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THE ENIGMA OF AUTISM: CONTRIBUTIONS TO THE ETIOLOGY OF THE DISORDER

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ABSTRACT. The lack of a definitive explanation for the causes of autism in children is an enigma that creates significant suffering among parents and difficulties for health professionals. This study is a critical review of the possible causes of autism, currently known as Autism Spectrum Disorder (ASD), spanning the period from the first description of the syndrome in 1943 until 2015. The objective of this article is to outline the current scenario of studies about this type of disorder in order to emphasize the points of convergence and the differences between the positions taken by the researchers who have dedicated themselves to this topic. The analysis suggests four main paradigms that attempt to encompass the etiology of autism: 1) the Biological-Genetic Paradigm; 2) the Relational Paradigm; 3) the Environmental Paradigm; and 4) the Neurodiversity Paradigm. By questioning these paradigms, we hope to deepen comprehension of this disorder in the current scientific context.
Keywords: Autism; Asperger's syndrome; literature review.

O ENIGMA DO AUTISMO: CONTRIBUIÇÕES SOBRE A ETIOLOGIA DO TRANSTORNO

RESUMO. A ausência de uma explicação definitiva sobre as causas que levaram ao desenvolvimento do autismo em seus filhos é um enigma que gera grande sofrimento aos pais e dificuldades aos profissionais da saúde. Neste estudo foi realizada uma revisão crítica sobre as possíveis causas da síndrome autista, atualmente denominada de Transtorno do Espectro Autista (TEA), desde a primeira descrição realizada em 1943 até 2015. O artigo visa delinear o panorama atual de estudos sobre este tipo de transtorno a fim de explicitar os pontos de convergência e as diferenças entre os posicionamentos de pesquisadores que se dedicam ao tema. A análise sugere quatro paradigmas principais que pretendem circunscrever a etiologia do autismo: 1) o Paradigma Biológico-Genético; 2) o Paradigma Relacional; 3) o Paradigma Ambiental; e o 4) Paradigma da Neurodiversidade. Problematizar esses paradigmas constitui uma maneira de aprofundar a compreensão a respeito deste transtorno no contexto atual do desenvolvimento científico.
Palavras-chave: Autismo; síndrome de Asperger; revisão de literatura.

EL ENIGMA DEL AUTISMO: CONTRIBUCIONES A LA ETIOLOGÍA DEL TRASTORNO

RESUMEN. La ausencia de una explicación definitiva sobre las causas que llevaron al desarrollo del autismo en sus hijos es un enigma que genera gran sufrimiento a los padres y dificultades a los profesionales de la salud. En este estudio se realizó una revisión crítica sobre las posibles causas del síndrome autista, actualmente llamado Trastorno del Espectro Autista (TEA), desde la primera descripción realizada en 1943 hasta 2015. El artículo busca delinear el panorama actual de estudios sobre este trastorno a fin de explicitar los puntos de convergencia y las diferencias entre las posiciones de investigadores dedicados al tema. El análisis sugiere cuatro paradigmas principales que pretenden circunscribir la etiología del autismo: 1) el Paradigma Biológico-Genético; 2) el Paradigma Relacional; 3) el Paradigma Ambiental; y 4) el Paradigma de la Neurodiversidad. Problematizar estos paradigmas constituye una manera de profundizar en la comprensión de este trastorno en el contexto actual de desarrollo científico.

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Palabras-clave: Autismo; síndrome de Asperger; revisión de literatura.

Introduction

"Autism? Why does this happened to my son? What does this cause?" Parents ask when they hear the diagnosis given to their child who is not developing as expected. This is one of the most difficult medical enigmas to be solved because it involves numerous pieces of a complex puzzle.

