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# Autism Spectrum Disorder Determent: A Lifelong-Oriented Strategy

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Abstract: A complex and highly heritable disorder, autism spectrum disorder (ASD) is caused by a combination of environmental variables and genetics that combine to enhance the disorder's risk and produce a range of clinical presentations and outcomes. Furthermore, throughout the first three years of life, children with ASD "create" what we understand to be autism syndrome due to the underlying, intrinsic deficiencies in social attention and interaction that are associated with the disorder. Later in life, an individual with ASD's quality of life and adaptability will be determined by their training and education, the existence and management of comorbidities, and social and vocational support throughout their career. In light of the overall prevalence of ASD, investigating preventative measures appears to be a financially prudent endeavor. We approach prevention in this study from a life course perspective. We will go over the options for controlling risk factors from the time of conception to the birth, for early intervention throughout the first three years of life, and for providing guidance and assistance from childhood to adulthood

Keywords: Autism Spectrum Disorder; prevention; life course approach; risk factors; early intervention

### I. INTRODUCTION

The neurodevelopmental disorder known as autism spectrum disorder (ASD) is characterized by impairments in social relationships, social communication, and confined, repetitive patterns of behavior, interest, or activity. The concept of a spectrum captures the differences between individuals with autism in terms of symptoms, intelligence and language abilities, neuropsychological underpinnings (cognitive style, information processing, and sensory deviations), aetiology and comorbidities, as well as functioning, adaptation, and wellbeing. Autism spectrum disorder (ASD) is a highly heterogeneous condition.

With a very conservative estimated ASD prevalence of 0.76, the 2010 worldwide burden of ASD was estimated to be 111 Disability-Adjusted Life Years per 100,000 persons. It should be noted that the prevalence estimate for ASD has increased to 1.85% based on the most recent study from the CDC (2020). Nevertheless, this remains the most common mental illness among those with a childhood onset, more so than the combined effects of conduct disorder and attention deficit hyperactivity disorder (ADHD). It ranks fifth among mental illnesses, behind major depressive disorder, anxiety disorders, schizophrenia, and bipolar disorder. In 2014, it was estimated that the lifetime financial expenditures associated with autism spectrum disorder (ASD) in the United States were \$1.4 million for individuals without concomitant intellectual disability and \$2.4 million for those who did. This total cost exceeded the gross domestic product of 139 nations worldwide. These numbers, combined with the fact that, despite recent advancements, there is currently no medication or other biological therapy for ASD, make the adoption of preventative strategies a sensible, economical, and highly necessary step for the scientific community, health authorities, and advocates for people with ASD.

### **Development of ASD**

Prior to talking about preventive measures, we need to understand how autism develops and changes throughout the course of a person's lifetime. This will assist us in choosing the strategies and times for our preventive actions. Strong evidence for a predominant genetic aetiology of ASD can be found in family and twin research. Heritability estimates ranging from 50–95% have been found for over 100 susceptibility genes that are significantly appointed to autism.

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Despite the overwhelming influence of genetic variables, research indicates that ASD may also be caused by environmental risk factors (such as the high but incomplete concordance in monozygotic twins) and gene x environment interactions. A deeper comprehension of epigenetics, on the other hand, has highlighted the idea that disorders are more accurately described as a "mismatch between individual and a specific environment, rather than an abnormality per se." This understanding can help to explain autism as a normal response to an environment that is not ideal for the individual in both a physical and psychosocial context. More specifically, unusual patterns of interactions with the environment and reduced involvement in social interactions are thought to be caused by a combination of hereditary and environmental variables in ASD. These "risk processes" will eventually facilitate the development of full-blown ASD (i.e., "autism will create itself") and will also lead to aberrant social and linguistic brain circuits. Alternatively, they might, as in a vicious cycle, use epigenetics to interfere with the environment and the disorder's indices, social, psychological, and biological aspects from childhood through adolescence and adulthood further influence the overall course of the disease and its burden.

#### **Prevention Approaches**

Three types of prevention are commonly recognized in the field of mental health. First, as a primary preventive strategy that targets a wide range of groups and attempts to lower the occurrence of an illness. Secondly, as a supplementary preventive strategy aimed at lowering the disorder's prevalence (or severity), it targets specific at-risk groups. Thirdly, in order to maintain functional adaptations, the individual's health, and prevent relapses, as a third preventive measure for indicated people. Depending on the age of the intended subjects, these tactics should be used in a life course approach, from the perinatal stage to old age, with a different focus for each type. A more fruitful framework could be to divide prevention in mental health based on the timing of the relevant measures being taken. This is because there is conceptual overlap between the different types of prevention and the populations they target (e.g., a primary intervention could target an at-risk population only), as well as between secondary and tertiary preventions with actual treatment interventions.

According to this approach, which is predicated on the development of autism as previously discussed, primary prevention for autism would involve preconceptional and intrapartum attempts to modify the disorder's underlying causes in an effort to boost resilience and lower incidence. These targets are recognized developmental risk and preventative variables that interact with genetic reasons to contribute to the etiopathology of ASD. In order to mitigate or even prevent the onset of autistic symptomatology, secondary or tertiary preventive strategies will target the developmental cascade that began before to birth over the first two to three years of life. In an effort to maintain or raise the autistic person's degree of adaption and overall wellness, secondary and tertiary preventative techniques are mostly used from childhood and adolescence through adulthood and old age when the ASD is completely evident. They will also stop secondary issues from developing, such severe disruptive behaviors, despair, trouble transitioning, etc. All therapeutic approaches for these age groups, taken as a whole, show inherent preventative value.

