communality statistic prior to factor extraction to be high, as it indeed is (initial: 1.00). Furthermore, it must remain high during factor extraction, which is also the case, as shown by values above .70 for the three independent variables or factors in the study, confirming that there is a high degree of commonality between the variables and the corresponding factors for the 'gender' variable code (see Table 11).

Table 11: Commonalities.

	Initial	Extraction
diseaseway	1.00	,84
genesway	1.00	,70
familyway	1.00	,79

a) Extraction method: Principal Component Analysis.

The total variance of the first factor, "genesway", explains up to 42.66% of the etiology and behavior of autism, slightly ahead of the "disaseway" factor (35.54%) and both already far ahead of the "familyway" variable (21.79%), from which it can be deduced that, for the male cohort, the total explanatory variance of a genetic nature is fundamental to explaining the etiological process of ASD (see Table 12).

Table 12: Total Variance Explained.

Component	Initial Eigenvalues			Extraction Sums of Squared Loadings		
	Total	% of Variance	Cumulative %	Total	% of variance	Cumulative %
genesway	1.28	42.66	42.66	1.28	42.66	42.66
diseaseway	1.06	35.54	78.20	1.064		
familyway	,65	21.79	100.00			

a) Extraction Method: Principal Component Analysis.

These same data have been corroborated by the matrix component analysis, which shows high association loadings between the variables, with the 'genesway' variable explaining a much higher percentage than 'diseaseway', although it rises slightly in the 'familyway' factor (.60). Component 2 is significantly influenced by the "diseaway" variable (.79) (see Table 13).

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Table 13: Component matrix.

	Component	
	1	2
genesway	,84	,04
diseaseway	,45	,79
familyway	,60	-,65

Extraction method: Principal component analysis.

Factor analysis for the 'women' code of the 'gender' variable

Firstly, a very high determinant value (.92) and a relatively moderate KMO value (.56) were observed, but there was no positive critical level of significance; nevertheless, this confirms that the sample is suitable for factor analysis (see Table 14).

Table 14: Correlation matrix[a]

KMO and Bartlett's test

Kaiser-Meyer-Olkin measure of sample adequacy.		0.56
Bartlett's sphericity test	Approximate chi-square	1.92
	dg	3
	Sig.	.58

a. Determinant = 0.92

The communalities statistic is initially very high (1.00) prior to factor extraction. However, following extraction, they decrease significantly in relation to the men's code, which is a clear point of difference compared to their female counterparts; consequently, the explanatory power of each factor in terms of variance is relatively lower (see Table 15).

Table 15: Communalities.

	Initial	Extraction
Genesway	1.00	,41
diseaseway	1.00	.40
familyway	1.00	,50

Extraction method: Principal Component Analysis.

However, as can be seen from the amount of explained variance, the percentage of variance explained is also much higher for the 'genesway' variable (44.01) compared with the other two factors: 'diseaseway' (29.29) and 'familyway' (26.69) (see Table 16).

Table 16: Total explained variance.

Component	Initial eigenvalues			Sums of squared saturations of the extraction		
	Total	% of variance	Cumulative %	Total	% of variance	Cumulative %
genesway	1.32	44.01	44.01	1.32	44.01	44.01
diseaseway	.87	29.29	73.30			
familyway	.80	26.69	100.00			

Extraction method: Principal Component Analysis

The component matrix thus reveals a single dominant component, with the 'genesway' variable (.64) ranking first, closely followed by the 'diseaseway' variable (.63) (see Table 17).

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Table 17: Component matrix.

	Component
	1
familyway	-.71
genesway	.64
diseaseway	.63

Extraction method: Principal component analysis.

The main conclusion regarding the regression analyses and factor analysis is that the male code is significantly more closely related to the genetic basis as the primary etiological factor in their diagnosis, unlike its female counterparts, who appear to share the etiology of ASD between the genetic type and the superimposed high incidence of severe illnesses before the age of three, including during pregnancy, without there being a specific genetic burden.

DISCUSSION

Indeed, most comparative studies, with regard to the 'gender' variable, suggest differences in behavioral regulation and in aspects related to cognitive ability; these studies would likely be more comprehensive if their data were also compared with that of neurotypical individuals. Studies published by Demetriou et al. (2021, *op. cit.*) show that women, both those with ASD and neurotypical women, show better cognitive performance in establishing connections between the perception and processing of stimuli compared to their male counterparts. This facilitates contextual retrieval and the relationship between the subcomponents of long-term memory, namely semantic memory and episodic memory. A priori, this may be due to sociocultural and environmental differences between the genders at a social level. During this process, women dwell more on the specific aspects that detail the initial sensory perceptual concepts, which facilitates access to the information received in a contextual manner rather than in isolation, as is the case with their male counterparts. This allows women to demonstrate greater resilience to the symptoms of autism (Kreiser & White, 2014; Reilly, Neumann, & Andrews, 2016; Van Wijngaarden-Cremers, van Eeten, Groen, Van Deurzen, Oosterling, & Van der Gaag, 2014). This difference, stemming from the dominance of social roles (Pellicano, 2007), influences both the basic diagnostic tests and the clinicians who administer them.