Data published in 2014 by the Center for Disease Control and Prevention [CDC] (Centers for Disease Control and Prevention, 2014) reveal that autism reaches a high rate of 1 in every 68 children aged eight years in the United States, in the proportion of 4.5 boys to 1 girl. The data from 2012 indicated 1 in every 88 children. It is assumed that part of the increase in the number of cases is due to the change in diagnostic criterion and the increased awareness of the disorder in all areas of health and in society. There are still no official statistical data in Brazil, but it is estimated that about two million people could be included in the diagnosis of autism, considering both the Brazilian population of approximately 200 million (Instituto Brasileiro de Geografia e Estatística, 2016) and the prevalence of the disorder of 1% in the population as reported by the American Psychiatric Association [APA] (2014).

Officially, its history began in the early 1940s, when, coincidentally, two Austrian psychiatrists, Leo Kanner and Hans Asperger, one living in the United States and the other in Austria, respectively, described, in the period of the World War II, a list of criteria to diagnose the autistic syndrome. Since then, a number of paradigms have been taking shape around the subject.

We opted to use Thomas Kuhn's (1978) concept of paradigm defined as a "constellation of beliefs, values, techniques, etc..., shared by the members of a particular community" indicating "concrete solutions of puzzles" (p. 218, our translation), because we identified different conceptions of autism implicit in the research studies.

The review of the scientific literature was performed using two databases, the Virtual Health Library (VHL) and PubMed (US National Library of Medicine/National Institutes of Health), with the keywords: "autism and cause"; "autism and etiology"; "autism spectrum disorder and causes"; "autism spectrum disorder and etiology". Initially, this review was restricted to empirical articles published between the years 2010 and 2015. However, articles considered relevant, prior to 2010, classic books on autism and theoretical articles, were included as well, for a better understanding of the subject progress. The selected research studies were published in Portuguese, English or Spanish.

It was observed that these research studies could be arranged into four groups, each with its belief or view of autism, its probable cause, and proposals to solve it. Thus, four paradigms that circumscribe the etiology of autism are suggested. They are: 1) Biological-Genetic Paradigm; 2) Relational Paradigm; 3) Environmental Paradigm; and 4) Neurodiversity Paradigm. These paradigms will be discussed from what is considered a starting point for each one of them until nowadays in the world and Brazilian scenarios.

Throughout this text, we will use the expression *person diagnosed* with autism and similar terms. This choice aims to prevent any bias towards one or another paradigm, as there is a subtlety that underlies the use of the expressions *person with autism* and *autistic person*, which will be discussed at the appropriate time.

First paradigm: Biological-Genetic

In 1943, Leo Kanner wrote the seminal article "Autistic Disturbances of Affective Contact", describing systematically 11 children with peculiar characteristics that he named early child autism. Kanner assumed that the disorder had a biological origin, and concluded that children were born with "innate inability" (p. 250) of establishing contact with other persons.

However, if on the one hand Kanner (1943, 1949) theorized that the origin could be biological, on the other hand he described complicated family relationships. He affirmed that among the children regarded as autistic at the time, there were very few really warm parents, the child seemed to live in a

frosty atmosphere coexisting with two strangers, and the pregnancy of most of these children was not welcome or even desired. He described the parents as highly intellectualized, however inexpressive, introverted, obsessive, uncreative, cold, and kept their children "in refrigerators which did not defrost" (Kanner, 1949, p. 425).

A year later, Hans Asperger (1991) carefully described four boys with behavioral characteristics similar to those described by Kanner, however milder. Asperger labeled the phenomenon observed autistic psychopathy in childhood, using a derivative of the word autism naming the syndrome without knowing the work of Kanner (1943). In this period of Second World War, Kanner lived in the United States and Asperger in Austria.

Asperger (1991) also identified unusual traits in parents or relatives, but associated them with a possible genetic factor. His hypothesis was based on a 10 years longitudinal study with 200 families with children regarded as autistic. Unfortunately, this study was never published. According to Grinker (2010), his work was lost after a bombing destroyed his laboratory during that war. Nevertheless, citing this preliminary study, Asperger (1991) concluded that the origin of autism should be researched in the genetic (genetic fault, heredity) and environmental fields (birth injury, encephalitis).