We shall examine the options and research findings for preventative initiatives during these three stages of an autistic person's life in this paper.

#### **Prevention Strategies**

#### **Preconception/Perinatal Period**

As was previously said, developmental regression provides evidence that the underlying causes of ASD are complex, involving the interactions of several genes with one another and with environmental influences both during fetal development and, most likely, in the early postnatal stage. Genes determine an individual's initial susceptibility, but epigenetic events resulting from a "toxic environmental load" that occur within a specific critical window cause physiological alterations that ultimately overcome an individual's capacity for adaptation and resilience to develop into a neuro-atypical phenotype. Prevention in this context is to manipulate these elements optimally so that the causative constellation is insufficient to create the ASD phenotype, or at least not fully manifested.

Three categories can be used to group the factors that have greater scientific backing or are the subject of debate and investigation: those that, for a variety of reasons, we are unable to control, those that pertain to the general health of the

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community, and those that are more specifically related to the development of ASD. The latter should primarily, though not alone, be addressed in high-risk groups, such as those with a positive family history of ASD.

The primary example in the first group of factors that raise the risk of ASD is the age of the parents (older fathers, older or very young moms, and increasing age disparities between parents). Similarly, in order to treat infertility in a marriage, assisted reproductive technology (ART) is frequently a required option. Despite worries from medical professionals, it is still unclear how ART affects the likelihood of ASD, particularly when looking at ART as a whole. However, certain pertinent therapies (such as intracytoplasmic sperm injection) may increase the additive risk of autism spectrum disorder. Because males have a significantly greater prevalence of ASD (4:1) and a higher risk of recurrence when they have a sibling with an ASD diagnosis, assessing the potential risk for ART becomes more relevant if the procedures are employed for gender selection. If a couple chooses a female and has one autistic child, the overall risk reduction is estimated to be 3%, and if they have two autistic children, it is 15%. Gender selection may increase the residual risk of ASD to the amount (unknown) that ART raises the chance of ASD; however, consideration should also be given to the increased risk of birth abnormalities and other unfavorable pregnancy outcomes associated with ART. This "preventive" method raises ethical questions as well. In general, the decision to select a gender seems more reasonable when there would be a significant reduction in risk, such as in the case of a positive family history of ASD.

Because the second category's characteristics have impacts that transcend beyond the pathophysiology of ASD, they should be targeted for preventative efforts affecting the entire population. The "prevention paradox" states that such successful broad-based preventative initiatives can have a greater predicted impact on the incidence of ASD than those that just target the high-risk demographic.

Although it has recently been demonstrated that maternal hypothyroidism during pregnancy also raises the risk of ASD, congenital hypothyroidism is linked to mental impairments and may even play a role in the pathogenesis of ASD. In most countries, screening for congenital hypothyroidism is a well-established technique that should be combined with pertinent tests when a woman is pregnant.

The general public's health is also impacted by chemical and air pollution. The effects of endocrine-disrupting chemicals (such as those found in pesticides, plastics, scents, etc.) and air pollution (i.e., hazardous air pollutants and air pollutants standards as defined in the USA) on the risk of ASD are not well-established. Nonetheless, several pesticides have been linked in research using more advanced designs for the measurements of actual exposure. Interestingly, increasing folic acid consumption during the first month of pregnancy can reduce the effects of prenatal pesticide exposure. The use of pesticides and other chemicals, as well as the permissible "safe levels" of different pollutants, must be strictly regulated by the relevant environmental protection agencies in light of data regarding ASD and other health impacts.

Preterm birth, stillbirth, reduced growth, difficulties during labor, miscarriages, and abrupt, unexpected infant death are all associated with maternal smoking during pregnancy, in addition to long-term consequences like asthma and behavioral issues or ADHD. A positive correlation is found in more in-depth analyses of confounding factors such as smoking severity and secondhand smoke, despite several meta-analyses reporting a noncontribution to ASD risk. Pregnancy-related smoking cessation programs must be implemented, especially for women from lower socioeconomic backgrounds.

Premature births and those with bilateral foetal growth deviances have been consistently linked to a higher prevalence of Autism Spectrum Disorder (ASD), in addition to the numerous negative effects of reduced or increased foetal growth and preterm deliveries on the overall health of the child. Although it's unclear if these are risk factors in and of themselves or the manifestation of other factors, general precautions for the best clinical care of expectant mothers should be taken to lower their incidence. Study after study has shown that children with ASD are more likely to experience pregnancy complications and obstetric suboptimality, such as preeclampsia. It appears more likely, though, that this is an epiphenomenon of ASD or the outcome of a common risk factor. On the other hand, a number of metabolic conditions (higher glucose, triglycerides, cholesterol, leptin, and proinflammatory immune markers) as well as maternal obesity and gestational diabetes may significantly raise the risk for ASD. Relevant to have a statement of the outcome of a common risk factor.

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programs should be implemented for the prevention of short- and long-term health risks for the mother, developing foetus, and offspring, as well as for the reduction of ASD risk.

