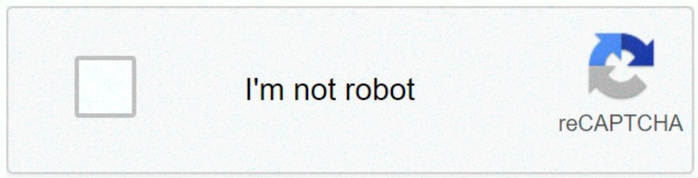


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# Autismo pdf articulos

Autism, now defined as autism spectrum disorders, was initially described in 1943. It is characterised by changes in communication, social interaction and a limited spectrum of the patient's interests. It is usually identified early in development from 18 months. Currently, autism is considered a neurological disorder with a spectrum that encloses varying degrees associated with genetic, non-genetic and environmental factors. Several syndromes associated with this disorder were referred to within genetic factors. Autism was also studied at the genetic, neurphysiological, neurochemical and neuropathic level. Neuroimaging techniques showed several structural abnormalities in these patients. Related changes have also been observed in the Serotonin, GABAergic, Catechomergic and Cholinergic systems. This work provides an update on information about the genetic and neuro-industry aspects of autism spectrum disorder. The autism spectrum disorder (ASD) was described in 1943 and is defined as a developmental disorder that affects social interaction and communication. It is usually identified in the early stages of development from 18 months of age. Currently, autism is considered a neurological disorder with a spectrum covering cases of different grades, associated with genetic factors, not genetically and environmentally. Among the genetic factors, several syndrome was described associated with this disorder. The neurobiology of autism was also studied at the genetic, neurophysiological, neurochemical and neuropathic levels. Neuroimaging techniques showed several structural abnormalities in these patients. There have also been changes in the serotonergic, GABAergic, categorical and cholinergic systems associated with this disorder. This paper provides an update of the information presented in the genetic and neuro-and-neuro-accentins aspects of autism spectrum disorder. 1Introduction Autism, now defined as autism spectrum disorders (ASD), was initially described by Dr. Leo Kanner in 1943. This is characterised by changes in social interaction, communication and a limited spectrum of patient interests. Diagnosis is made in infant stages. Patients are described as withdrawn children, which are an unusual way of relating, with little language or little communication, attached to routines, which display foreign repetitive behaviors, unusual forms of play, and lack of emotional rehearsal towards people1. Currently, autism is considered a broad-spectrum neurological disorder that entails cases of varying degrees associated with genetic and environmental factors whose manifestation is variable. It is usually identified early, starting at 18 months of age. In addition, it has been suggested that changes in several genes in combination with the presence of non-genetic factors are the cause for developing the phenotype corresponding to autism, which represents, in itself, a set of atypical genetic changes that generate the same phenotype2.2DiagnosisThe diagnosis of autism, or ASD (ASD) , is based on clinical study. A fully reliable biological marker has so far not been identified. However, based on the various symptoms indicating asD, different specialists suggested an understandable, structured and systematic strategy, both for diagnosis and treatment, in order to identify the different specific capabilities and limitations in each patient with ASD4. Some of the symptoms occurring in autistic patients also occur in children with mental disabilities without autism. For example, patients with autism may show different degrees of cognitive deficiency, and in turn, patients with intellectual disabilities can develop stereotyping and communication problems characteristic of ASD patients. However, this is a problem for the accurate diagnosis of autism5. This is why several tests are required for the contem of this set of pathologies. To do this, medical specialists, as well as researchers in the field, designed various questionnaires. In addition, the criteria established by the American Psychiatric Association are found in the recently published Diagnostic and Statistical Manual of Mental Disorders or DSM-V-TR, in which diagnostic criteria were enhanced by agreeing and simplifying them. In addition, the latest findings derived from genetic and neuroimaging studies, specifically for each of the ASDs, are integrated into this tutorial. Similarly, the symptoms that prevent and cover different categories of diagnosis are recognised and outlined, after which the clinical perspective is expanded. Specific criteria have been consolidated and include autism disorder, Asperger's syndrome and widespread developmental disorder on the autism spectrum. Finally, the classification of other disorders, such as bipolar and depression, is optimized. All this to help in a manner consistent with diagnosis in clinical practice6. The specific criteria for diagnosing ASD are derived from three domains. Influence of social interaction: a) impairment of the use of non-verbal communication such as eye contact, facial expression and body; b) inability to develop relationships with age peers; c) lack of sharing or communicating love and interests with others; d) limited interest or idea about the reactions and emotions of others. Qualitative changes in communication skills: a) delay or lack of language acquisition; b) inability