More than forty years later, in 1968, the English psychiatrist Michael Rutter suggested again a longitudinal study of families who had twins with the then rare autistic condition, in order to investigate the genetic-based etiology hypothesis of autism (Rutter, 1968). Later, Folstein and Rutter (1977) published a study with 21 pairs of twins, 11 identical (monozygotic) and 10 fraternal (dizygotic), in which 25 of them were diagnosed as autistic. The results reinforced the hypothesis of genetic determination, and demonstrated that environmental influences, such as brain injury in the perinatal period, could cause autism independently or combined with a genetic predisposition. However, what and how it was inherited remained unknown.

In the subsequent decades, many researchers tried to answer this question, either through more detailed longitudinal studies with a much larger number of participants that reiterated the genetic inheritance (Constantino et al., 2013; Nishiyama, Notohara, Sumi, Takami, & Kishino, 2009; Sandin et al., 2014), or through research studies with contrasting results that sought to identify possible hereditary genes of autism (Freitas et al., 2014).

The findings showed different defects in the candidate's genes. These sporadic genes were attributed to spontaneous mutations (*de novo*). The terminology, *de novo*, represents the mutations that were not inherited from parents and are written in italic in the scientific articles. This means that segments of DNA were deleted or duplicated, however these same variations in the DNA were not found in the parents (Freitas et al., 2014).

Thus, the genetic factor for the development of autism could occur by genetic inheritance background and /or by spontaneous and random mutation of the genes.

One of the most prominent research studies in 2014 indicates that the genetic factor, in itself, is responsible for 50% of the cases of autism. The research was conducted from 1986 to 2006 with more than two million of families in Sweden, including identical twins, fraternal, brothers, half-brothers from the father's side, half-brothers by the mother's side, and cousins. Families with single children were excluded. The remaining 50% would be caused by environmental factors not described or identified in the research (Sandin et al., 2014).

A new study in 2015 with 85 families with two children diagnosed with Autism Spectrum Disorder (ASD), of the University of Toronto, Canada, indicated by sequencing of the genome that brothers have, in the majority of their DNAs, different genetic mutations. This was the opposite of what was expected (Yuen et al., 2015). The coordinator of the study said in an interview: "We believe each child with autism is like a snowflake; one is unique from another" (University of Toronto, 2015, para. 6).

Is it possible to say that future medicine will be personalized in order to find the best medicine for each person? This is what the biologists Patrícia Braga and Alysson Muotri, who are at the head of the research project, are investigating in the University of São Paulo (USP), called "The Tooth Fairy", which uses a cell reprogramming technique (http://www.projetoafadadodente.com.br/index1.html). The project collects baby teeth of children diagnosed with autism when they fall or are extracted. The cells in the tooth pulp are reprogrammed, becoming again embryonic cells (stem cells). These cells, in turn, can become any cell, even neurons to be studied *in vitro*. The objective is to identify biological differences in

autistic neurons, monitor their functioning and test new medications with the likelihood of a cure for autism (Gomes, 2014).

The biological factor that composes this paradigm refers more to research studies that claim the existence of general alterations in the central nervous system, such as sensory problems in the process of symbolization (theory of mind), in the mirror-neuron system and in the brain anatomy.

Caminha and Lampreia (2012) considered a sensory perspective when citing two assumptions: Carl Decalato's 1974 study defending that an innate impairment of sensitivity leads the child to perceive the environment in a singular way, making it difficult to communicate and relate to other persons; and Cindy Hatch-Rasmussen's 1995 study suggesting that the senses operate in isolation and the brain is not capable to organize these stimuli in a meaningful way.

The study of Baron-Cohen, Leslie and Frith (1985), conducted with children regarded as normal, autistic and with Down syndrome, concluded that children diagnosed with autism do not have the ability to attribute mental states to oneself and to others, a phenomenon that they designated theory of mind.

Giacomo Rizzolatti discovered the so-called mirror-neuron system in the 1990s, perceiving that mirror neurons were related to imitation, empathy and learning, and presented the hypothesis that a problem in this system could explain the difficulties in social communication of children regarded as autistic (Rizzolatti & Fabbri-Destro, 2010). However, other research published later challenged this theory by demonstrating that the functioning of the mirror-neuron system could be preserved in persons diagnosed with ASD (Fan, Decety, Yang, Liu, & Cheng, 2010).

