THE EMERGENCE OF NEURODIVERSITY

by

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Note to the reader:

Currently, there are many terms for Autism Spectrum Disorder (ASD). Some of the terms for ASD are neurodivergent, Asperger's, and Autism. A diagnosed individual may identify with one term over another; however, to remain neutral in this paper, I will refer to the terms used for ASD listed in the textbooks I researched.

Abstract:

Research compiled from thirty years ago is different from the research we compile in today's time, no matter the subject. My first question is how much has research about Autism Spectrum Disorder changed in the last 30 years? My second research question is how do people's lived experiences compare with information in textbooks? ASD is classified in the DSM-5 as "deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions." The addition of ASD to the DSM-5 in May 2013 is fairly recent considering it was first discovered in 1943. The purpose of my research project was to compare how far Autism research has come between various published textbooks over the span of four decades and how that compared to the experiences of autistic young adults, parents of autistic children, and therapists who work with autistic people. In textbooks, autism is discussed on three broad categories: causes, diagnosis, and support. I am using the previous three terms based on information provided by members of the autism community. Previously collected de-identified interviews were evaluated for comments about cause, diagnosis, and support. The comments were compared to information available in the textbooks. The results of this yielded high variability across research textbooks. Many texts contradicted each other and my conclusion from my thesis project is that the variability in research contributes to the evidence based practice gap. This gap is seen from researchers not using their findings in a clinical setting, which leads to different texts saying different things about

the same subject. Future studies need to incorporate more than written information, as suggested by people from the autism community.

Chapter 1: Introduction

ASD was first discovered in 1943; however, how ASD was discussed in textbooks has changed over the last 40 years. Textbooks are sources of information for students that will work as future therapists; therefore, it is essential to evaluate how ASD is discussed for future professionals serving people with ASD. Research compiled forty years ago will be different from current research, no matter the subject. I completed a review of how research-based textbooks discussed ASD from 1990 to 2020. The term Autism Spectrum Disorder(ASD) was first added to the *Diagnostic and Statistical Manual of Mental Disorders-5* (DSM-5) in May 2013, relatively recent considering ASD was first discovered in 1943. Autism Spectrum Disorder is classified in the DSM-5 as:

> Deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions (DSM V, 50).

My motivation to research this thesis topic was to record and report the changes in ASD over forty years, and my determination to discover the facts kept me searching. I created a standard set of questions that I used when reading each textbook. The unanswered questions proved to be more beneficial to me than those answered. In earlier textbooks, there were no explanations for causes at all; however, the unanswered questions in one text would be answered decades later in another as research advanced.

A review of these textbooks revealed changes in how ASD has been discovered and discussed. There have been new developments to suggest that ASD carries heavy genetic importance. This genetic influence overpowers the idea that ASD is thought to be caused by a mother's diet and parenting styles. Although the genetic input comes from the mother's side, being a *refrigerator mother* is not the cause of this diagnosis. The term

refrigerator mother was used in the 1940s by Austrian psychiatrist Leo Kanner; his reasoning was that their "cold" demeanor was why their child would retreat into an autistic state (Jack, 36). The most current textbooks have revealed that there is no definitive cure for ASD as opposed to the diet changes and antipsychotic medication referenced as cures in the earlier books (Jack, 34).

There were three main categories of questions that I looked for: *causes, assessment,* and *support*. These categories contained subcategories of specific questions that I will touch on later. In these main categories, some content stayed the same from textbook to textbook, while others differed. The texts that had significant variability were causes and support. There were many discrepancies in what or who caused an ASD diagnosis. Many textbooks blamed *refrigerator mothers* suffering from postpartum for their child's disability. Evidence debunking Psychiatrist Leo Kanner's theory did not surface until the 1980s. Even then, it was still thought to be the common cause of ASD. High variability was also seen in the support approaches. The earlier textbooks proposed that there were options to support individuals diagnosed with ASD. It was not until the late 90s and early 2000s that proposed remedies such as diet changes and antipsychotics were given to diagnosed individuals. The most current textbooks contradict the earlier textbooks and urge the significance of individualized therapy for an incurable disorder.

