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Original Paper

Tracing the Origin of Autism

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Abstract

There is still no universally agreed definition on autism spectrum disorder (henceforth, autism) within the context of increasing prevalence of the condition worldwide. Perhaps the best way to define autism is to trace its developmental history in order to provide us a better insight of its genesis. In the beginning, nobody knew about autism. The biblical records of characters, such as Yusof of Jacob and Rachel in the Old Testament and a sick boy whose father brought him to Jesus to be healed in the New Testament, have provided us with telltale signs of the yet unknown condition. In the East, in the records of the Traditional Chinese Medicine (TCM) during the times from Song dynasty to Qing dynasty, prodromal symptoms of the yet unnamed condition of autism were noted. However, it was the West, in the early 19th century with the great European nosological tradition coming into existence, that the term of autism was eventually coined. Through tracing the developmental history of autism, the authors hope to gain a better understanding of the enigmatic syndromic disorder for the purpose of preparing appropriate inclusive treatment plans for individuals with autism throughout the world.

Keywords: autism, definition, nosology, symptoms

1. Introduction

In order to understand the origin of autism, we need to first understand what signs and symptoms are and how they are different from each other. Signs are physical manifestation of a condition which can be a disability, disorder or disease. They are objective and that means they can be observed and measured, usually by a professional diagnostician. Symptoms are not the same as signs though they have often been used interchangeably in literature. Symptoms are physical or mental features that indicate a condition (i.e., of a disability, disorder or disease) as experienced by an individual, especially when such features are apparent to him/her. Symptoms are more subjective and are also intangible in that only the individual can feel or experience but cannot see them. In addition, symptoms can be either positive or negative. The former ones are those that can be observed in a person with an abnormal condition, but not seen in typical or neurotypical (Note 1) people, while the latter ones refer to the absence of a function or feeling normally present in a neurotypical person. In this paper, we shall use signs and symptoms interchangeably to mean subjective features of a condition unless we specify that they are objective.

To understand what autism is, we need to trace its history – more precisely, its symptomatic history and how it is what it is today. It has never been an easy task to draw out its history, but we have decided to go along the line of studying the symptoms of an unknown, enigmatic condition of what was later called autism reported in the past. We are also quite aware of the Neanderthal theory of autism (Ekblad, 2001/2019; Ludwig, 2017; Morgan 2001/2007) but have decided not to include here for two reasons: first, it is not within the scope of this chapter, and second, more rigorous research is needed to validate the hypothesis. With our current findings of past records in our literature search, we are able to come up with four main phases of historical development in autism based on subjective/objective symptoms (see Figure 1):

(1) Phase of Prodromal Symptoms (subjective);

(2) Phase of Coexistent Symptoms (subjective/objective);

(3) Phase of Syndromic Symptoms (subjective/objective); and

(4) Phase of Complex Syndromic Symptoms (subjective/objective).

Each of these four phases (presented in a triangle), with the first phase having the biggest area and broadest base as there is a wide and diverse range of symptoms – primary, correlated, secondary and artifactual – through the second and third phases to the fourth phase at the peak with the smallest area to signify that further research will help to narrow the focus down to the etiology of autism, will be briefly described with examples below.

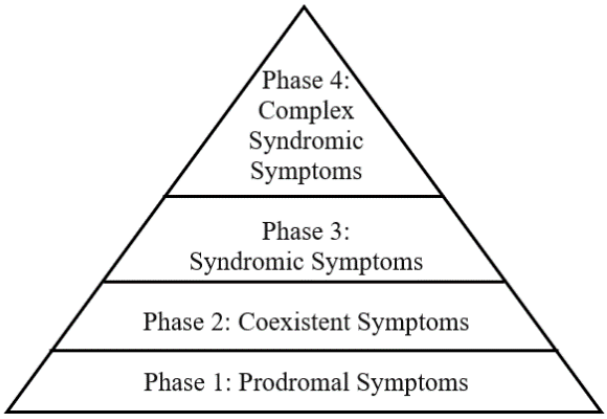

Figure 1. Symptoms-based Historical Development of Autism

The first phase involving prodromal symptoms. The term *prodrome* comes from the Greek word *prodromos*, meaning "running before." Hence, prodromal symptoms refer to subjective tell-tale signs of an unknown or yet to be identified condition or a condition of unknown origin. For example, prodromal symptoms of acrodynia, being very subjective or unclearly defined, may resemble those of pheochromocytoma or Kawasaki disease, and that can lead to a misdiagnosis of the condition. Hence, a differential diagnosis is needed to prevent such a problem from arising. In the ancient times, people would attribute such subjective symptoms to some kind of a spiritual disturbance caused by the demons or other spirits (see Xie, 2020, for detail).