The study of brain images of people diagnosed with autism resulting from magnetic resonance imaging, computed tomography, *post-mortem* and *in-vitro* brains, identified conflicting irregularities in the cerebellum, amygdala, hippocampus, limbic system, among others (Bernardi, Kirsten, & Trindade 2012; Freitas et al., 2014).

Second paradigm: Relational

The hypothesis of biological causality raised by Kanner (1943) was not suitable to the psychoanalytic theory that prevailed at the time, which focused on the symptoms' psychogenesis. Therefore, psychoanalysts gave more emphasis on Kanner's description of family relationships subsidizing the hypothesis of psychological origin of the disorder.

1967 can be considered the starting point of this paradigm with the publication of the reference book on autism by the psychoanalyst Bruno Bettelheim, although there are earlier publications on the psychological issue. Bettelheim (1967) affirmed that the disorder was caused by some destructive situation in the child psychological development, consequently, leading him/her to reject the world around him/her. Survivor of a Nazi concentration camp, Bettelheim defined these children as prisoners and the parents as Nazi guards.

It was in this context that the term *refrigerator mother* became popular in the United States, spreading, in the following decades, the hypothesis that attitudes, particularly the mothers', contributed or caused autism in children.

The parental blame effect, especially towards mothers, can be recognized clearly in biographies, where the parents expressed a desired pregnancy, described the gestation and arrival of the baby, as if they needed to defend themselves from moral judgment (Barnett, 2013; Paiva Junior, 2012; Salvador, 1993).

Psychologist Bernard Rimland (1964) contested the refrigerator mother theory, reiterating the biological origin of the disorder. When reading Rimland's book, Donald Winnicott (1997a), prominent English pediatrician and psychoanalyst, declared that, by defending the autistic syndrome based on a biological dysfunction, Rimland "does not show himself be fully up to date in regard to the study of the earliest stages of integration of the personality" (p. 178, our translation), during which the child depends entirely on the maternal care. He emphasizes that a book of this magnitude should not be written without also considering emotional aspects. Winnicott (1997b) considered autism as a problem of emotional development, and not a disease, and affirmed it to be possible to counteract the propensity to autism with psychoanalytical treatment, reversing this picture.

In Brazil, the Lacanian psychoanalytic branch assumes that autism can be caused by a failure of *maternal function* during the child's development making him isolate himself, preventing or hindering the development of his *self* and thus producing the autism. In this context, two possible failures are discussed: the lack or the excess of maternal function (Kupfer, 2000).

Third paradigm: Environmental

The emergence of the third paradigm, that is, of autism caused only by an environmental factor, can be situated in 1998, when the British physician Andrew Wakefield and his staff published the article "MMR vaccination and autism" in the journal The Lancet, linking the MMR triple vaccine (measles, mumps and rubella) to the development of autism. As a result, media linked the increase in number of diagnoses to the vaccination programs. However, the data was a fraud, and in 2010 Wakefield lost his medical license for unethical conduct (Haertlein, 2012; Poland, 2011).

Despite the identification of fraud, several other studies were conducted without any indication of correlation between childhood vaccinations, or thimerosal (mercury used as a preservative in the vaccines), and the development of autism. However the myth persists. In the United States and Europe, many parents still fail to vaccinate their children for lack of confidence in the immunization program, increasing the incidence of serious diseases such as measles (Poland, 2011).

A recent example of this situation occurred in December 2014, when the Department of Public Health of California reported a great outbreak of measles in the group of visitors and employees of the Disney parks. Most of them had not been vaccinated (California Department of Public Health, 2015).

Although vaccines and poisoning by mercury are no longer regarded as a threat by the scientific community, this paradigm gained strength in recent years with the increase of research studies linking other environmental factors to the development of autism, among which is noticeable Sandin et al. (2014).