The third category's risk factors are more relevant to ASD, without, of course, having broader effects on the progeny. In high-risk cases, these factors ought to be the main targets of preventive measures. Whether the immune reactions resulted from maternal autoimmune conditions or from infections—of which congenital rubella is the historical paradigm—they represent additive risk factors during pregnancy. It has been discovered that the risk of ASD can increase up to threefold in cases of maternal influenza, other viral infections in the first trimester, bacterial infections, and prolonged fever in the second trimester. Together with vitamin D levels or pesticide use, these latter data may help to explain the seasonality of births reported in multiple studies. General preventive measures to avoid infections should be put into place for the entire population in order to reduce the risk of ASD. For mothers at high risk for ASD, vaccination programs should be offered, especially for such well-established risk infectious factors like rubella, while a more aggressive treatment of the fever itself should be the clinical practice. In the case of the circulation of maternal autoantibodies, several therapeutic interventions have been proposed as possible preventive measures.

Short interpregnancy intervals (IPI) (<12 months) increase the risk of autism, pos- sibly through nutrient deprivation (which can be further enhanced if the mother breastfeeds) and the subsequent negative effects on birth weight and risk of small-for- gestational-age. The latter two effects have been shown to be mitigated by folic acid use. An alternative explanation for this finding could be bias from cultural parameters and/or lifestyle ones, since a long IPI (>60–84 months) has also been associated with a higher ASD incidence. Thus, in high-risk families, short IPIs should be avoided, and in the case of one, intense supplementation with vitamins, folic acid and minerals, as well as generous nutrition intake during the new pregnancy, could play a protective role.

Although maternal folic acid supplementation is a common preventive measure proposed by many national authorities like the CDC in USA to prevent neural tube defects, its connection to ASD is less conclusive in the literature. However, some studies reported significant protective effects when taken around conception and, especially, in the presence of inherent inefficient folate metabolism. Preconception supplementation in planned pregnancies in high-risk families is a safe and promising prevention measure to help decrease not only the ASD risk but, also, other behavioural problems and delays in language.

Other nutrient deficiencies like vitamin D, iron and polyunsaturated fatty acid (PUFA), especially omega-3, have also less robustly been correlated with a higher risk for ASD. Iron is a critical mineral for brain development and functioning, but the data from two studies for the specific risk for ASD are contradictory. Omega-3 supplementation or fish consumption is associated with a higher IQ and better neurodevelopment, but its correlation with ASD is less clear. However, a big prospective study showed that a higher fat fish consumption prenatally had a protective effect on ASD, even after controlling for the mercury levels bioaccumulating in them. The role of vitamin D in brain development and its functions has been demonstrated in multiple animal and human studies, but its specific role in ASD is still unclear, given the multiple confounding factors in the assessment of their relationship. However, a small open-label prospective study in siblings of probands with ASD found that vitamin D supplementation during pregnancy and breastfeeding or for the first three years of life for those infants that did not breastfeed led to a four-fold reduction in the recurrence rate, possibly through the enhancement of insulin-like growth factor 1 (IGF-1) levels (see below). Vitamin D alone or with omega-3 supplementation has been also tried as a secondary measure of prevention in children with ASD, with equivocal results. Given the current state of data and the relative safety of such an approach, relevant supplementation during pregnancy could be suggested, at least for omega-3 and vitamin D.

Lately ASD has been hypothesised to be an insulin-like growth factor 1 (IGF-1)- related brain dysconnectivity; thus, measures to increase the availability of this molecule postnatally could represent a plausible prevention. This can be attained through breastfeeding for up to one year, and research data has shown that children with ASD were significantly less likely to have been breastfed. Of course, an adequate breast milk IGF-1 concentration is required, a figure that we currently have not determined. As mentioned above, prenatal or postnatal vitamin D supplementation also increases the circulating IGF-1, offering significant protection. Finally, the administration peros of modified forms of IGF in the first year of life can increase the availability of free IGF-1 and protect the hypomyelination of the brain that leads to its dysconnectivity. Although extended breastfeeding could be easily applied as a general prevention measure, intensive vitamin D supplementation or the use of IGF forms can only be suggested for high-risk infants (e.g.,

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siblings of ASD probands, preterm infants, etc.) and, especially, to those with low levels of IGF-1 in the umbilical cord blood at delivery. The effectiveness of these interventions, however, awaits confirmation from relevant studies.

Several medications administered during pregnancy have been incriminated as ASD risk factors. These include the serotonin-reuptake inhibitors—SSRIs (although some debate still exists around the effects of depression itself), the antiepileptics, especially valproate and anti-asthmatic  $\beta$ -2 adrenergic receptor agonists. The avoidance or the substitution of these medications, when possible, sounds like a logical preventive measure, especially for high-risk populations.

The use of modern high thermal intensity ultrasound in the first trimester of preg- nancy of children with ASD was found to correlate with more repetitive behaviours and lower nonverbal IQ, especially so in boys with genetic (ASD-associated Copy Number Variants/CNVs) vulnerabilities. The effect is plausible considering the effects of an ultrasound in utero on the brain structure and behaviours in animal models. Since the toxic window covers the first trimester, avoidance or reasoned use of an ultrasound during this period may decrease the overall risk for ASD.