As time progressed, changes in the research were not the only thing that showed drastic improvements. The most recent textbooks even changed the name for which it is appropriate to reference autism. At first, diagnosed individuals were called by their diagnosis name. It wasn't until decades later that the name changed to individuals who have a diagnosis of autism. This change was to signify that the person was more than

their diagnosis. An individual is still a person, despite their diagnosis. To go even further, there is now another name change being enforced. This diagnosis is now being referred to as *neurodiversity*. As research is changing, social norms are also changing. The discrepancies seen in what the official name for a diagnosis of autism comes from is from what side of the spectrum the individual is on.

Stating that an individual is *on the autism spectrum* is a broad statement to make. There are many levels of severity that goes with being "on the spectrum". One side of the spectrum includes low cognitive functioning, and the other includes genius intelligence. Lower cognitive functioning is associated with being non-verbal and mute (DSM V, 64). The other side of the spectrum features higher functioning cognition and allows for highly expressive speech. For example, Elon Musk, the founder of Tesla, is on the higher side of the spectrum. Those are the two opposite sides of the spectrum, but what about the individuals who are in the middle?

Since what we know about ASD is still changing, what we know about the spectrum's complexity is also changing. The 1994 DSM-IV categorizes the spectrum into four stages:

> Autistic Disorder, Asperger Syndrome, Pervasive Developmental Disorder-not otherwise specified (PDD-NOS), and Childhood Disintegrative Disorder.

These categories were added to the new DSM-V in 2013, the current *Diagnostic and Statistical Manual of Mental Disorders*, which does not label ASD as neurodiversity; however, a progressive movement is presented by individuals on the spectrum to be referred to as neurodivergent (DSM IV, 299). The spectrum has always been present, though not documented until 2013. ASD is an extremely complex disorder that contains

many unknown factors. Fortunately, it appears that as more individuals are diagnosed with ASD, funding and research are increasing.

Chapter 2: Methods

In my comparison of textbooks, the earliest reference to ASD in textbooks occurred in the 1990s. In the 1990s, the prevalence of autism was four diagnoses for every 10,000 children (Cohen, 113). The current prevalence of autism is 1 in every 44 children (CDC). What changed? Has this disorder always been prevalent, but there was no research to back a diagnosis, or are rates of increasing as time goes on? The textbook comparisons over the past four decades depict a timeline related to the rate increase.

The topics that I researched as I read each textbook were divided into three categories: causes, diagnoses, and support. I will break down each category and compare how each textbook addresses these main topics. The advances in research will add to a comparative timeline that shows how neurodiversity has become common in society.

Chapter 3: Research

Causes: What are the proposed causes across various textbooks?

In the first decade, I looked at textbooks from **1990-1999**. Plomin & McClearn (1993) *Nature, Nurture, & Psychology* suggested that the causes of ASD may have contributed to psychiatric disorders. It is known that ASD has a vital genetic factor, but what is unknown at this period is what the exact genetic factor may be. The heritability is estimated to be between 91-93%, and the rate for familial diagnosis increases in siblings by a rate of 50-100. This text implies that the genetic component lies on the X chromosome, and this finding is known as *Fragile X.* This still does not tell us which genetic chromosome is responsible for ASD, but it is a step in the right direction. This text talks about the parental influence being primarily passed down from the mother due to the X chromosomal link associated with fragile X (Plomin, 272).

Another text from the first decade, Happe (1994) *An Introduction to Psychological Theory,* contributed causes of ASD to have a traumatic brain injury. This was a proposed cause since there were links to damage in the cerebellum and limbic system that reflected the emotional deficits. This book noted that another cause of ASD is from maternal parents with complicated pregnancies or a history of schizophrenia. The theory of having a genetic link to schizophrenia was seen in the similarities seen in the behavioral outbursts of the two disorders. These two disorders have overlapping symptoms: social withdrawal, behavioral issues, and communication difficulties (Happe, 92).

The third text from this same period is: Cohen (1998) *Targeting Autism: What We Know, Don't Know, and Can Do to Help Young Children with Autism and Related Disorders.* This text proposed that the causes of ASD are brought out by the *Fragile X* chromosome. This text is consistent with the text from 1993. Unlike the other texts, this text references previous researchers saying that the truth about ASD is that there are no known reasons why ASD presents in some individuals and not in others. Although this entire textbook had a multimode of research, there is no concrete cause of autism (Cohen, 62).