to start or have a conversation; (c) the use of language in a stereotypical or repetitive manner, or the use of idiotic language; d) non-existent development of age-appropriate imitation or pretend games. Presence of restrictive or repetitive patterns in behavior: (a) concern exacerbated by a limited number of unusual interests; b) uncompromising adherence to certain habits or routines; c) auto stereotyping; d) concerns or exaggerated attachment to parts of objects. Diagnosis of autism is confirmed when the individual demonstrates a total of six or more behaviors from the three domains mentioned, including at least two of the first domain. Once the diagnosis is made, confirmation and severity of the symptoms are present important. For this, a second questionnaire is usually applied, such as the CARS (Childhood Autism Rating Scale) consisting of 15 questions, each with seven possible answers5. Longitudinal studies in children at high risk of developing ASD from an affected older sibling as well as retrospective analysis of high-year videos of children of a year with this condition confirm that in some of these patients the characteristic symptoms can be identified from 6 to 12 months of age, these symptoms are more evident from 18 months8. It is important to mention that although the literature cites as symptoms initial to the various changes in language and communication, the longitudinal studies mentioned above have shown that other not-so-distinctive symptoms can be identified before 6 months, such as irritability, hypoactivity or hyperreactivity to the environment, hiccups or hyperactivity, as well as a deficiency in the development of thick movements. Therefore, it is important that first-level doctors are aware of the warning signs within the symptomatic spectrum that a child can present with ASD, so that relevant screening tests are carried out in the first case, and subsequently confirmation of diagnosis as early as possible.3Prevalence Epidemiological studies in the world have identified ASD patients in the included countries, with similar prevalence rates10. Autism disorder is estimated at 2/1,000 individuals4. Currently, about one in 175 children around the world are born with this disorder, although the frequency varies in each country. In the United States, Based on the Network's Community Report of the Autism and Developmental Disabilities Monitoring Network, the Centers for Disease Control indicates that one in 68 children is diagnosed with autism. Men have five times the risk of developing ASD as girls. Similarly, the data in this report suggest that most diagnoses are made after the age of 4, and the prevalence is higher in Caucasian children than in African-Americans or Hispanics11. By 2013, the estimated incidence for autism in Mexico was one in 300 children. As a prognosis, you can talk about at least 115 thousand children with autism in Mexico, with a risk of 6,200 new cases a year12.4Genetics of ASD Autism considering genetic changes, which are usually heterogeneous, among its main causes. These changes occur at different levels of organization of genetic material. Genetic material, during cell division, is organized into superstructures called chromosomes, demonstrating so-called chromosomal changes in which macro- or micro-dentions, duplications, insertions and insured investments of the genetic material can occur. It should be said that, even at the molecular level, there may be de novo point mutations in the DNA sequence that alter genes or promoters and affect gene expression. Many of them are related to the development of the nervous system. The main autism-related syndrome is mentioned below.4.1 Prader-Willi syndrome (SPW)The syndrome is the result of a drop of chromosome 15, region q11-q13, whether through mutation of the paternal gene or by single parent dysomy of maternal origin, that is, when the paternal allele is not expressed or there is a change in the methylation pattern. This syndrome occurs 1-4% of cases of autism, and its symptoms are hypotonia, intellectual disability, obesity, food greed, obsessive-compulsive disorder, low sociality; are individuals who speak in excess and who have high levels of oxytocin13.4.2 Angelman Syndrome observed 2-4% of autistic and are affected the same region as SPW, although the change comes from the mother side. This can occur as an inversion, duplication, or mutation of the UBE3A/E6AP gene. This gene encodes for a ubiquitous E3 leagues participating in the protein degradation pathway in neurons. Individuals who have this change have hyperactivity, hand flapping, seizures, intellectual disability, epilepsy, strativism and very low language skills; there may also be manger guideism or microcephaly14,15.4.3Sydrome Fragile XWith has an incidence of 4-8% in patients diagnosed with autism, this syndrome is characterized by intellectual disability, macroarchy, pervasive and repetitive language, poor eye contact and distinctive facial dysmorphia. Alteration of the FMR1 gene (fragile X-mental delay 1) located on the X-chromosome usually comes from a dated state of the mother side, so the effects are seen mainly in males. In this gene, a series of repeated trinukleoticides (CGG) (5 to 45 times) is found at the end of the messenger RNA, which regulates the translation of ed gene. However, duplications of this region can increase to more than 200 repetitions of trinucleotide, producing the syndrome. This increase in