Finally, although the broad public fear about the influence of vaccinations and, notably, of the Measles Mumps Rubella vaccine, research has established that there is no association between them and the risk of ASD. The modern tendency, though, of parents avoiding the vaccination program of their children not only will have no effect on the ASD risk but also has detrimental effects, such as a substantial increase in morbidity and mortality from preventable diseases, as stated by the WHO, the CDC and the health agencies of many countries.

### Early Intervention in the First Three Years of Life

According to the social motivation theory, the majority of traits associated with ASD impairments in social communication are the "adaptive" results of the infant's innate incapacity to focus and integrate social cues. The aforementioned "mismatch" between the demands of the environment and this heritable fundamental disability cause the newborn to adopt abnormal compensatory reactions and associated deficits, such as a failure to develop language or facial recognition skills. This theory made it possible to implement an intervention that would improve parent-child connection and enrich the environment in an effort to nudge the development of behavior and the brain in the direction of more typical pathways. Animal models that show how environmental enrichment can lessen the impacts of genetic and environmental stressors provide credence to the viability of such an endeavor.

To increase the likelihood and amplitude of the developmental redirection, this intervention should be implemented as early as possible. Other critical points of this approach include the extent of the targeted impairments, that of the second domain, i.e., range of interest/flexibility, and the presence of comorbid intellectual disability and/or language impairment. The brain develops most sensitively during the first two years of life, but as maturity advances, the brain's adaptability decreases. This emphasizes how crucial it is to accurately identify infants who are at risk early on, and research has already made significant progress in this area. With the use of pertinent clinical instruments, it has been demonstrated that by the time a child is 12 months old, they can be distinguished from typically developing children even more at the age of 18 months. Researchers are looking into neurobiological markers like event-related potentials to faces or speech sounds and the brain's rapid growth from birth to 12 months, particularly whether it is followed by a deceleration after that time. A promising sign if larger studies confirm its clinical usefulness is the EEG, which Bosi et al. employed in a series of recent studies to accurately detect subsequently confirmed ASD in newborns as early as three months of age. Parents might not offer the information, especially in more mild cases, so family physicians and pediatricians should be trained to screen for developmental regression as well. This method can also be utilized for a reliable early diagnosis of patients. According to a recent study, the rate of regression quadrupled in prospective studies involving infant siblings, despite the fact that parents report regression in 30-40% of cases when looking back. Early detection can also result from clinical "red flags" (see the CDC or AutismSpeaks lists) and, most importantly, parental concerns. Even with a probable but unconfirmed diagnosis, the existence of behaviors consistent with ASD or general developmental deviances should signal the physician for a thorough examination and the start of intervention, as in these situations, sensitivity is more important than specificity. When parents have worries or warning signs, waiting it out may not be the best course of action for the child's long-term adaptability.

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We chose to examine the real long-term outcomes of early treatments in order to show the viability of such preventative measures. While there are a number of published interventions that have been well-described and have sufficient supporting data, such JASPER, we will only share data from two early interventions here:

Through play and parent-child contact, Paediatric Autism Communication Therapy (PACT) seeks to improve social communication as well as patterns of restricting and repetitive behavior. Parents can learn how to modify interactions in their daily lives and generate opportunities for communication by watching and responding to videos. To achieve the highest degree of contact, the therapists empower and support parents in communicating in a way that is matched to their child's unique competencies. Twelve 1.5-hour sessions are held every two weeks for a duration of six months, with an additional six months of optional monthly maintenance sessions. In order to gradually integrate PACT into their regular family life, parents are urged to practice PACT tactics for at least thirty minutes a day during play or other natural interactions with their kid in between sessions.

Its lack of sensitivity to change may have led to the development of the Brief Observation of Social Communication Change (BOSCC), which was derived from the Autism Diagnostic Observation Scale (ADOS). In the initial evaluation of the intervention compared to treatment as usual, equivocal results were reported, with no significant effects on the prespecified primary outcome measures—total score of the ASOS-G. An additional concern might have been the study sample's mean age of 45 months (compared to the younger group's 24-42 months), which is relevant to the previously discussed decreasing plasticity and has been shown to have a significant long-term impact in early intervention studies. But when the data were reexamined using the updated ADOS-2 calibrated severity ratings (CSS), the PACT intervention was found to have significantly changed the outcomes. Additionally, both assessor- and parent-rated outcomes—language and social communication—as well as parent-rated outcomes. These were thought to be favorable indicators of eventual social and communicative abilities.

More than 70% of the dyadic communication improvements and the symptom changes in ADOS were found to be mediated by gains in dyadic interaction. Therefore, it was hypothesized that enhanced adaptive gains would be detected at a longer follow-up by achieving this intervention's proximal target. In fact, the PACT group had a considerable reduction in ADOS CSS after almost five years, along with improvements in social communication and (surprisingly) symptoms related to repetitive behavior. An additional intriguing discovery indicated that while the initial improvements in parent synchrony vanished during the follow-up, implying parents lost the abilities they had initially acquired, the initial gains in child initiations remained intact, demonstrating the intervention's enduring impact.

The Early Start Denver Model (ESDM) is a play-based, developmental, relationship-focused, and all-encompassing approach designed to support the growth of autistic children's language, play skills, social communication abilities, and interest in other people. The Applied Behavior Analysis (ABA) techniques are further enhanced by the ESDM, which employs a kid-tailored program with skill-development-focused objectives, goals, and exercises. When the parents and other caregivers are with their children, the therapist also teaches them how to use the program. Every three months, the child's progress is evaluated in order to adjust this strategy.