The final text reviewed from the 1990s is: Mahwah & Erlbaum (1999) *Identification, Education, and Treatment.* This text does not attribute a name to the genetic component of ASD; however, it does note that the genes involved affect the brain's structure. It was noted that researchers in this text compared electroencephalograms (a test that measures electrical activities in the brain using electrodes) of individuals with and without an ASD diagnosis. These comparisons showed that there is a different brain structure in the limbic and cerebellum parts of the brain, as well as differences in neurotransmitters that regulate serotonin. When serotonin is not regulated, this can cause disruption in mood, sleep cycle, wound healing, blood clotting, and digestion. This was the first text that I read that does not agree with previous research that ASD is linked to schizophrenia. This text points out that although these disorders are similar, they differ upon further research (Mahwah & Erlbaum, 71).

The second decade I researched was **2000-2009**. The first text reviewed from this period was: Gabriels & Hills (2002) *Research to Individualized Practice.* As I moved on to the next decade of research texts, I noticed that *Fragile X* was still the only name given

to the reasoning for ASD. This text contributed *Fragile X* to a diagnosis that is specific to ASD but different from Asperger's. This text recognized these as two different disorders not under the same umbrella as autism. At this point, there was no spectrum that listed all the other disorders that were a part of ASD (as the spectrum only recently included other disorders under ASD in the 2013 DSM-IV). This text did not contribute to this gene being passed down by any specific parent; however, Damasio & Maurer (1978) *Executive functioning* draw attention to other theories regarding the causes of ASD other than a genetic factor. The referenced text talked about the genetic link to autism being triggered by a vitamin deficiency that caused the immune system to go into overdrive and essentially awaken genes, causing mutations. I would like to note that this second reference had no data or research on this. This was a proposed theory that had absolutely no backing to it (Gabriels & Hills, 56).

The second book reviewed from this decade was: Department of Health (2005) *What we know*. The text starts by saying that scientists don't know what causes autism now, but there is strong evidence that genetics play a significant role. Since there does not seem to be one specific gene that is the cause of ASD, the researchers in this text suggest that at least 10 genes on various chromosomes may be involved. The idea behind this disorder having a multimodal genetic factor comes from researchers thinking that when specific genes are affected, they will trigger specific reactions. This is only a theory, and it was based on the idea that some genes are responsible for brown hair and others for blonde. But just because we have genes causing different hair colors, does that mean different genes can lead to how severe autism may or may not be? This text also gives ideas to different theories that may trigger the genetic factor. In one sentence, this

text relates autism being linked to chromosomal changes from the effects of vaccines, but in another sentence, the text states that there is no conclusive scientific evidence to support this claim. The variability across decades matches the variability seen in this one textbook (U.S. Department of Health, 3).

Moving toward the end of this second decade: Cohen (2007) *Targeting Autism: What We Know, Don't Know, and Can Do to Help Young Children with Autism Spectrum Disorders.* I would like to note that this book is the updated version of the book I referenced in the first decade. It is written by the same author. This updated text links autism to having a solid genetic foundation, as it is more prevalent in some families than others. This is the first text I read that gives the prevalence rating of autism around 6% of the population. Within the family, the texts record a 90% chance of identical twins both having autism and no difference in prevalence rating for non-identical twins or siblings. These texts refer to autism as a different entity from Asperger's syndrome. Asperger's is more common in families that have high-functioning individuals with autism. This text talks about one family in which the parents had high-functioning autism, known as Asperger's, who had two children. One of their children was diagnosed with autism, and the other, higher-functioning child was diagnosed with Asperger's. In this text, a survey was sent out to individuals with a diagnosis in their families, asking if they noticed more people in their extended family having traits of autism. There was high reporting that many of the children in this family (cousins, nieces, nephews, etc.) have mild forms of autism. This evidence is what strengthened the hypothesis of autism having a genetic basis. The updated version of this text was still congruent with the 1994 version saying that autism has a genetic component. But both texts were still unsure about the exact

component. The theory still stands by the possibility of the Fragile X chromosome contributing to a diagnosis. But, even a decade later, the updated textbook does not have that updated research (Cohen, 12).