The second phase concerns coexistent symptoms (they can be subjective or objective), which refer to a cluster of co-existing symptoms that may constitute a condition or a symptom (or more) that is shared by two or more conditions. For example, inattention, hyperactivity and impulsiveness are three objective coexistent symptoms that result in attention deficit- hyperactivity disorder (ADHD). The disorder also has other subjective coexistent symptoms, such as emotional dysregulation that can swing from rejection-sensitive dysphoria (Dodson, 2018) on one end to recognition-responsive euphoria (Hallowell, 2019) on the other end. In the DAMP syndrome (see Gillberg, 2003, for detail), which is a specific kind of developmental disorder, its three coexistent symptoms are **deficits in attention, motor control and perception with the initial of each symptom contributes to the acronym DAMP.** Between ADHD and DAMP syndrome, inattention is the coexistent symptom shared by the two different conditions (Landgren et al., 1996).

The third phase of syndromic symptoms refers to those signs and symptoms that are correlated with each other and often associated with a particular disability, disorder or disease. One good example is dyslexia that has been defined by Lyon, Shaywitz and Shaywitz (2003) as "a specific learning disability that is neurobiological in origin. It is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive

abilities and the provision of effective classroom instruction. Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge" (p. 3). The three coexistent symptoms are "difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities." The underlying or correlated symptoms (i.e., syndromic symptoms) are "a deficit in the phonological component of language … often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction" and "problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge".

The fourth and last phase focuses on complex syndromic symptoms. A complex refers to a core pattern of emotions, memories, perceptions, and wishes (EMPAW for short) in a person's unconscious mind being organized around a common theme (e.g., wealth, status and power). When the word *complex* is added to syndromic symptoms, it encompasses all prodromal, coexistent and syndromic symptoms that are experienced by an individual in his unconscious mind (Note 2), resulting in "distorted thought and sensory pattern that has been deeply ingrained into the person's psyche" (Ray, 2015, para. 1). In fact, the unconscious mind takes up a huge chunk of one's perception and decision-making in terms of how s/he relates to others, emotional experiences and sense of self. Psychological complexes (e.g., dependency complex, superiority complex, inferiority complex) are good examples with complex syndromic symptoms.

2. Phase 1 – Prodromal Symptoms

In beginning of the history of autism, nobody knew about the condition. Signs and symptoms related to autism were noted only centuries or even millennia later, but still nobody knew what they were. These constituted the prodromal symptoms of the yet unknown condition of autism.

Levine (2019) did an insightful study the character of Yosef (Note 3) (Joseph) (b.1 or 27 Tammuz-d.1444/45 BCE), son of Jacob and Rachel (as recorded in the Book of Genesis in the Old Testament of the Holy Bible), through Torah, Midrash, and classical Jewish sources and found that "Yosef's behavior, interpersonal relationships, and personal journey and development are often difficult to understand, and at times seem to defy explanation" (an abstract taken from Elsevier Social Science Research Network [SSRN], 2019). Levin (2019) as well as Greenberg (2019) postulated that Yosef could be on the autism spectrum. For instance, Yosef was not overly sensitive to the effects of his words and actions on, especially those close to him, like his brothers. According to Levine (2019), the absence of understanding the emotional world of others or "deep empathy" (also known as affective empathy (Note 4)) is understood as a clear indication of Yosef's autistic disorder. The Jewish texts refer to him as a *na'ar*, which implies his immaturity, such as playing with his hair and eyes, an indication of self-stimulation rituals rather than prettification (Greenberg, 2019, p. 61). In addition, there are several critical moments when Yosef spoke without waiting for responses, such as when he interpreted the chief butler's dream, when he told the Pharaoh the meaning of his dream, and when he revealed his identity to his brothers. These are monologues, described by Levine (2019) as "rambling", that "are understood as expressions of his lack of social appropriateness" (Greenberg, 2019, p. 61). This is an inferential diagnostic evaluation based on recorded narratives to identify Yosef as a potential candidate for autism.

Yosef's condition is also described as a form of psycho-emotional complex known as Yosef Complex (or Joseph Complex). According to the Segan's Medical Dictionary (2011), Joseph Complex is described as "[A]n allegorical term for intense sibling rivalry, which derives from Joseph – the favorite and youngest of the twelve sons of Jacob and given to birth by Rachel – the Israelite boy was cast out by his brothers" (para. 1). According to Chia (2021, slide #3), "A psychological and/or emotional complex is taken as a core pattern of behavioural or instinctual, neuro-cortical, and spiritual dimensions, respectively, consisting of the following: (i) emotions (affective; limbic system; spirit/heart); (ii) memories (cognitive; neocortex; mind); (iii) perceptions (cognitive-sensory; neocortex; mind); and (iv) wishes or desires in the personal unconscious (instinctual; reptilian brain; flesh) organized around a common theme, such as power or status" (also see Schultz & Schultz, 2009, for detail). Primarily, it is a psychoanalytic term, it is found extensively in the works of Carl Jung and Sigmund Freud.