In the first randomised controlled trial study, parents were educated to provide ESDM for at least five hours per week (actual mean 16.3) for two years, while toddlers under 30 months received a mean of 15.2 hours per week of the intervention. They also received 5.2 h/week of additional therapy. The standard community interventions (COM) that were offered in each area—mean 9.1 hours per week of individual therapy and 9.3 hours per week of group interventions—were given to the controls. In the first year of treatment, no significant differences were seen; however, in the second year, significant improvements were seen in the cognitive domains of language and adaptive functioning, but not in the severity of the core autistic symptoms as determined by the ADOS. Probably, the most notable discovery was that the ESDM group's adaptive functioning in everyday life, contrary to what is normally found in ASD, as well to what happened in the COM group, maintained the same speed of growth as the neurotypical children, without falling further behind. A larger multicentre trial only partially replicated the findings. Notably, however, ESDM intervention was found to normalize abnormal EEG activity patterns associated with social engagement and attentiveness, which likely accounts for the changes in social behaviors. In addition to this extremely intensive one-on-one intervention, ESDM has been tested with positive outcomes in group-based community settings and as a parent-coaching format.

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Children in the ESDM group maintained the gains from the first study in the two-year follow-up study, while the COM group overtook them in all domains with the exception of adaptive scores (socialization and composite) and the decrease in ASD symptom severity (repetitive behaviors and ADOS total). The fact that this result was obtained with less hours/week of services (including ABA ones) provided to the ESDM group than to the COM group and that these services were more frequently provided in a group setting lends credence to the intervention's cost-effectiveness.

Using the combined data from these two distinct early interventions, one could contend that secondary prevention during this stage of life is not only necessary and affordable (despite the limitations of the current interventions), but also feasible and can even lower the disorder's prevalence. This is especially true on an individual basis, where such interventions might assist lessen the long-acting impact of the genetic deficits, steering the person towards a more usual developmental trajectory.

The child's increased social interest in contacts, the richness of his or her answers, the synchronization with others, and learning in naturalistic contexts are the fundamental building blocks of these early treatments. In order to do this, a number of elements from other programs can be blended, adjusted, and customized on an individual basis. These should be given to all newborns exhibiting possible ASD behaviors as early as 12 months of age, and they should be given especially to those who are at risk, such as siblings of probands with autism, premature babies, and people with significant visual or auditory impairments.

### From Childhood to Aduldhood

In this third area, we address preventive approaches to maximize and sustain adaption, whereas in the previous sections, we concentrated on prevention tactics primarily aimed at minimizing the incidence, prevalence, and severity of autistic symptoms. While neither the IQ nor the severity of autism symptoms are entirely independent of adaptive functioning, they do not dictate it either. Rather, adaptive functioning captures the interaction between an individual and their contextual settings. The DSM-5's severity classes, which are determined by the degree of support required, represent this. The support that autistic persons get throughout their lives has the power to change their generally unfavorable adult results. Therefore, every intervention, policy, or support provided to an individual from early childhood to old age has a preventive element for their total quality of life.

The (rare in the literature) conceptualization of autism interventions beyond the first few years of life has certain benefits as a preventive measure as well. From the standpoint of the individual, the therapeutic/supportive measures not only improve the quality of life currently experienced, but they also avert certain negative future repercussions that are anticipated as a result of the current "difficulties" that the particular measures are intended to address. For example, sensory integration therapy and techniques will not only benefit behavior, communication, and learning immediately but will also shield the individual from the detrimental cascade that occurs when these are neglected, such as increased or decreased hyper- or hypoactivity, decreased play and learning, challenging behaviors, etc. Viewing the efforts put into various interventions—like creating a visual schedule at home, adapting the classroom, or providing support for an employee with ASD—as an efficient way to achieve a significant future effect—like preventing disruptive and challenging behaviors, etc.). Last but not least, from the standpoint of the family and intervening environment agents (schools, employers, etc.). Last but not least, from the standpoint of the health system, the cost-effectiveness and rationale of any intervention that is taken into consideration for implementation mostly depend on its gains in prevention (e.g., see the applicable ESDM research).

Another theory regarding the preventive effects of the interventions beyond the first crucial years of life is that skill gains come from different compensatory mechanisms, most likely related to dyslexia, rather than from alterations in the fundamental underlying deficits and a return to more normal trajectories. This alternative route is frequently mirrored in learned abilities that, while improving adaptability, can occasionally be somewhat rigid, situation-specific, non-generalizable, and underdeveloped.

### **Preschool to Prepubertal Period**

Preschoolers and younger children are the target audience for most interventions; all chosen components of the intervention should be based on the individual's strengths and weaknesses, with specific short and long-term goals in mind. The following common preventive goals should be the basis for these incredibly diverse intervention efforts:

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(1) Develop social, communication, and everyday living abilities to the fullest extent possible in order to safeguard future autonomy. The TEACCH program (Treatment and Education of Autistic and Related Communication Handicapped Children), intensive behavioral interventions, particularly those with developmental components like the Naturalistic Developmental Behavioural Intervention (NDBI) or the Social Communications/Emotional Regulation/Transactional Support (SCERTS), and a plethora of other targeted interventions (social skills, speech and language, occupational therapies) all claim to target increased adaptation.