trinucleotides in the gene's messenger RNA prevents its translation, and since the rna binding protein it negatively regulates no messengers that modulate synaptic plasticity, Timothy's intellectual development16.4.4Fieldme in this syndrome has been detected spontaneous mutations in the CACNA1C gene, which interferes with the functioning of calcium channels. This gene is located on chromosome 12, region p13.3. In addition to the autistic phenotype there are deadly a rhythmias, congenital heart disease, immune deficiency, hypoglycaemia and cognitive disorders17.4.5 Rewrite Syndrome Rett mainly affects women, and in the case of men heterozygous is fatal. In this syndrome, the MeCP2 gene is mutated, in the long arm of the X chromosome. This gene is related to brain development. This syndrome is characterized by severe auto-autistic phenotype, sheal regression, stereotypical movements, atatoxic gait and lack of social interaction. The MeCP2 protein is responsible for silencing methyromatin in cytocine of the CpG18.4.6 paresOtras genetic changesIn common, autism is associated with various syndrome in which behavioral changes occur, language development or socialising, thus genetic diagnosis is used to detect whether the individual has any of this syndrome (Table 1)19–32. In addition to the syndrome's own shamptomatology, a percentage of cases are diagnosed with autism if the diagnostic criteria prevail33. Several of these syndromes also have components such as mental delay, epilepsy and heart disorders. In this regard, autism also has a high percentage mental delay (75%) and a lower percentage of epilepsy component (42%). Most autism-related mutations correspond to genes involved in neuronal development and synaptogenic. As for unicuniting autism with epilepsy, mutations in genes involved in the exculpator system (glutamate) and neuronal inhibitor (GABA). The type 6 glutamate receptor gene (mGluR6) is in ligation imbalance in some individuals with autism; that is, they do not separate independently and have low recombination because the two loci in question are usually on the same chromosome. In addition, the decline in GABAergic system enzymes and gaBA availability were detected in autistic. In addition, changes in the 15q11-13 region include genes from GABAA receptors. On the other hand, the family of neuroligines (NLGN1, NLGN2, NLGN3, NLGN4X and NLGN5, genes distributed on chromosomes 3, 7, X and Y) also plays an important role in synopsis and the imbalance between inhibition and neuronal arousal. Polymorphism association studies in the NLGN3 and NGLN4 genes with autism have not found a clear relationship. However, isoforms of these genes related to autism were

found34. Genes described in some syndrome are also factors that cause epilepsy in autistic, such as the CDK5, FMR1, ARX (aristaless-related homeobox) genes, which in turn regulate in brain development, neuroblast spread and migration of GABAergic interneurons) and the MeCP2 gene, which in turn regulate DLX5. In addition, other genes with mutations encoding for voltage-dependent neural sodium channel subunits SCN1A (Alpha 1) and SCN2A (Alpha 2) trigger attacks in autistic35. The tuberculosis sclerosis complex is caused by mutations in the TSC1 and TSC2 genes. Symptoms such as epilepsy, neurocognitive disorders and autism occur. TSC1 and TSC2 proteins are moduleming cell growth mediated by the mTOR signaling pathway, which also liquidates synoptogenesis36. Linguistic development is one of the critical components of autism. Several language-related genes are changed. The changes described are localized in the AUTS loci and include genes involved in brain development. The AUTS3 bacus, located in the 13q13.2-14.1 region, contains genes for neural migration and development (NBEA, MAB21L1, DCAMKL1 and SMAD9). The AUTS1B bacus (7q31) contains at least two genes associated with autism. Candidate genes affecting the development of the central nervous system are WNT2 (7q31-33), which is expressed in thalamus, and FOXP2, which regulates genes for language and speech development. In the same lure is the MET genes, whose alternative promoter halves their expression in autism, affecting the maturation and growth of the neocortex (new bark or the most recent bark). In 7q35 there is another important gene for the CNTNAP2 of the contactin-related protein in the neuroxin family. In mice that do not express this gene, it has been observed to exhibit symptoms of autism; Similarly, the decreased expression of this gene due to variations in its promotion region or loss of methylation sites occurs in some autistic individuals. However, changes in this gene are related to a large number of neural development disorders37. The AUTS1A locus (7q36) contains the EN2 gene, the mutation of which involves reducing Purkinje cells and cerebellar hypoclasia. The long arm of chromosome 2 contains the auts5 locus, which binds to the delay in the construction of sentences and for which no responsible gene has yet been identified, but was associated with polymorphisms of the RAPGEF4 gene in 2q31-32. The 15q11-13 region with the auts4 bacus contains the genes UBE3A, ATP10A, GABRB3, GABRA5, GABRG3; In addition, it is subject to influences in its methylation pattern (genomic imronta) leading to linguistic dysfunction38. Autism genetics show the involvement of genes involved in nervous system development and implications for language development, socializing, behavior, and even neural