If Yosef's case is not considered the first recorded narrative of autism, perhaps, according to Xie (2018),

the first case of autism can be found in three of the four gospels in the New Testament of the Holy Bible. It occurred sometime between AD 30 and 32, when a father brought his young son with purportedly autistic symptoms to seek healing from Jesus Christ. Xie (2018) listed five broad spectra of autistic symptoms – social interaction, communication, stereotyped behavior, sensory response/reaction, and task behavior – that have been noted in the gospel according to Saint Mark (see Mark 9:17-26), "ranging from one end that displays signs of low passivity to the other end that shows signs of high activity or reactivity to either external or internal stimuli (or even to both external and internal stimuli simultaneously)" (p. 28) as shown in Figure 2 below.

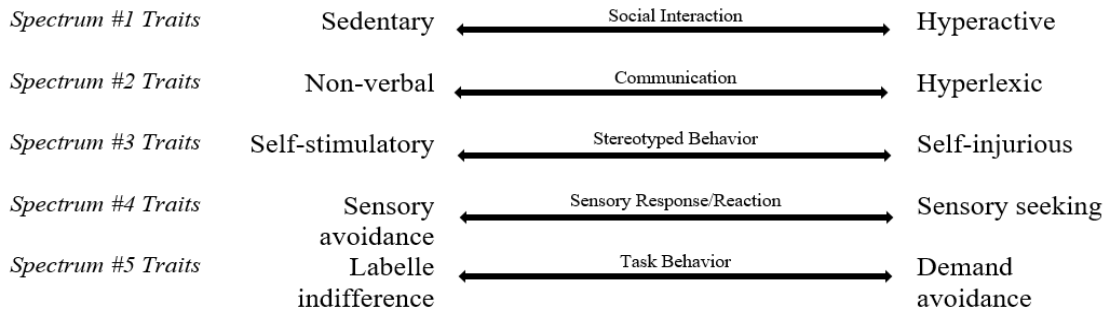

Figure 2. Broad Spectral Symptoms of Autism (Xie, 2018)

During the period between AD 960-1279, especially from the times of Song dynasty to the Qing dynasty, the ancient Traditional Chinese Medicine (TCM) records have been found on treatment for people with autistic conditions. In fact, it was during the Ming dynasty (AD 1368-1644) that peaked the highest number of TCM prescriptions for autism treatment (Cai et al., 2015).

During those ancient dynastic eras, TCM did not have a disorder called autism, but it recorded seven prodromal symptoms that today we recognize them as traits of autism (see Cai et al., 2015 for detail): (i) dullness (also known as dementia); (ii) mutism, being reluctant to speak and/or reply; (iii) soliloquy, i.e., absence of mind and muttering to oneself; (iv) five kinds of retardation, which include "slowness in standing, walking, hair growth, tooth eruption and speech" (Cai et al., 2015, p. 21); (v) five weaknesses, i.e., "flaccid head, flaccid nape, flaccid hands and feet, infantile flaccidity of muscle and flaccid mouth" (Cai et al., 2015, p. 21); (vi) fetal toxicity due to environmental hazards, such as exposure to certain toxic substances during pregnancy; and (vii) infantile metopism or congenital disfigurement of the forehead as a result of unclosed frontal suture. These autistic traits, in today's terms, are: low intelligence, impaired communication and socialization, monologuing which may include logorrhea, global developmental delay, hypotonia, poor health due to epigenetic changes caused by environmental risk factors, and persistent metopic suture.

Slightly more than a century later, Chia and Yang (2021) reported that in the year 1747, the earliest well-documented case and maybe even being the first court case of autism, involved the brother of a man by the name Hugh Blair had petitioned to annul Blair's marriage to his wife in order for him to get his inheritance. According Houston and Frith (2000), Blair was noted to show very strong autistic symptoms.

Another interesting case was reported in 1798. It concerns a feral child, popularly known as the Wild Boy of Aveyron, who had been found living in the woods near Saint-Sernin-sur-Rance for seven years. When he was discovered, numerous scars were found in his body. By the time the wild boy was brought back into society, he was estimated to be about 12 years of age. His case soon was taken up by a young French physician, Dr Jean Marc Gaspard Itard (b.1774-d.1838), who worked with the boy for five years and gave him his name, *Victor* (see Itard, 1802, for detail). Victor was not deaf and dumb. Neither was he mute, but he lacked speech and communication skills. Itard was interested in determining what Victor could learn. He devised different strategies to teach the boy words and

recorded his progress. Based on his work with Victor, Itard eventually published a memoir/report in 1801/1806 and broke new ground in the education of the developmentally delayed (also see Itard, 1962). He continued to make several attempts to teach the boy language and social skills but without much success. The only things that Victor was able to do was to utter "*lait*" (milk) and "*Oh Dieu*" (Oh God) with rudimentary progress. His lack of exposure to human contact during the critical or sensitive period, which is essential for the normal development of language and social skills, had made it challenging and quite impossible for the boy to develop these functions later in life. Moreover, Victor also preferred to eat raw meat than cooked meal. He would also frolic naked in the snow during winter, indicating the boy had been in the wild most of his life.