(2) Prevent the emergence of difficult behaviors. This might be the outcome of interventions that ensure that the individual's communication needs are met (using the Picture Exchange Communication System, for example); address sensory needs and deviances (using the Sensory Integration Therapy and Diet, appropriate environmental accommodations, etc.); and increase predictability to lower anxiety (using visual schedules, calendars, and other visual cues, for example). Identification and treatment of common medical and psychiatric comorbidities and conditions, such as general health and pains, seizures, constipation and other GI symptoms, sleep issues, ADHD, intellectual disability, learning disorders, bullying, anxiety, etc., at an early stage are essential to achieving this preventive goal.

(3) Encourage and maintain parental involvement in their children's upbringing and acceptance. Parental involvement is a crucial element of professional-based and parent-mediating programs alike, as it helps to reduce costs and time constraints while facilitating the generalization of learned abilities. Parental education and training, though, shouldn't be given in a vacuum. Reduced adaptive adjustment—acceptance and increased feelings of guilt and hopelessness—have a detrimental impact on a parent's physical and psychological health (anxiety, depression, etc.), making it more difficult for them to manage and teach their child. Therefore, any "demands" for training that professionals make of parents should always be supported with actions meant to foster greater acceptance and guarantee the parents' well-being.

(4) Complete academic coursework at a level appropriate to the learner's true cognitive capacity. Despite the fact that savants and those with extraordinary academic accomplishments are known to mimic ASD, the truth is that there is a significant risk of underperformance and a significant variation in their academic accomplishments. In order to ensure a successful inclusion in the appropriate school setting for each child, the previously mentioned goals for obtaining appropriate abilities and behaviors should be covered in conjunction with appropriate support (e.g., visual aids, extra academic or psychological sessions, bullying prevention, social skills training at school, etc.).

### **Older Children and Adolescents**

While most of the earlier objectives are still being pursued in this developmental phase, interventions ought to focus more on providing targeted answers to the pressing issues of the day. Maintaining the consistency of which actions should be taken requires constant attention. The whole clinical picture varies greatly as the individual matures, with some experiencing greater issues while others are doing just as well or even better.

As primary school draws to a close, there may be a propensity to scale back or discontinue certain interventions, particularly if the kid is less inclined to attend or the family becomes weary. But since mild stressors (like switching schools) can have disproportionate effects like catatonia, this could have serious consequences. To avoid behavioral relapses, parents and other caregivers should be extra watchful and thorough while preparing people for changes. During this phase, the parents should continue receiving individual or group training and emotional support. Their developing children's demands are evolving (sexuality is starting to show), and their own emotional needs are still having an effect on them and their relationships. Additionally, parents should constantly reiterate the diagnosis and its implications because they frequently have a tendency to "forget" about it due to the belief that their child will eventually "grow out of" it or that they would "maturate by age." Less than 0.5% of children are no longer diagnosed with autism, and 6.5% more just have it misdiagnosed as another neurological issue, per a recent study.

But additional complex requirements can arise, particularly in higher-functioning persons, which can jeopardize the objectives stated for the preceding phase (autonomy, avoidance of problematic behaviors, and academic successes). These requirements include body changes, sexuality, and more complex peer connections, as well as the revision of all established relationships—a frequent need for adolescents. Preparing the preadolescent autistic person (as well as his or her family) for the impending physical changes, the necessary self-care and hygiene, and the new safety regulations is unquestionably valuable in preventing future incidents. The same is true for sexuality education investigating sexual

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orientation and identity, and taking preventative steps against sexual abuse and early detection of its symptoms. Reports have indicated that individuals with ASD exhibit greater relevant diversity than their neurotypical counterparts.

Although a desire for a social life and relationships is evident during this time, especially for a significant portion of high-functioning ASD individuals, social and relationship-based challenges tend to worsen during this phase, resulting in a deeper sense of isolation and frustration later on. Social skills education, such as that provided by the Program for the Education and Enrichment of Relationship Skills (PEERS), can shield a person from greater isolation and preserve their quality of life in the future. Relevant to the aforementioned are the associated concerns of self-worth and understanding and accepting one's own diagnosis, which have to be explicit preventive goals in psychotherapy and psychoeducation. Because they have reduced self-esteem, people with ASD are more vulnerable to various psychopathological manifestations (such as sadness and anxiety). On the other hand, teens with high-functioning autism frequently have a pressing need to understand and accept their own diagnosis, making it not simply a beneficial practice. By employing the right technique, it is possible to prevent persistent dissatisfaction stemming from a number of misunderstandings and incorrect assumptions about the reasons behind their challenges and to increase ASD self-awareness without lowering self-esteem.

For older kids and teenagers, a last line of defense is to carefully screen for any co-occurring psychopathology that may manifest during these quickly evolving years. Apart from the diagnoses that were confirmed in the previous phase, we can also have conduct, impulse control, and disruptive disorders; OCD; bipolar disorders; schizophrenia spectrum disorders; and an increased prevalence of sadness and anxiety. Because it can be challenging to identify these frequently overlooked supplementary symptoms, particularly in those with lower functioning levels, a diagnosis may go unnoticed, which could have a negative impact on the person's general functioning. However, if the comorbidities are identified, appropriate interventions such as cognitive-behavioral therapy (CBT) and mindfulness-based interventions should be implemented, in addition to psychopharmacology.