For a better understanding of the current environmental risk factors, three main groups will be presented according to the kinds of agents, namely: (a) infectious agents that derive from a disease, such as congenital rubella and the cytomegalovirus; (b) the chemical agents that derive from contact with chemical substances, such as the use of valproic acid and the exposure to atmospheric pollution; and (c) what we called "associative agents", which derive from, for instance, the increase in parental age and gestational maternal diseases, such as diabetes and hypertension.

In the scenario of infectious agents, a study associated the congenital rubella with a high autism rate observed in 243 cases (Chess, 1971). Ten children were diagnosed with the classic autism of Kanner and other eight presented a "partial syndrome of autism" (p. 35). It was assumed that these eight children could be diagnosed with the mildest autism, of Asperger, whose work was not yet known in the United States in the 1970s.

Sweeten, Posey and McDougle (2004) reported three cases of congenital or perinatal cytomegalovirus in association with autism. One of the assumptions was that the immunological response to certain congenital infection would be related to the etiology of autism in babies already genetically vulnerable.

In the scenario of chemical agents, there is Schlickmann and Fortunato's (2013) literature review that suggests that the maternal ingestion of valproic acid during pregnancy may be related to autistic behavioral traits in children. Similarly, Raz et al. (2014) showed the influence of a greater maternal exposure to air pollution in the third trimester of pregnancy as a possible cause.

The scenario of associative agents refers more directly to the parents' health, for example, the increase in the mother and father's ages at conception. Grether, Anderson, Croen, Smith and Windham (2009) indicated that an increase of 10 years in the maternal age is associated with 38% rise in the chance of the child developing autism, and an increase of 10 years in the paternal age is associated with an increase of 22%. Kong et al. (2012) pointed out that the father's advanced age increases the possibility of spontaneous genetic mutations (*de novo*) that can cause developmental disorders.

The study of Walker et al. (2015) indicated that a pregnancy associated with hypertension increases twice the probability of autism or other developmental problems in the infant. Xu, Jing, Bowers, Liu and Bao (2014) associated maternal diabetes with a greater risk of autism in children.

Although environmental factors are solely correlational, and not entirely consistent, there are several alternative treatments proposed, depending on which agents are assumed in each case. According to the pediatricians Levy and Hyman (2008), the alternative treatments found to reduce the symptoms can be classified into: (a) Food supplements: vitamin B6, magnesium, dimethylglycine, melatonin, vitamin C, amino acids, omega-3, folic acid and secretin; (b) Pharmacological: antibiotics, antifungal agents, gastrointestinal medicines, hyperbaric oxygen therapy and immune therapies; and (c) Other therapies with gluten-free or casein-free diets, or chelation therapy, which promise the detoxification of the organism. However, there is still not positive scientific proof of these treatments.

Fourth paradigm: Neurodiversity

The fourth paradigm emerged from the discussion about disability and normality induce to by the first discussion on neurodiversity in 1999, conducted by the sociologist Judy Singer, diagnosed at the time with Asperger syndrome (Ortega, 2009).

The paradigm of neurodiversity sees autism as a diversity of human nature due to the atypical neurological development, consisting in a matter of identity that does not need to be treated. Therefore, it demands respect for differences, political positions and social rights (Akhtar & Jaswal, 2013; Jaarsma & Welin, 2012; Ortega, 2009).

This implies shifting from *to have* towards *to be*, in another words, people do not have autism, but they are autistic and is implicitly present in the use of expressions: person with autism and autistic person. By using the preposition *with*, the autism is seen as something acquired by the person at a certain time of his life and can be cured. Without the preposition and followed by a predicative, autism is seen as a different form of understanding the world.

The neurodiversity movement, supported by people diagnosed with high-functioning autism, is opposed by the parents of children diagnosed with low-functioning autism and intellectual disability, who affirm that their son or daughter does not have conditions to self-organize and defend himself/herself. These parents see the autism as a disease that helps them obtain rights to, for instance, treatments provided by the government (Ortega, 2009).