The recent publication of a book – *In a Different Key: The Story of Autism* (Donvan & Zuker, 2016) – has rekindled interest in the historical precedents of autism in feral children. In fact, the idea of this book has been borne out of an earlier publication *Autism: Explaining the Enigma* (Frith, 2003) where the author, a German developmental psychologist, narrated the case of Victor of Aveyron.

Many other feral children like Victor share some common symptoms that are indicative of autism (see also McNeil, Polloway, & Smith, 1984, for detail): failure to socialize with others, lacking in attention, rocking themselves back and forth, repetitive movements and ritualistic behaviors, normally observed in caged or otherwise confined animals, and are often suggestive of stress, anxiety and depression, whose initial letters constitute what is known as SAD syndrome. In fact, Bettelheim (1959) argued that the behavior of feral children strongly resembled that of severe infantile autism with seemingly animal-like traits and habits.

However, Casanova (2016) warned readers that "[T]he great majority of reported cases of feral children are hoaxes. True accounts of feral children … describe individuals who have trouble walking upright, prefer to eat raw food, and have great difficulties in acquiring language" (para. 3). He further added that "[I]t is difficult to decipher the nature for their difficulties in socializing and acquiring language. Without a previous history, some authors have speculated that the children could have been abandoned due to pre-existing mental and/or physical impairments or could have suffered severe abuse of trauma" (Casanova, 2016, para. 3).

3. Phase 2 – Coexistent Symptoms

According to Shorter (2015), the beginning of the 19th century witnessed the great European nosological (Note 5) tradition, which was a symptom-based classification system, with the French dominating the field of nosology in the first half of the 19th century. In order to classify different abnormal conditions (and anomalies), certain coexistent symptoms must be present in order to identify a condition first, next to differentiate it from other conditions, and lastly to classify it with a name given to the condition. In the United States, the first official American nosology was issued in 1917 by the American Medico-Psychological Association (Kendler, 2016). With the nosological system in place, it became easier to identify, differentiate and classify diverse conditions for the purpose of accurate diagnosis in order that appropriate treatment could be provided.

In 1911, Dr Eugen Blueler (b.1857-d.1939), a Swiss psychiatrist, first coined the term *autism* observed in people with dementia and also in schizophrenia. Almost 30 years later, in the United States, Dr Leo Kanner (b.1894-d.1981), an Austrian-American psychiatrist, reported his young patients manifesting symptoms of infantile autism. A year later in 1944, in Germany, Dr Hans Asperger (b.1906-d.1980), an Austrian pediatrician, reported his high-functioning patients who exhibited autistic traits. The latter condition has been named as Asperger's Syndrome.

Autism was originally described as a form of childhood schizophrenia, whose cause was believed to be the result of cold parenting. Dr Eleanor Mildred Creak (b.1898-d.1993), a British psychiatrist, known for her work on autism and organic (physiologic) mental disorders, developed nine main characteristics to describe childhood schizophrenia (see Creak, 1961, for detail). In fact, many of her characteristics overlapped with Kanner's criteria for infantile autism. Creak (1961) believed that these nine behavioral traits constituted childhood schizophrenia. However, she failed to further operationalize his criteria or indicate how these behavior patterns were specific to childhood schizophrenia (Matson & Minshawi, 2006). It was later regarded "as a set of related developmental disorders, and finally, as a spectrum

condition with wide-ranging degrees of impairment. Along with these shifting views, its diagnostic criteria have changed as well" (Zeldovich, 2018, para. 2). Nevertheless, Creak's criteria of childhood schizophrenia have given a new direction for many descriptions of autism and commonly used autism assessment tools today (Sultana, 2020).

In 1968, childhood schizophrenia was listed in the Diagnostic and Statistical Manual of Mental Disorders-Second Edition (DSM-II; American Psychiatric Association, 1968). However, infantile autism was not included in the diagnostic manual. It waited for another 12 years before infantile autism was officially recognized and listed in DSM-III (American Psychiatric Association, 1980).

In 1972, Dr Michael Llewellyn Rutter (b.1933-d.), a child psychiatric researcher at Maudsley Hospital, London, in the United Kingdom, published a paper arguing for the childhood schizophrenia to be reconsidered. He concluded that "childhood schizophrenia includes at least three main disorders differing in age of onset: Autism, schizophrenia, and disintegrative psychosis, which constitute disparate conditions requiring separate classification" (cited in Stevenson, 2020, p. 14). He also added that "the evidence suggests that autism develops on the basis of a central disorder of cognition which involves the impairment of both the comprehension of language and defects in the utilization of language or conceptual skills in thinking" (cited in Stevenson, 2020, p. 14). In addition, Rutter was cited in Sultana (2020) to claim that "the autistic child has a deficiency of fantasy rather than an excess" (para. 6). The term *fantasy* refers to imaginative fantasy, which is one of the eight types of imagination (Hunter, 2013). The meaning of the word *autism* was re-formulated from a description of someone who fantasized excessively (hyperphantasia) to one who did not fantasize at all (aphantasia). This concerns some kind of impairment that has afflicted the imagination, which refers to "the ability to form mental images, phonological passages, analogies, or narratives of something that is not perceived through our senses" (Hunter, 2013, p. 113).