The final decade I researched was **2010-2020**. The first book reviewed: Oller & Oller (2010) *Autism: The Diagnosis, Treatment, & Etiology of the Undeniable Epidemic.* This text attributes the causes of autism to either genetics or a mother's diet during pregnancy. Researchers suggested that since there is possibly an immunologic deficiency and vitamin deficiency of B6, depending on the mother's diet while pregnant, what she ate or didn't eat could have contributed to the genetic differences seen in diagnosed individuals. This theory is just that: A Theory. This theory seemed impossible to prove, since collecting data on various mothers' diets showed high variability (Oller & Oller 141).

The following year the following text was published by Oxford University Press: (2011) *Autism Spectrum Disorders.* Oxford: Oxford University Press. This textbook suggests that there is a genetic factor, but the genetic factor is linked to prenatal abnormalities. The genetic mutations on the PTEN (phosphatase and tensin homolog) have been identified in individuals with autism and macrocephaly. Macrocephaly is the cause of neurological deficits and developmental delays due to abnormal head circumferences. This text also relates other disorders as having autistic-like characteristics, such as Cowden Syndrome, Bannayan-Riley-Ruvalcaba syndrome, and Proteus syndrome, which are characterized by germline PTEN mutations. All this data proves that there is evidence suggesting that autism contains a genetic factor; however,

this text does not entertain the possibility of comorbidity. This text does contribute to an autism diagnosis being passed down from mothers, but this text emphasizes that the increase is due to socioeconomic levels. It was reported that mothers with a postgraduate degree were twice as likely to have a child on the spectrum than mothers without a higher education level. This survey sent out did not account for the lack of financial means needed for an autism diagnosis test. So, the proposed causes should not be attributed to social or economic class (Oxford University Press, 48).

One of the last books I researched in this time period was: Simmons (2018) *The Official Autism 101 Manual.* This text identified that it is likely that there are over 100 genes that cause autism. It was thought that many genetic disorders, including Fragile X, Angelman syndrome, tuberous sclerosis, chromosome 15 duplication syndrome, and other single-gene and chromosomal disorders contributed to 15% of the population diagnosed. This text referred to these varying chromosomal disorders as fitting on the spectrum of ASD. This text also stated that there appears to be parental influence from both sides when the parents are at an advanced age. Environmental factors are seen pre-birth and birth, such as low birth weight, prematurity, and complicated pregnancies resulting in oxygen deprivation. There was also talk about vaccines being the cause that was debunked in this text. This text noted that this "myth" was causing more fear than good in parents with diagnosed individuals. It should be noted that this text found no distinct gene variations that contributed to which end of the spectrum an individual with autism was on (Simmons, 21).

The most current text I reviewed was: Das, Papaneophytou & El-Kour (2020) *Autism 360°F.* This most current text embodies all the previous texts' proposed causes; however, this text mentioned GBA (gut-brain axis) being a contributing factor to the etiology of autism. This is the theory that gut microbiota triggers a genetic mutation post-birth due to deficiencies seen in the diet or diagnosed individuals (Das, 302).

Summary of Causes: **1990-2020:**

While reading these various texts, it is difficult to draw a linear timeline of research advancements when there was no concrete reason for the etiology of ASD. All these texts have high variability, and as one proposed cause emerges in one text, it disappears in another, just to resurface in another text. There is not a single answer that can be given to the cause of autism. But the lack thereof should be noted when discussing research advancements. It should be concluded that research is not as advanced as it claims to be and that much more research needs to be done. Research about ASD should be more than just theory.

Diagnosis: **How is ASD diagnosed in individuals and across different genders?**

The text referenced above in the first decade, Plomin & McClearn (1993) *Nature, Nurture, & Psychology*, contributed to the diagnosis of ASD being largely male dominated. This text mentioned that females are the least likely gender to get diagnosed,

although this disorder is suggested to have a *Fragile X* chromosomal factor which is the chromosome differing males from females. This text does not talk about whether females are less diagnosed due to different characteristic traits brought out by gender (Plomin, 279).