To better understand the etiology of gender differences, whilst genetic alterations or the consequences of illnesses suffered in early childhood—including during pregnancy—may not differentiate between genders, the effects arising from these can indeed influence outcomes in highly differential ways. From visuospatial networks in the reception of stimuli to the language associated with such information between the two sexes. However, interrelated connective limitations in processing will always be observed in comparison with their neurotypical peers. Women demonstrate better coping during the connectivity process, at social, academic and behavioral levels.

It is logical that linear regression analyses indicate that people with ASD in the male group are more likely to score higher on the items of the diagnostic scales and tests than people without ASD; however, compared to the female group, they tend to score lower on the symptomatic processes characteristic of ASD, particularly on the language index, whilst higher scores on sensitivity measures, which allows them to focus on the stimulus and its context in a much more holistic way than men, whilst also demonstrating a greater capacity to tolerate thresholds of disconnection, compensating for these with better joint attention than men.

In neuropsychological terms, the impairment observed in people with ASD in terms of the functional connectivity of neural networks is less pronounced in women, due to the compensatory mechanisms that occur in many cases at the genetic level. In men, reduced activation is observed in the medial prefrontal cortex, the superior temporal sulcus, the anterior insula, the anterior cingulate cortex and the amygdala during the neuropsychological processing of information. This leads to impairments in cognitive-social and behavioural components throughout life. At the diagnostic level, this is reflected in the difference in scores obtained on various tests and scales developed on these principles, often leading to fundamental errors in diagnosis. (Prat, Stocco, Neuhaus, & Kleinhans, 2016; Subramanian, Brandenburg, Orsati, Soghomonian Hussman, & Blatt 2017).

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CONCLUSIONS

As can be seen, despite the study's limitations, it is possible to conclude that, in men, there is a higher value for the KMO and Bartlett's Test factor analysis statistic regarding genetic load in relation to the 15q11-q13 chromosomal region (sig: .02); however, with regard to women, the 'diseaseway' variable shows a higher explanatory load, even yielding a critical index in the coefficients of the linear regression analysis, in the determinant statistic, at a significance level of 0.02. However, in no case can a significant general differential analysis be corroborated in the diagnostic process between the codes in relation to genetic burden or early-onset diseases. Nevertheless, the coping component in the face of genetic burden or early-onset diseases is clearly differential in the codes of the "gender" variable; which, in a detailed analysis of the existing correlations, already hints at the initial contingency analysis. This difference relates to women's greater resilience, due to their specific attentional condition, which is already observed from the moment the initial perceptual stimulus is perceived; furthermore, although to a limited extent, they pay attention to the context in which it occurs. This allows working memory to cognitively execute nodes that facilitate incoming information not only accessing semantic memory, but also creating a slight episodic memory. This subsequently facilitates the establishment of a link between semantic information and the relevant context, enabling a response and subsequent action appropriate to the situation. Whereas the male code, due to its more hyperactive nature and scattered attention, gathers the concept or information directly, without paying attention to the learning context, making it difficult for working memory to transfer the concept to semantic memory. This would be an impediment to form relationships that generate a minimum of episodic memory. When faced with a specific request, they would make many errors in their responses or answer questions that have nothing to do with the specific request, as the information is not contextualized. Therefore, in addition to any differences in etiological factors, the greatest differences become evident in how they cope with psychosocial situations, which clearly differ between men and women. This may even mislead the clinician conducting the diagnostic process, due to current tests and scales, which are designed according to extreme values based on male norms.

In summary, there appears to be a greater genetic predisposition in men; however, the most significant differences in the 'gender' variable may relate to how the brain circuit copes with symptoms in men. In women, we face the challenge posed by the specific nature of the diagnosis and the design of tests that are biased towards one gender.

LIMITATIONS

Logically, two fundamental limitations must be highlighted: firstly, the small size of the total sample; and secondly, the limitation arising from the higher number of responses from the male group (n=87) compared to the female group (n=29), as it is very difficult to obtain adequate samples in such specific studies that have the necessary consent. Nevertheless, the study serves as a reference for subsequent studies of larger scale and empirical validity.

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