To subsidize this discussion, it is necessary to reflect on the categories of this disorder. According to Baron-Cohen (2002), there is no specific limit that can differentiate a high-functioning autism with mild severity (called Asperger's or Aspies's) from that of low-functioning with more severity (of Kanner). However, there seems to be some consensus that people diagnosed with autism who do not have a history of speech problems and have a normal, or above 85, intelligent quotient (IQ), present high-functioning autism. Data from CDC (2014) point out that 31% of children diagnosed with ASD have IQ below 70; 23% are in the boundary zone, between 71 and 85; and 46% have IQ above 85.

However, in the recent publication of the Diagnostic and Statistical Manual of Mental Disorders, DSM-5 (Associação Americana de Psiquiatria, 2014), the distinction was excluded, and today all types of autism are part of a single group, the ASD.

The following researchers are interested in discussing neurodiversity: Happé (1999) proposed the autism as a cognitive style; Baron-Cohen (2002) defended that the Asperger's individuals should not be seen as disabled; Voineagu et al. (2011) supported the concept of neurodiversity with their study of the human genome analysis; Ekblad (2013) defined neurodiversity through an interactive process using a questionnaire "aspie"; and Akhtar and Jaswal (2013) questioned whether normal is not just limited to "western, educated, industrialized, rich and democratic children", citing, for instance, that in China to point a finger and keep eye contact are not regarded as significant behaviors, and, therefore, are not considered a problem.

Meanwhile, in Brazil, families see the person diagnosed with the disorder as disabled, and in this sense, are mobilized to acquire specific rights for this group, in addition to the rights already granted to people with disabilities. As a result, on 27 December 2012 Law No. 12764 (2012) was passed, establishing the National Policy to Protect the Rights of Persons with Autistic Spectrum Disorder. This was considered as a progress in the area.

However, in the celebration of the World Awareness Day on Autism, in 2014, a banner was wielded by activists of the pro-autistic movement with the following message: "Autism: just a way to see and feel

the world" (Agência Senado, 2014). In other words, this message advocated that human diversity must be respected, and the person diagnosed with autism should not be regarded as a disabled person who needs different treatment.

Table 1 identifies these four paradigms disclosing their corresponding literature.

Table 1. Map of the literature used in the identification of the paradigms

Paradigm	References
Biological-genetic	Asperger, H. (1991). "Autistic psychopathy" in childhood. In: U. Frith (Trad.), *Autism and Asperger syndrome* (p. 37-92). Cambridge: Cambridge University. (Original publicado em 1944).
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Relational	Bettelheim, B. (1967). *The empty fortress: Infantile autism and the birth of the self*. New York: Free Press.
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Environmental	Chess, S. (1971). Autism in children with congenital rubella. *Journal of autism and childhood schizophrenia*, *1*(1), 33-47.
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Final considerations

The etiology of the autistic disorder remains inconclusive until now, thus generating suffering for the families who are unsuccessful at understanding what happened to their child. In the same way, this situation restricts health professionals – among them, psychologists – to make a decision about the appropriate way to assist children and parents. Figuring out the ASD puzzle is, undoubtedly, an important step, because it will allow more accurate diagnoses, prognoses and personalized treatments; in the best scenario, even eliminating the autistic symptoms.

As discussion about the etiology of autism has progressed, several assumptions for the solution of the enigma have been proposed according to views of man, not always explicit, assumed by different scientific communities.

The scientific community defender of the **biological-genetic paradigm** sees autism as a congenital neurological disease and defends the idea that its origin reside in the alterations of the central nervous system, particularly in the genes, which can be he inherited and/or undergo some spontaneous genetic mutation. Today, there is a debate regarding what genes are responsible for the manifestation of autism, since they are not found the same genes in the cases studied. Estimates indicate hundreds of genes involved in a complex genetic combination. Possibly this would be the explanation for a spectrum of symptoms and severity so varied.

The scientific community that advocates the **relational paradigm** sees autism as a psychological problem triggered in childhood because of a failure in the mother-infant relationship. This second paradigm affirms that the severity of characteristics is related to the singular combination of failure in maternal function and the way the child was affected in his emotional development.