Toward the end of the first decade the text, Cohen (1998) *Targeting Autism: What We Know, Don't Know, and Can Do to Help Young Children with Autism and Related Disorders* states that this is largely a male disorder. In the case that a female is diagnosed with ASD, they tend to have more severe symptoms and lower IQ than males that are diagnosed. There is no explanation for why one gender is diagnosed more than the other. The current prevalence in 1998 is around 4-5 per 10,000, and this text suggests that it should be higher, but much is still undiscovered about autism (Cohen, 11).

In the last text reviewed in the 1990-1999 decade, Mahwah & Erlbaum (1999) *Identification, Education, and Treatment*, the prevalence increased and was reported to be 21 in 10,000 individuals. Unlike other disorders that can be recognized through physical features or DNA testing, autism is more challenging to diagnose early in life. Testing is usually done in the first 18-24 months of life, and the most common type of diagnosis is done by watching social situations to find deficits (Mahwah & Erlbaum, 15).

In the 2000-2010 time period, Gabriels & Hills (2002) *Research to Individualized Practice* referenced wellness checkups becoming standardized for screenings of autism. These screenings started after parents became worried about their children not smiling or making eye contact. This screening tool was known as *Checklist for Autism in Toddlers* and was often paired with the *Autism Screening Questionnaire*. After these screenings

went out, the prevalence rate increased to 4 in every 2,000 individuals. As research advances, the prevalence rate is increasing rapidly (Gabriels & Hills, 28).

The second book from this decade, Department of Health (2005) *What we know*, stated that a diagnosis of ASD is 4-5 more likely to be found in males than females. The age of acquisition is around 18 months of age and is diagnosed by the lack of social and emotional skills. Another factor in determining a diagnosis of ASD is when children miss their developmental milestones and are categorized as late talkers (U.S. Department of Health, 5).

Later in the 2000-2010 decade, a text targeting autism was published: Cohen (2007) *Targeting Autism: What We Know, Don't Know, and Can Do to Help Young Children with Autism and Related Disorders*. Cohen updated her 1998 text and published under the same title. This new version of the text restates that ASD is largely a male disorder; however, Cohen does talk about other autistic-like disorders that are primarily present in females. Rett's disorder (or Rett syndrome) is one of the pervasive developmental disorders that contains several features of autism but is virtually exclusive to females. This syndrome has more severe impairments in cognition, hand movements, gross motor functioning, and language. The typical age for an autism diagnosis is around 3, so in the early 2,000s is when pediatric facilities start screening for it. The American Academy of Neurology, the Child Neurology Society, and the American Academy of Pediatrics are diagnostic tests used to determine if a child should be watched for autism. Autism may be hard to diagnose based on how variable this disorder may be, so this text

suggests that watching the child as they mature will lead to a more straightforward diagnosis later (Cohen, 10).

Oller & Oller (2010) *Autism: The Diagnosis, Treatment, & Etiology of the Undeniable Epidemic* had far less information about the diagnosis. This text only implies that diagnosis is difficult pre-birth and is primarily done around the ages of 3-5. This means that there is no way to genetically test a child in the womb to diagnose them with autism before they are born (Oller & Oller, 90).

In the last decade, 2010-2020, Oxford University Press: (2011) *Autism Spectrum Disorders* stated that the diagnosis is brought out by noticing social deficits. The deficits seen in a diagnosis "include failure to respond to one's name, poor eye contact, and an array of unusual reactions to sensory properties of objects." Diagnosis is complex, and even many professionals are not well-versed in diagnosis, which can lead to a prevalence skew (Oxford University Press, 46).

Jack (2014) *Autism and Gender: From Refrigerator Mothers to Computer Geeks* is one of the first textbooks to mention the possible reasons for such a gender difference in autism diagnosis. This text refers to autism only being understood when seen in terms of the male gender. An interesting conclusion as to why females are rarely diagnosed is that during the 1980s and 1990s, females were often excluded from neurological studies. There is also evidence that boys present differently from girls, but this text offers no insight as to why prevalence rates vary between genders (Jack, 40).

Simmons & Alderson (2018) *The Official Autism 101 Manual as well as* Das, Papaneophytou & El-Kour (2020) *Autism 360°F both have* similar information regarding diagnosis. These two texts relate this disorder being more common in males than females and the age of diagnosis in post-birth to the end of year 3 (Simmons, 91) (Das, 316).