### **Transition to Adulthood**

The necessary additional treatments during this crucial time are intended to avoid the depressing circumstances that the CDC has identified for young adults with autism: high rates of underemployment or unemployment, little involvement in post-secondary education, and most of them staying with family. This is especially important for people with higher cognitive abilities, who, while being in need of the aforementioned resources, are frequently denied extra assistance by an adult healthcare system that is underfunded and lacks the necessary expertise.

A detailed plan that takes into account the living conditions, educational and career goals, cognitive capacities, preferences, reasonable expectations from the parents, and community resources should be developed for the lower-functioning individuals. Prevocational and vocational training may be required for this strategy. After that, a variety of options may be pursued, including competitive employment (with some support), employment in sheltered workshops or day services, semi-independent living (with support), and living under constant supervision. In the event that they work in the community, they might require assistance finding and receiving training for the position, communication with employers and other employees, and ongoing personal support to avert unfavorable circumstances that could result in losing their employment (such as the TEACCH program or Project SEARCH).

For those at the top of the range, things are more challenging, so steps along the tripartite of independent living, work, and education should be taken. In order to facilitate person-job matching, a first step should be a career counseling assessment conducted by experts with solid training in both counseling and the disorder. Standardized assessment tools, such as the Autism Work Skills Questionnaire (AWSQ), should be used, along with qualitative approaches to address social communication challenges.

The next phase could lead to either direct work placement or higher study at the appropriate level (postsecondary vocational training, college, university, etc.), depending on the professional option made by the individual (and, to some extent, in partnership with his family). First, supportive measures that target both the academic environment and the students themselves—such as social planning and organization skills—are necessary for the academic and social success of postsecondary education. Few colleges and universities provide this kind of help, and its quality may make the difference between an autistic student who is successful and involved in society and one who drops out or becomes frustrated and unhappy.

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No matter how advanced their cognitive abilities may be, the process of obtaining and maintaining employment particularly one that requires competence and regular compensation—can be extremely difficult for those with ASD. The degree of assistance provided to avoid unemployment varies based on the community's resources as well as the individual's actual needs. It is important to remember that the relevant US law and EU directive, as well as Article 5 of the United Nations Convention on the Rights of Persons with Disabilities, require organizations to provide employees with disabilities with support and "reasonable accommodations" that do not place an undue burden on the business. In order to do this, interventions such as the ones described above for employment help and community coaching are provided, and interview training is chosen in order to reduce a significant obstacle to finding employment. Employment schemes that provide tailored job placements, pre-employment training, lobbying, and ongoing support to guarantee job retention are examples of a more all-encompassing strategy. These more intense programs were determined to be costeffective because they had positive long-term effects on employment.

Independent living presents the last (and related to the earlier ones) obstacle to a successful transitional plan. In addition to the issue of physical housing, independent living for individuals with ASD involves a number of other significant and pertinent concerns, such as daily living activities, socializing, gaining access to health and welfare services, and interacting with the legal system. In order to ascertain the kind of independent living the individual who is about to leave his or her parental home will be able to handle, a methodical and comprehensive evaluation of the individual's overall adaptive skills as well as the community resources that are accessible must be conducted. This evaluation should cover topics such as the kind and location of residential arrangements, transportation requirements, funding sources, kind and quantity of support required, therapeutic support team, etc. The individual with autism must fully participate in the creation of the final plan.

The triptych above illustrates the fundamental difficulties in making the adjustment to adulthood. The only paths to a successful and happy existence are its effective settlement and the previously specified continual demands (social skills, comorbidities vigilance and treatment, and environmental adjustments).

### Adulthood and Old Age

The achievement of a decent quality of life (QoL) is the primary preventative aim for this final (longest) era. Relevant statutes in the USA (Autism CARES Act) and the UK (Autism Act) have lately indicated that urgent needs persist even after a successful transition to adulthood. In light of these regulations, the public is asked to provide persons with ASD who do not have intellectual disabilities with supportive measures in order to maintain and increase their autonomy and involvement, as well as to keep their mental and physical health from declining.

### Prevention for Adult Ages Consists of

(1) The extension, with assistance from the welfare system and local community initiatives, of the practical measures (housing, employment, and social and daily functioning) outlined for the transition period. A person's quality of life can be greatly enhanced by meaningful work, which can provide structure, social inclusion and participation, financial independence and stability, improved housing and recreational opportunities, improved subjective well-being, and protection of their bodily and mental health.

(2) The development of social skills acquired in earlier phases and their adjustment to the environment of the modern age. Group therapies focused on social skills have demonstrated efficacy in enhancing participants' social skills and participation.

(3) Early treatment and vigilance for comorbidities. This is especially crucial at this point since, regardless of the existence of an intellectual handicap, physical and mental health disorders are quite prevalent in adulthood. Individuals diagnosed with Autism Spectrum Disorder (ASD) exhibit innate risk factors, such as difficulty expressing emotions and ideas, inflexibility, and so on. These factors, when combined with pertinent environmental stressors like peer pressure, bullying, social distancing, and so on, can exacerbate mental health issues, particularly anxiety and depression. Before taking medication, these can be lessened with preventive measures such programs that promote social interaction and leisure activities or psychotherapy methods like modified CBT and mindfulness. It is interesting to note that, even in the absence of depression, people with high-functioning ASD (particularly women) report suicidal ideation and planning at higher rates than either psychotics or the general population. In addition, there is a 2.5-fold rise in motality rates among

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people with ASD compared to the general population due to suicide, and there is a higher incidence of general health issues from nearly all systems—neurological epilepsy being the most common—in this community. Physical ailments are more common, which affects people's quality of life (QoL) and increases mortality (16 years lost). The degree to which these increased morbidities and fatalities are a result of health care providers' inadequate awareness and diagnosis, the autistic population's decreased need for assistance (due to communication problems and access issues), as well as health and lifestyle problems associated with ASD, indicates that these conditions can be easily prevented.