The scientific community that proposed the **environmental paradigm** sees autism as a neurological lesion caused by the exposure to environmental agents during the prenatal, perinatal or postnatal period. In this third paradigm, the severity of the autistic characteristics depends on the duration and intensity of exposure to risk factors. However, researchers agree that there isn't a sole environmental agent responsible for autism, and that a genetic predisposition in the child may exist, concomitantly.

A combination between the genetic and the environmental paradigm is, today, the most widely accepted theory in the scientific community in general as the cause of autism, that is, a complex set of environmental and genetic factors that would lead to a neurological disorder with consequences for brain functioning (Chaste & Leboyer, 2012; Freitas et al., 2014; Sandin et al., 2014). Currently researchers are trying to demonstrate how genes interact with each other and with other environmental factors, in order to understand how the neuronal network produces autistic behavior, and hence discovering a medication capable of turning the autistic neurons into healthy ones.

The medication aiming at a cure proposed by the biological-genetic and environmental paradigms is contested by the **neurodiversity paradigm**, which sees autism as one of the several ways that human beings expresses their uniqueness, needing only to be respected and accepted as such, without great interferences, from a social or health area standpoint. In this respect, it was questioned whether a drug therapy, while reestablishing the brain functioning, would not make a person lose everything they have achieved in their development until then; and worse, whether this would not trigger a eugenics cleansing already in the pregnancy, when the biomarkers of autism are identified, preventing the birth of these babies. Besides, high expenses for families and government (Buescher, Cidav, Knapp & Mandell,

2014) and several other challenges to be faced daily during a lifetime are also mentioned (Hoefman et al., 2014).

Regarding a possible stigmatization of people diagnosed with autism, Asperger (1991), who experienced the horrors of Nazism, already manifested his concerns by emphasizing that he was convinced that they had the right to a place in the community.

The biological-genetic community defends itself emphasizing that medicines would only be another option for the treatment of people diagnosed with autism and, thus, the discussion about a "cure" or "non-cure" with drugs remains open and causing controversy.

In order to expand the debate of whether autism is or not a disease considering that the concept of normality is increasingly in check, we should think about paradigmatic changes in order to prevent stigmatizing and unnecessary labels. When considered disabled, the person diagnosed with autism differs from people with other forms of disability, since there can be changes in his/her position in the range of severity during his/her lifetime and could even leave the spectrum, as suggests by research conducted by Fein et al. (2013).

The results of Fein et al.'s (2013) study indicate the existence of a group of people with an early history of autism that, after therapeutic interventions, no longer meet the diagnostic criteria. However, how many children diagnosed with autism could achieve these results? Helt et al. (2008) asked this question, and after a review of studies on the improvement in clinical condition, concluded that between 3% and 25% of the cases that received continuous therapeutic treatment left the diagnostic characterization.

The DSM-5 (Associação Americana de Psiquiatria, 2014) seems to be in line with these results when informing that the severity of the two current diagnostic criteria – a) deficits in communication and social interaction and b) restricted and repetitive behaviors – may vary over time and according to the context, opening the possibility for autism to move in a scale for better, for worse or towards a remission of symptoms.

It can be inferred that a key piece of this puzzle would be to consider the person in question not as disabled, but being disabled. When considering him as disabled, there is a risk of imprisoning him in a label that freezes possibilities of good prognoses, while the use of the verb to be denotes an opening. It would mean a more dynamic view of the human being, which is developed from experiences with people who are significant to him. Even if a person has very severe autistic characteristics, the potential for development remains, beyond the label that can be attributed to him according psychologist Carl Ransom Rogers's propositions (Rogers & Kinget, 1975) of human psychological development: the actualizing tendency.

The actualizing tendency is an innate capacity that incessantly seeks to develop the person's potentialities. However, for this tendency to manifest itself effectively it is necessary for the person to experience affective relationships that are perceived as favorable to their growth.

The authors who pioneered the understanding of autism already advised about this since the 1940s, when, on one side of the ocean, Leo Kanner opted to call his patients by name and not by their diagnoses (Grinker, 2010, p. 51) and, on the other side, Hans Asperger concluded: "good and bad in every character are just two sides of the same coin" (Asperger, 1991, p. 89).

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