Diagnosis Summary: 1990-2020

Diagnostic traits among these three decades had the least amount of variability, but they also had the least number of answers. I aimed to answer whether there are different diagnostic procedures for different genders. The textbook consensus was that autism was more common in males than in females, but my question was not answered. I wanted to know if this was just a male-dominated disorder or if females are less likely to get diagnosed because they present with different symptoms. I wanted to know if there were different diagnostic procedures for different gender. I was not able to answer this question.

Support: What options did the texts offer to support individuals diagnosed?

Regarding support, Cohen (1998) *Targeting Autism: What We Know, Don't Know, and Can Do to Help Young Children with Autism and Related Disorders* offers a variety of supportive options. The supports suggested in this text include:

> speech therapy, occupational therapy with a sensory integration focus, interactive play therapy, an interactive school program that uses *floor time. (*Floor time is when a diagnosed child will play with therapists and work on social skills and turn-taking through interactive play.)

Another goal in mind that this text referenced was initiating children to communicate their needs and wants. This text focused more on findings relating to helping individuals express their want and need rather than vitamin therapies or medication to work on behavioral differences (Cohen, 115).

Toward the end of the decade, Mahwah & Erlbaum (1999) *Identification, Education, and Treatment* discusses use of antipsychotics being followed by stimulants and antianxiety drugs to reduce behavioral problems rather than support the individual with ASD. This text stated that this medication was primarily used as a sedative rather than to help the individual achieve meaningful skills (Mahwah & Erlbaum, 122).

Gabriels & Hills (2002) *Research to Individualized Practice* referenced the use of Fluoxetine. Fluoxetine is an antidepressant, a part of the serotonin reuptake inhibitors drug group. This text said that these drugs helped control behavioral outbursts, but these drugs did not target social and behavioral deficits seen in diagnosed individuals, such as lack of eye contact. The other approaches aimed to target ASD characteristics through behavior-learning interventions. The goal of the intervention was to decrease

inappropriate behavior and improve the quality of parent-child relationships (Gabriels & Hills, 81).

Toward the middle of the 2000-2010 decade, Department of Health (2005) *What we know* stated that the clinical trial of secretin hormones being injected into individuals with autism through the approval of the *Network on the Neurobiology and Genetics of Autism: Collaborative Programs of Excellence in Autism (CPEAs).* This foundation's goal was to aid in the digestion of individuals to promote gut health to reverse the immunological theory. The results of this theory found that there were no differences between the secretin hormone and the placebo. This text talked about various medical treatments that were used in clinical trials that did not work. It was interesting reading all the clinical trials that took place when there was no concrete causation to how ASD developed. The use of clinical trials to inject diagnosed individuals with various hormones seems premature when there is no known cause and no specific hormone causing ASD (U.S. Department of Health, 11).

In Cohen (2007) *Targeting Autism: What We Know, Don't Know, and Can Do to Help Young Children with Autism Spectrum Disorders* the term Applied Behavioral Analysis (ABA) was stated for the first time. ABA aims to control unwanted behavior and seek compliance. This text talks about ABA as the only intervention treatment out to date that shows positive progress in decreasing core ASD deficits. This text does not mention the need to communicate with a diagnosed individual on their level but rather bring the diagnosed individual to the level of their communicative partner. To date, no texts focus on the individual instead of their diagnosis (Cohen, 103).

The last book I reviewed in the 2000-2010 decade was: Oller & Oller (2010) *Autism: The Diagnosis, Treatment, & Etiology of the Undeniable Epidemic.* The main point of this text is the premise that prevention is the best protection. I included a chart that states that "Stage 0" should be the first stage when combatting a diagnosis to try to reverse these effects. This chart alarmed me, and only further pointed out the high variability of knowledge seen in what we know about this disorder. Previous texts defraud the theory that dietary changes are what affect autism, while this text makes a chart on prevention starting with diet (Oller & Oller, 346).