4. Phase 3 – Syndromic Symptoms

When exactly the third phase began, we do not know. We put it around the 1970s and 1980s when more research studies were carried out to identify the signs and symptoms that are correlated with each other in differentiating the condition of autism from other anomalies. Once the syndromic symptoms have been identified and confirmed, the next step is to design appropriate intervention strategies that can help to treat the condition of autism.

The year 1970 witnessed the founding of the Society of Neuroscience based in Washington, DC, USA, and was also considered as the year of the birth of neuroscience. Its first significant contribution began in 1973, when Dr Candace Pert (b.1946-d.2013), an internationally renowned American neuroscientist and pharmacologist, discovered opiate receptors in the brain. This discovery led to several other deeper studies on the opiate receptors and almost one and a half decade later, Sahley and Panksepp (1987) found that brain opioids modulate social-emotional processes, and the possibility that blockade of opioid activity in the brain causes children with autism to lack the desire to socialize with others and result in an inappropriate use of language for communication. With the first Positron Emission Tomography (PET) scanner invented in 1974, visual information about brain activity could now be better observed than before and a mouse became the subject of the first nuclear magnetic resonance (NMR) scan. With advancement made in neuroscience, especially in neuroimaging studies, we have a better understanding of how the autistic brain develops and functions (see Chia, Lim, & Lee, 2017, for detail) and how new discoveries will help to shape the definition of autism.

Another domain of science is genetics that also plays a key role in our understanding of autism. Researchers have been quite aware that genes contribute to autism since the 1970s, and in 1977, the first autism twin study was carried out and discovered that identical twins often share the condition. Folstein and Rutter (1977) conducted a systematic study consisting of a representative group of 21 same-sexed twin pairs, involving 11 monozygotic or identical (MZ) and 10 dizygotic, fraternal or non-identical (DZ), in which at least one twin showed the syndrome of infantile autism (Folstein & Rutter, 1977). The study found that autism had a high chance of occurring in one twin if their MZ twin had autism. However, in the DZ twins, there were no occurrences of autism in both siblings. In other words, Folstein and Rutter (1977) showed the important role that genetics plays in the development of autism and hence, set to challenge the controversial view that autism was the result of bad or poor

parenting. Today, genetic studies have found that autism is 80 percent reliant on inherited genes while environmental causes are responsible for just 20 percent of the risk (Sandin *et al.*, 2017). While no specific gene has been identified to cause autism, Locwin and Entine (2016) have provided some rough estimates that over 100 to 200 or more genes are involved or implicated in autism.

In 1980, the Diagnostic and Statistical Manual of Mental Disorders-Third Edition (DSM-III; American Psychiatric Association, 1980) recognized autism as a developmental disorder separated from schizophrenia, and entered it as a separate entity. Its previous associations with the presence of hallucinations were removed. There were four subcategories of autism listed in the diagnostic manual: infantile autism, residual autism, childhood-onset pervasive developmental disorder, and an atypical form (cited in The Recovery Village, 2020).

Seven years later, a revised edition of DSM-III was published and introduced as DSM-III-R (American Psychiatric Association, 1987) with a broader concept of autism to include the milder symptoms of the condition. Beginning as a system for collecting census and psychiatric hospital statistics as well as from a United States Army manual, DSM from its first edition published in 1952 until today has evolved through the decades. Several revisions (as in DSM-III-R) or text revisions (as in DSM-IV-TR) were made and the total number of mental disorders were incrementally added with its newer editions. At the same time, those conditions that were no longer regarded as mental disorders were also removed from the diagnostic manual.

It was also during that same year, Dr Ole Ivar Løvaas (b.1927-d.2010), a Norwegian-American clinical psychologist and professor at the University of California, Los Angeles, published his study to show that children with autism could improve after intensive behavioral modification or therapy (see Løvaas, 1987, for detail). Dr Ivar Løvaas pioneered what is now known as Applied Behavioral Analysis (popularly known by its abbreviation, ABA) based on his development of discrete trial training and early intensive behavioral intervention for children with autism.

In 1988, with the worldwide screening of the movie "Rain Man" that revolved around the story of an adult with autism, Raymond Babbitt (portrayed by the Oscar Award winning actor Dustin Hoffman), who was also an autistic savant. As the result of that movie, there was an increase public awareness about the condition of autism. However, there was also a downside of it: the general public began to stereotype the abilities of individuals with autism.