changes. New molecular tools, such as identification in the number of copies of variation, de novo mutations, gene expression micro trees, genome sequence, and mass follow-up will allow the vast majority of cases of idiopathic autism to be investigated39. Identifying genes associated with language development, socialization and behavior will allow to establish the interactions between them and introduce the molecular mechanisms involved in autism40,41.5Neurendocrine aspects of ASDS are disorders in neurodevelopmentally characterized by changes in social interaction, communication and repetitive behaviors. They affect 1% of the population and their appearance is higher in males. Therefore, most studies were done in the male sex. Autism neurobiology was studied at the genetic, neurophysiological, neurochemical and neuropathic level. Neuroimaging techniques have shown several structural abnormalities, but not consistently. Changes were found in the serotonin, GABAergic, catecohollyrgic, cholinergic, among others, but without specificity or diagnostic value42. Bauman and Kemper showed the neuropathological findings of a 29-year-old man43 in 1985. In 1998, they completed a series of nine cases of which four suffered from epilepsy and five, intellectual disability, without obvious malformations and normal myelinization. Compared to non-autistic subjects, they increase in cell density in the limbic and cerebellum system. In addition, they observed the decline in the range of pineritic branches in pyramidal neurons CA1 and CA4 from the hippocampus44. Recent studies have established that main perimeters in autistic neonates are normal at birth, but at the age of 2 there is an extension of the head, and from 3-4 years it increases about 5-10%45.46. This increase in head perimeter has been associated with a decrease in cortical layers and in the maturation of the cortex. Another theory suggests there is a secondary response to neural remode events that cause overgrowth46. There is also dysfunction in cortical areas, including frontal, temporary and cingulated cortex, which affects and promotes attention issues and executive function in charge of planning and organization, causing a lack of autonomy and decision-making, as well as dependence on authentics47. Other authors link hippocampus and amygdala dysfunction (medial temporary lobe structures), which affect work or recognition memory and verbal coding depending on the severity of autism48. Moreover, studies conducted by Lai and collaborators at the Autism Research Centre at the University of Cambridge have suggested that autism affects different parts of the brain in women and men. Magnetic resonance imaging found that the anatomy of a person with autism's brain differs depending on their sex49. This may involve physiological mechanisms leading to sexual dimorphism, such as prenate sex hormones and sex-linked genetic mechanisms. Because the frequency of autism in women is lower than in men, this difference is an important example of diversity within the spectrum50. The importance of some neurotransmitters, such as Serotonin, in behavioral disorders has been shown. In hyperkinetic children who have low plasma Serotonin levels, their clinical improvement has been shown to depend on the increase in Serotonin. Schain and Freedman51 also have a high concentration of serotonin (26%) in the autistic investigation; these Serotonin levels decreased by limiting the content of Tryptophan (precursor amino acid) in the diet. Based on the above, a common phenomenon has been observed in subjects with ASD hyperserotoninmia52. However, treatment with selective Serotonin Recurrence Inhibitors (SSRI), such as fluoxetine, paroxetine, fluvocamine and venlafaxine, has positive effects on the stereotype of repetitive behaviors, social deficiency and elsewhere, the dopamine system is linked to analysis, planning and execution functions; in addition, with behavior of motor activity, social activity and perception. Barthelemy and collaborators analyzed urinary categolamine levels in autistic, and found low dopamine levels and high norepeinephrine, causing passive behavior as seen in autistic 54. In the brain, minors with autism showed poor connections in areas that release dopamine in response to rewards, compared to children without autism. In the left lobe of the brain, autistic children showed poor connections to the accumbens nuclear and ventral tegmental area. And on the right there was a weak connection to the amygdala, which processes the emotional signals. In subjects with ASD with the treatment of dopamine anthagonists such as haloperidol and risperidone, which are antipsychotic medications, an improvement in irritability and hyperactivity behavior has been observed55. On the other hand, in postmortem tissue from autistic was found to be a decrease of GABAergic compounds in Purkinje cells in cerebellum43. Recent studies have shown a decrease in receptors (GABAA and GABAB) and proteins in cerebellum and cortical areas, suggesting a deregulation of GABA's inhibitory system in autism, Affecting circuit and behavioral regulation56,57.6Errors in metabolism in ASDThe main metabolic changes that present an autistic phenotype are phenomenon stonuria, changes in the urea cycle, changes in the metabolism of purines and deficiencies of the enzyme succinate semialdehyde dehydrogenase. Some authors have found hyperuricaemia in patients with intellectual disabilities and personality