Stage 0: Prevention

The health and wellness intervention cycle logically begins at Stage 0 with prevention. It is better, of course, not to be injured by toxins—pesticides, preservatives, dental amalgam, thimerosal, foreign proteins in vaccines, animal wastes in food or drink, industrial pollution, and so on. It is also better

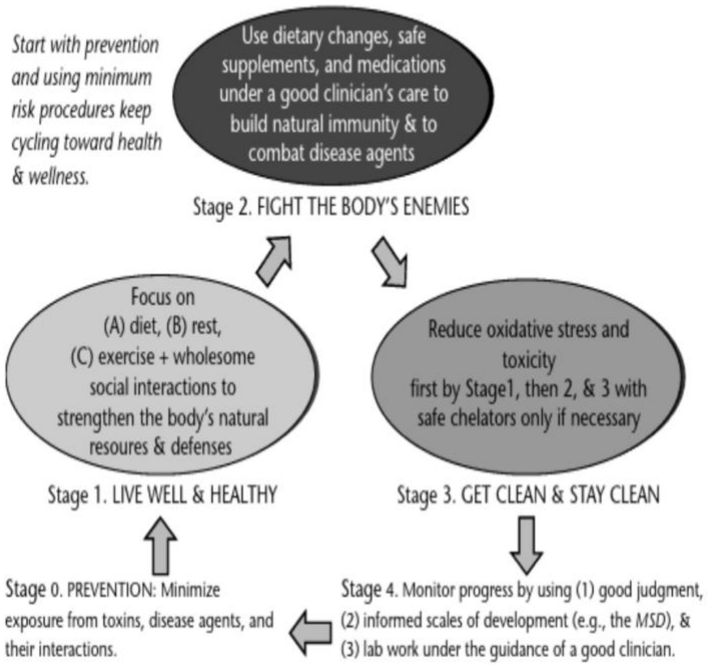

Figure 12-1 A summary of the health and wellness cycle distilled from research on the biochemistry and treatment of autism.

Oxford University Press: (2011) *Autism Spectrum Disorders* states that FDA-approved Risperidone is used to treat behavioral characteristics of autism. This drug is an antipsychotic used to treat schizophrenia, bipolar disorder, and irritability caused by autism. This medication is still currently prescribed to cases of individuals who have severe autism. This text refers to medication as a form of treatment for autism and talks about how antipsychotics should be combined with ABA. The recommendation for ABA is that this text is intensive therapy consisting of 20-40 hours a week. This text also noted that these interventions were only conducted on a handful of children and do not account for changes seen when used on a larger population with different severity of autism (Oxford University Press, 150).

Simmons (2018) *The Official Autism 101 Manual* stated that long-term outcome is highly variable and that "a small percentage of children lose their diagnosis over time, while others remain severely affected." This text talked about the SRIs (Serotonin Reuptake Inhibitors) help with symptoms of anxiety and obsessive-compulsive behaviors, but there is no guarantee, and the medication has varying effects from individual to individual. However, the side effects seen in using this type of medication should be noted, and the outcomes are not worth the risks of SRI drugs being used as trial and error. Some medications were known to cause seizures, while others caused weight gain or weight loss. The side effects were variable, and this text stated that they could cure a disorder that is claimed incurable by other texts (Simmons, 94).

The text, Das, Papaneophytou & El-Kour (2020) *Autism 360℉* supports inclusion with the help of medication. The medication given to children in this research

text were antipsychotics and antidepressants. Instead of these medications reversing autism like the intended purpose, they appeared to cause more harm. The side effects of the medication included significant weight gain from the serotonin-blocking actions seen in these drugs. The blocking action decreased the sensitivity of the satiety feedback center, leading to overeating and weight gain. Another side effect was reduced awareness leading to the medication becoming more of a sedative than an enhancing drug to improve quality of life. Following the text's discussion of medication, was a discussion on the importance of inclusion. This text talked about how the inclusion of children with ASD into classrooms of children who did not have ASD would allow them to learn material and social skills; however, many children who were integrated into neurotypical classrooms were heavily medication and required an inclusion assistant. This text did not discuss neurodivergent thinking in classrooms but rather how to integrate neurodivergent learning to fit in with neurotypical learning (Das, 304).