Seven years after the revised edition of DSM-III (or DSM-III-R) was issued, the newer DSM-IV (American Psychiatric Association, 1994) was released with Asperger's syndrome added as a separate subcategory of autism. The other subcategories of autism include Pervasive Development Disorders (PDD), Autistic Disorder, Rett Disorder, Childhood Disintegrative Disorder (also known as Heller's Syndrome) and PDD-Not Otherwise Specified (PDD-NOS). In 2004, a text revision of DSM-IV (or DSM-IV-TR) was issued by the American Psychiatric Association.

5. Phase 4 – Complex Syndromic Symptoms

The decade of 1990s and beginning of the new millennium, as the world entered into the 21st century, constitute the new beginning when autism literature is reporting more complex syndromes, such as Angelman Syndrome discovered by Dr Harry Angelman in 1965; Pitt-Hopkins Syndrome discovered by Drs D. Pitt and I. Hopkins in 1978; Timothy Syndrome discovered by Dr Katherine W. Timothy in 1989. These syndromes have been found to share common genetic bases with some forms of autism. They are also considered to be syndromic forms of autism (Bader et al., 2011; Peters et al., 2012; Phan et al., 2020) or just called syndromic autism (Melillo, 2013). It was also during this time, a lot of other studies were carried out to consolidate of the different subtypes of autism, e.g., Heller Syndrome (Note 6) (discovered by Dr Theodore Heller in 1908), Kanner Syndrome (discovered by Dr Leo Kanner in 1943), Asperger Syndrome (discovered by Dr Hans Asperger in 1944), and Rett Syndrome (discovered by Dr. Andreas Rett in 1966), before the release of DSM-5 in 2013 when autism spectrum disorder becomes the umbrella term to include all its subtypes with the exceptions that Asperger Syndrome and Pervasive Developmental Disorders-Not Otherwise Specified (also known as atypical autism) were removed from the diagnostic manual.

In 1998, Dr Andrew Wakefield and his colleagues (Wakefield et al., 1998) published their study

involving 12 subjects without any control group in *The Lancet*, suggesting the possibility of a link between the MMR vaccine, a form of bowel disease and a type of autism. As a result of its publication, the paper received widespread media attention. In Sweden, Gillberg and Heijbel (1998) carried out their study comparing the number of autism cases in children from two towns before 1982 when local doctors first began giving the MMR vaccination. Their findings showed no difference between the two groups of children in either town. The study showed no association between the MMR vaccine and autism. However, in the meantime, there was a drastic decline in MMR vaccination rates leading to a number of outbreaks of measles around the world. A study done by Taylor et al. (1999) in the following year also failed to find any link between MMR vaccination and autism. More studies (e.g., DeStefano, 2007; Taylor, Swerdfeger, & Eslick, 2014) were subsequently carried out and their findings agreed with the earlier findings of Gillberg and Heijbel (1998) and Taylor et al. (1999). Several more extensive investigations were carried out throughout the world and found the claim of the MMR vaccine-autism link to be unsubstantiated or false (Di Pietrantonj et al., 2020). The Wakefield et al. (1998) paper was eventually retracted from *The Lancet* (see Editors of *The Lancet*, 2010, for detail) based on the findings of the British General Medical Council, which had conducted an inquiry into allegations of professional misconduct against Wakefield and two former colleagues (BBC News, 2006). In 2011, Dr Wakefield issued a press release stating that "I want to make one thing crystal clear for the record – my research and the serious medical problems found in those children were not a hoax and there was no fraud whatsoever. Nor did I seek to profit from our findings" (PRNews, 2011, para. 3). He went on to defend himself stating: "I stand by the Lancet paper's methodology and the results which call for more research into whether environmental triggers cause gastrointestinal disease and developmental regression in children. In fact, despite media reports to the contrary, the results of my research have been duplicated in five other countries" (PRNews, 2011, para. 4). Several papers (e.g., Balzola *et al*., 2005; Galiatsatos, Gologan, & Lamoureux, 2009; Jarocka-Cyrta, Wasilewska, & Kaczmarski, 2011) have been cited to support the findings of Wakefield et al. (1998), but, sadly, they have been largely ignored.

The 1990s also witnessed the MMR-autism link controversy from the paper of Wakefield et al. (1998). This has led to more challenges in autism research, such as thimerosal-autism link (Bernard et al., 2001; Kidd, 2002), Vitamin D deficiency-autism link (Cannell, 2017; Jia et al., 2015), and the leaky gut hypothesis (Heberling, Dhurjati, & Sasser, 2013; Liu, Li, & Neu, 2005) and the surge of controversial autism treatments, such as the Facilitated Communication (Biklen, 1993; Mostert, 2010), the Son-Rise® Program (Kurtz, 2008; Simpson, 2005), GFCF diet (Elder et al., 2015; Reissmann et al., 2014), and using chloride dioxide to treat or cure autism (Martin, 2015; Newnham, 2015). Many autism experts and professionals, such as Simpson (2005), Worley, Fodstad and Neal (2014) and Chia (2015), have warned of the risk involved in autism treatments that have been peddled for quick improvement and/or miraculous cure when they are not scientifically proven to be effective.