### Data for Autism in Old Age is Scarce

Although the trajectory of the neuropsychological indices of autism is unclear, certain symptoms, like restricted and repetitive behaviors and anomalous sensory experiences, may improve, most likely as a result of compensatory mechanisms. The needs and risks associated with neurotypical old age are, however, made worse by the accumulation of issues discussed for the earlier stages (economic, social, affective, and environmental, as well as physical and mental health, plus the effects of long-term antipsychotic use) and the disorder's characteristics, which are combined with the decreasing or elimination of supportive interventions. People with autism are therefore more likely to experience rejection and isolation throughout this age period, as well as more psychological symptoms (which defy expectations associated with aging), less well-managed physical challenges, and the startling realization of rapid changes in their bodies. The majority of the recommendations made for older adults in general (healthy lifestyle, balanced diet, frequent exercise, involvement in social activities, attending to medical needs) must to be extended to the group with ASD. In addition, we ought to be aware of any potential decline in our eyesight or hearing and promptly make up for it. The elderly autistic person can also benefit from assistance from a relevant supporting group in accepting his evolving circumstances.

The 2010 article in Atlantic magazine on Case No. 1 in the original 1943 paper by L. Kanner, Donald T., then 77 years old, is a good example of how effective preventive measures throughout the lifespan can retain a good quality of life for ASD people. Donald was first institutionalized for a year and was what we would now call medium-functioning. He had a sharp, repetitive language, a strong interest in numbers, savant math skills, a great rote memory, and a preference for spinning items or himself. After that, he lived for four years on a small farm in the country, where his caretakers made good use of his interests. He went to school there and then in his own tiny village, where his eccentricities were tolerated without being made fun of. After graduating from college, he went on to work as a teller in his family bank, once more utilizing his love of numbers. At the time the article was published, Donald was living alone and receiving financial support from a special fund established by the family. He also drove every day to play golf, had coffee with friends every morning with little to no verbal conversation, and was among the most traveled Americans, often taking up to six days to see new places and take his own pictures of things he saw in pictures. Donald's success story is largely a result of his parents' perseverance and wealth, which can generally be replaced by the community welfare system. However, what really allowed Donald to maintain and improve his exceptional quality of life were the opportunities for social interaction and acceptance from his parents, fellow students, and townspeople.

While the diagnosis of autism is more common in adults and older people, and as adulthood is the longest life stage and hence requires the greatest resources, it is also the life stage with the least amount of funding and research. All of the above-mentioned primary components of human life should be addressed in comprehensive treatments geared to achieve an optimal quality of life. For this demographic, tertiary prevention is essential to the individual's well-being and has generally shown to be an economical strategy.

### **II. CONCLUSION**

The current work chose to summarize the chances for ASD prevention across the course of a person's lifetime. There is proof that autism incidence can be reduced by primary prevention starting in the preconception stage and continuing through the postpartum stage. However, we suggest the establishment of an expert panel to continuously evaluate the published data on the risk factors for ASD and produce state-of-the-art preventive guidelines for each one of them, given the extreme variability in the quality of data published and the frequently contradictory results presented. In addition to the apparent benefits of this endeavor, it will make it easier to conduct more precise preventative intervention trials.

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Targeting the underlying mechanisms of the full phenotype through specific early interventions (<three years of age) has shown promise as a secondary prevention to lower the prevalence of ASD by restoring development to more normalized trajectories rather than just teaching partially functional compensatory skills. This approach's primary concern is not only the creation and accessibility of these early treatments, but also the early identification of people who are at risk, if not confirmed cases, in order to avoid postponing the start of intervention.

We contend that all interventions, regardless of age, possess an innate quality of tertiary prevention, which ought to be the benchmark by which their efficacy is assessed. Enhancing adaptive functioning (better behavioral and psychosocial functioning) through appropriate compensation of skills, behaviors, and impairments and through increased acceptance and understanding from their surroundings (parents, school, peers, and community) is a crucial aspect of this conceptualization. These interventions should be comprehensive, coordinated, based on the needs and developmental realities of each phase, continuous (not ending upon entering adulthood), supported by the welfare system, and combined with ongoing monitoring for medical or psychiatric comorbidities or complications in order to be beneficial for and protective of the quality of life of the individual with autism. These interventions should focus on "maximizing potential, minimizing barriers, and optimizing the person-environment fit," as noted by Lai et al. (2020). Even though these endeavors are difficult, they will only improve the quality of life for those who have autism and allow them to live in society with dignity on par with other people.

In the absence of an aetiological therapy, preventative methods are proving to be both appealing and practical for such a diverse and debilitating disorder, as evidenced by the growing body of scientific data and clinical experiences. Therefore, public health as well as researchers, activists, and stakeholders should prioritize prevention initiatives.

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