Support Summary: 1990-2020

Across these texts, there were no consistent areas where methods of supporting children diagnosed with ASD seemed beneficial. Many texts would offer antipsychotics as a form of medication to help reduce inappropriate behaviors brought out by this disorder. However, many of these prescribed medications had harmful side effects and did more bad than good. I found it interesting that these were the same textbooks I used for causes, and none of these texts could pinpoint a direct cause but were prescribing medications anyway. What is the point of using a medication to fix something that has no

area of cause? These texts seemed to want to fix a behavioral problem and focused more on the diagnosis rather than the individual. The later texts talked about education for diagnosed individuals, but the form of education desired was to reach neurotypical assimilation. There are different areas of the spectrum, so there needs to be individualized support options. Not every person learns or thinks the same way, so this should be considered when discussing autism. Most, if not all, of these texts looked at autism as a broad term rather than realizing autism is a complex disorder with many moving parts. On one end of the spectrum may be more severe, and the support system for that individual may need to be more hands-on. An individual may need less hands-on support on the higher-functioning end of the spectrum. It appears that the use of antipsychotic medications has been branded as a quick fix but may be doing more harm than good.

Chapter 4: Summary of Research

Going into this thesis project, I thought that creating a timeline of research advancements would be clear-cut and almost linear. It was not until I completed the research that I realized how high the variability is for autism research. As one subject emerges, it disappears in another text to resurface again. It is like everyone writing these texts is on a different planet. For example, the leading cause in the first decade (1990-1999) was linked to schizophrenia, but in the following decade (2000-2009) it was debunked. The real cause is yet unknown. When we look at the most recent decade investigated (2010-2020), the recommended medication for one with autism is antipsychotics because of the link seen in schizophrenia. In the beginning of my research, I was naive to think that autism research was more advanced than it truly is.

There is a gap is known as the research-to-practice gap. This is where there is ample research but no clinical practice to back up research findings. The article, Greenwell & Walsh (2023) *Evidence-Based Practice in Speech-Language Pathology: Where Are We Now?*, states the Evidence-Based Practice (EBP) gap is widely recognized in the field of Speech-Language Pathologists (SLP). This article sent out surveys to 2,762 school-based SLPs, and it was found that 70% of the SLPs knew about EBP but were not exposed to it through ongoing education. The results of this study concluded that the last time SLPs used EBP was in graduate school. The lack of continuing education comes from the lack of time and funds, which further elongated the EBP gap (Greenwell & Walsh 190).

The research behind using EBP is few but beneficial. The article I read found that the results were improved health outcomes, reduction in care costs, and advancements in clients' overall well-being. The barrier behind more use of EBP is simply from lack of time and funding. EBP takes a long time to carry out, and when SLPs are in their workplace, the workload does not allow for extra time for research. If there were more time allocated to EBP, this would close the research-to-practice gap.

Conclusion:

The variability in research on autism is undeniable. There is no consistency among the textbooks, and many questions have been left unanswered. But what is left unanswered can be just as important as what has been answered. The increasing prevalence rate and lack of qualitative research can be a great start to spending more time researching what we know about autism. Knowledge of autism is primarily written by

researchers who don’t have autism and are writing about things they know nothing about. Textbooks would be more reliable if researchers began to include individuals diagnosed with autism. Although this would be difficult with nonverbal individuals, there are individuals who are higher functioning and can articulate what it is like to be someone living with autism. If their personal accounts were considered, then the way to diagnose and support neurodivergent would change for the better. The lack of representation should also be considered a contributing factor to the research-to-practice gap.

Chapter 5: Future Developments

I would like to continue my research project by talking with individuals on the spectrum to see when they were diagnosed, then compare the timeline to each decade that I have researched. This would show me a smaller-scale version of the EBP gap. For example, if a 20-year-old individual were diagnosed at 10, then I would go back to the year 2010 and see what the diagnosis procedure was and compare that procedure to the procedure they received. This would show me if they were diagnosed with the current 2010 procedures or if their diagnosis was outdated and more in line with the 1990-1999 procedures.

I would also like to ensure that my future projects include comprehensive information from individuals diagnosed with ASD, so that the research represents both textbook research as well as life experiences from those who live with ASD. My desire is to document Autism Spectrum Disorder, or Neurodiversity, most accurately, which means researching and including life experiences of those diagnosed with ASD.

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