Moving forward to the year 2001, when thimerosal (Note 7) – a mercury-based preservative used in vaccines – was discontinued in childhood vaccines, such as MMR, due to the fear and speculation concerning the link between thimerosal and autism. There were also reports on the use of thimerosal in dental amalgam. However, Schultz (2010) reported that "[T]he risks for autism from other mercury exposures such as from dental amalgam restorations or environmental mercury release into the atmosphere is ambiguous" (p. 187). Results from the current research remain inconclusive but it does raise the public awareness about what goes into the ingredients of a vaccine or dental amalgam should be taken seriously even by a lay person when they are seeking medical or dental treatment. There are several studies (e.g., Bernard et al., 2001; Blaxill et al., 2001; Kidd, 2002) with findings that support the thimerosal-autism link hypothesis, while others (e.g., Gillberg & Heijbel, 1998; Kaye, del Mar Melero-Montes, & Jick, 2001; Hviid et al., 2003) have failed to find the link. However, according to the website of Centers for Disease Control and Prevention (CDC, 2020), it states that "[I]n July 1999, the Public Health Service agencies, the American Academy of Pediatrics, and vaccine manufacturers agreed that thimerosal should be reduced or eliminated in vaccines as a precautionary measure" (para. 1).

In addition to DSM and ICD as the diagnostic classification systems, there are also other lesser known nosological classification systems such as the *Educator's Diagnostic Manual* (EDM) of disabilities and disorders (Pierangelo & Giuliani, 2007) for special education professionals and educational therapists;

the *Psychodynamic Diagnostic Manual-Second Edition* (PDM-2) (Lingiardi & McWilliams, 2017) for mental health practitioners including counselors; and the *Diagnostic Manual-Intellectual Disability-Second Edition* (DM-ID-2) (Fletcher, Barnhill, & Cooper, 2016).

Among them, EDM provides a set of comprehensive diagnostic criteria for autism. Published by the American Academy of Special Educational Professionals in 2007, EDM is an invaluable source of reference meant for educators, and more so for those working with individuals with autism in the field of inclusive, remedial and special education. The diagnostic manual uses the specific IDEA 2004 classification to define autism: "a developmental disability significantly affecting verbal and nonverbal communication and social interaction, usually evident before age 3 that adversely affects a child's educational performance. Other characteristics often associated with autism are engagement in repetitive activities and stereotyped movements, resistance to environmental change or change in daily routines, and unusual responses to sensory experiences. The term does not apply if a child's educational performance is adversely affected because the child has an emotional disturbance" [34 C.F.R. 3000.7(c)(1)].

The EDM lists autism under its diagnostic code AU at Level I, and it is further categorized under eight specific disorders at Level II: AU1.00 Asperger's Syndrome, AU2.00 Autistic Disorder, AU3.00 Childhood Disintegrative Disorder, AU4.00 High-Functioning Autism, AU5.00 Hyperlexia, AU6.00 Multiplex Developmental Disorder, AU7.00 Rett Syndrome, and AU8.00 Other Types of Autism-Be-Specific. Specific types of disorders are categorized under Level III while specific subtypes of disorders come under Level IV. Finally, Level V concerns the degree to which the disability concerned adversely affects an individual's educational performance. This 5-level diagnostic coding system helps special educators and educational therapists to understand how autism affects a child's educational performance and how an individualized education plan (IEP) team "can decide the extent to which the child needs support in terms of modifications, related services, assistive technology, classroom accommodations and a restrictive educational program outside the regular classroom" (Chia, 2015, p. 15).

In 2013, the American Psychiatric Association released the fifth edition of DSM that addresses inconsistencies in the diagnostic criteria to include all the subcategories of autism into a single diagnostic entity called autism spectrum disorder (ASD). In DSM-5, autism is described as having two key impairment areas: (i) social communication; and (ii) restricted, repetitive patterns of behaviour, interests and activities. Differences in sensory perception have also been included in the up-dated DSM diagnostic criteria for autism. In addition, a rating scale on three levels of support for individuals with autism has been included, but it is not an assessment indicator for long-term support requirements. As autism affects people in different ways where no two persons with autism are alike in every respect, with their different needs and requiring different intensity of support, the 3-level support offers some form of guidelines suggesting the extent of required support to be included in an individualized treatment plan. Briefly, Level 1 indicates an individual with autism requires some kind of support. Level 2 concerns substantial support is needed. Level 3 means very substantial support is necessary for the individual with autism.

6. Conclusion

Despite much research has already been done on autism, its primary cause remains largely unknown for the vast majority of reported cases. Though previous studies indicate that the etiology of autism involves both genetic (e.g., Bailey et al., 1995; Geschwind, 2008; Steffenburg et al., 1989) and environmental (e.g., Grabrucker, 2013; Karimi et al., 2017; Volk et al., 2014) factors, it is still the topic of an ongoing debate among the autism experts and researchers (e.g., Colvert et al., 2015; Gaugler et al., 2014; Hallmayer et al., 2011). We are expecting more new findings to come from epidemiological studies (e.g., Atladóttir et al., 2010; Lee et al., 2015; Zerbo et al., 2013) to provide us insights into how and to what degree that environmental factors can cause maternal infections during pregnancy resulting in autism.