disorders, as well as other biochemical defects that can be caused not only by inadequate intake of their precursors, but also by defective absorption, as can be seen in celiac disease characterized by fat and gluten intolerance. Such gluten intolerance causes damage to the intestinal epithelial, causing bulky stools of fats and other uninsured substances (statorrhea), while a growth disorder in autistic people with celiac disease is seen. When these children underwent a gluten-free diet, the stiptomatology of autism disease decreased. The presence of two diseases in the same patient does not necessarily mean that one is a consequence of the other, but can have the same genetic bases and for that reason they are presented together. Finally, the neurotoxics that cross the placenta barrier are dangerous. Roberts and co-workers reported that if the mother is exposed to pesticides during pregnancy, In agricultural areas, the risk of the fetus developing autism58.7Treation7.1 increases six times. Pharmacological treatmentUp until now there is no specific or curative treatment for autism. Existing treatments can be divided into pharmacological and psychopathic. All drug treatments are symptomatic. Many drugs have been used in the management of this condition,59-61 but there is no one unanimously accepted or helpful to all patients. Haloperidol can be useful in reducing impulsivity and aggressiveness, 59,61 as well as stereotyping and emotional accountability, but it is important to be aware of its possible early and late side effects (dyskinesias, excessive seductiveness, etc.). It is recommended to use it for short periods of time or intermittently. Other reports showed equal effectiveness of risperidone, albeit with fewer side effects, making it the most widely used drug currently62. There have been reports that indicate a high activity of endogenous opioids in the central nervous system of autistic63,64, and this motivated the use of naltrexone65.66 opiate antagonist. However, the results were poor and are currently almost unused. Similarly, it is claimed that there are changes in Serotonin metabolism, with significant height of Serotonin concentration67. This led to the use of serotonin recurrence inhibitors, such as fluvocamine and sertraline,68,69 with good results in reduced repetitive thoughts and ritualistic behaviors, as well as reduced aggressiveness, in addition to improvement in language use and social behaviour, although it is noted that the beneficial effect can only be transient. There are no medications to act on the basic manifestations of autism. Sometimes some of the associated issues need to be addressed. Epilepsy is treated according to epileptological principles, without any specific aspect. Because most seizures are complex focus, carbamazepine is one of the drugs indicated. When there is an activity disorder with indicative deficiency, ritalin (three daily doses of 0.4-1mg/kg) can be used. Buspirone (5mg, three times a day) can be used for anxiety. Naltrexone (0.5mg/kg/d) was used for aggression. Studies have been conducted in recent years with risperidone, an atypical antipsychotic that blocks post-synoptic serotonin receptors; it can be more easily displaced by endogenous dopamine, which reduces the risk of collateral neurological effects. The dosage used is 0.01-0.03mg/kg of body weight in two daily doses over 8 weeks. Its favorable effect on self and heteroagresiveness, abnormal movements, inaction and hyperactivity are quite noticeable. Side effects are moderate drowsiness and decay, especially at the beginning. One problem that sometimes forces medication to stop is increased appetite and marked weight gain. In girls, amenorrhea may occur, another indication of stopping the medicine. When dosages greater than 3.5mg/d are used and for long periods, dyskinesia and tremors can occur70.7.2. Psychophomic treatment Psychopathic therapy plays a central role in the treatment of autistic people. The most accepted management today is the early onset of treatment, intensive and multimodal type: language therapy, socialization programs, multiple sensory stimulation (hearing, visual, aesthetic), recreational therapy, etc. Unfortunately, in this area has emerged several methods covered with pseudoscientific bases, which only bring confusion and false expectations into the family of these patients (delphynotherapie, equilibrium, use of other pets, aromatherapy, music therapy, among others)59-61,71. Treatments used with autistic are educational and behavioral programs, which focus on developing social skills, speech, language, self-care and work skills. Mental health professionals provide advice, training and treatments based on each child's needs as they cannot be generalized as each case has specific characteristics and needs. Specific treatment will be determined by the doctor based on the following criteria:•The age of the child, general health and medical history•The degree of disorder•The symptoms of the child•Tolerance of certain medications or therapies•Expectations for the evolution of the disorder•The opinion or preference of parents Is important to note that the treatment of this developmental disorder is focused on certain symptoms. However, this does not mean that treatment will eliminate the disorder or change the behavior of child71. Conflict of interestThe authors declare they have no conflict of interest. 

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