It is difficult for us to draw a conclusion on what actually causes autism in order to provide a clear definition of the condition. Autism Speaks (2021) in its website has summed it nicely by stating that "[W]e know that there's no one cause of autism. Research suggests that autism develops from a combination of genetic and nongenetic, or environmental, influences" (para. 3).

In our conclusion to this paper, we have proposed our definition of autism as follows: It is a multi-faceted spectrum disorder of either primary (essential/idiopathic) or secondary (syndromic/complex) type, within a continuum of personality ranging from introversion through ambiversion to extroversion, that is a neuro-developmental syndrome (with varied subtypes) of constitutional origin (genetic) and whose causes could also be epigenetic and/or non-genetic, and its onset is usually around 36 months of birth, with empathizing, mentalizing and/or contextualizing deficits that cause impairments in (i) communication (verbal and/or non-verbal) and social interaction (sedentary disposition) (i.e., dissociating), (ii) sensory processing and modulation, and (iii) imagination or presence of stereotyped behaviors (ruminating), but may, on the other hand, also subconsciously mimicking/mirroring behaviors, to display (by autistic savants) or hide (by autistic crypto-savants) a strong systemizing drive with good attention to detail, deep narrow interests, and islets of ability (Xie & Yang, 2020). This inclusionary conceptual definition of autism is updated and adapted from the earlier definitions provided by Chia (2012) and Xie (2019).

From this proposed definition, we have identified six key processes that individuals with autism need to work on. They are briefly described below with suggested treatments:

(1) Contextualizing (also known as context processing): ability to respond appropriately to a stimulus within its context. Suggested autism treatments: social scripts and routines (Aldred, Green, & Adams, 2004; Loveland & Tunali, 1991).

(2) Dissociating: ability to separate normally related mental/cognitive processes, resulting in one group of mental/cognitive processes functioning independently from the others or the rest. Suggested autism treatments: mindfulness-based techniques (Hartley, Dorstyn, & Due, 2019).

(3) Empathizing: ability to identify another individual's emotions and thoughts, and responding to these with an appropriate emotion. Suggested autism treatments: affective computing (El Kaliouby, Picard, & Baron-Cohen, 2006) and music therapy (Greenberg, Rentfrow, & Baron-Cohen, 2015).

(4) Mentalizing: ability to attribute mental states to others or another individual. Suggested autism treatments: computer-assisted face recognition (Rice *et al.*, 2015) and language-cognition interface technology with iPad application (Durrleman, 2020).

(5) Mirroring (also known as mimicking): ability to observe an action and then performing an equivalent action. Suggested autism treatments: parent-implemented imitation (Ingersoll & Gergans, 2007) and video-modeling approach (Corbett & Abdullah, 2005; Delano, 2007).

(6) Ruminating: ability to think repeatedly. Suggested autism treatments: mindfulness-based therapy (Spek, Van Ham, & Nyklíček, 2013) and controlled eating and differential reinforcement (McKeegan, Estill, & Campbell, 1987).

(7) Systemizing: ability to analyze the variables in a system by deriving underlying rules that govern the behavior of the system in order to construct, reconstruct or deconstruct it. Suggested autism treatments: interactive multimedia (Golan & Baron-Cohen, 2006).

We do not advocate any particular autism treatment as provided in the examples above. Readers are strongly advised to review each of the autism treatments objectively and ensure that whichever ones they choose to use must be appropriate to meet the needs of individuals with autism.

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Notes

Note 1. According to Healthline.com, *neurotypical* is a newer term describing people of typical developmental, intellectual, and cognitive abilities. "In other words, it is *not* used to describe individuals who have autism or another developmental difference" (Brusie, 2017, para. 1).

Note 2. The unconscious mind is "a concept in which processes of the brain happen automatically or without thought" (Theodore, 2020, para. 9).

Note 3. Yosef (Joseph) has an Egyptian name, Zaphnath-Paaneah, given by the Pharaoh during the period of the Hyksos dynasty (i.e., the 15th dynasty of Egypt, reigning from c. 1650-1550 BC).

Note 4. Deep or affective empathy refers to "the ability not just to imagine, but also to actually feel what other people are experiencing" (Chia & Camulli, 2020, p. 5).

Note 5. Nosology (noun) and nosological (adjective): It refers to a specific branch of medical science that deals with the classification of diseases (later, including disabilities and disorders).

Note 6. Heller Syndrome is also known as Childhood Disintegrative Disorder.

Note 7. Thimerosal contains ethylmercury. This form of mercury is cleared from the human body more quickly than methylmercury. Hence, it is therefore less likely to cause any harm.