Explora Tu Potencial A.C.

Nuevos Avances en el Trastorno del Espectro Autista y Estrategias de Intervención para Padres, Educadores y Profesionales

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- Maestría en Ciencias en **Patología de Habla y Lenguaje** de la Universidad de Texas en el Paso en el 2007.

- **PhD, Psicología** - Canterbury Christ Church University en el Reino Unido en el 2023, miembro de la facultad en el departamento de Terapia de Lenguaje.

- Estableció **True Potential PLLC** en 2008 en Tucson, Arizona, que hasta la fecha atiende a niños pequeños diagnosticados con Trastorno Autista y a sus familias a través de servicios educativos y de terapia de habla y lenguaje. También fundó la organización no gubernamental **Explora tu Potencial, A.C.** en 2013, que sigue dedicada a desarrollar atención especializada ampliamente disponible, efectiva, accesible y sostenible para personas con TEA y sus familias en Ciudad Juárez, México.

- Diseñó y dirigió **múltiples programas de capacitación para padres y educadores**, tanto a pequeña como a gran escala, en escuelas privadas y públicas de México.

Objetivos

1. Revisar el contexto histórico de la conceptualización del autismo.

2. Integrar información relevante y reciente.

3. Sugerir ideas prácticas para padres, educadores y profesionales.

Agenda

PARTE 1
Historia del Autismo

PARTE 2
Neurobiología del Autismo

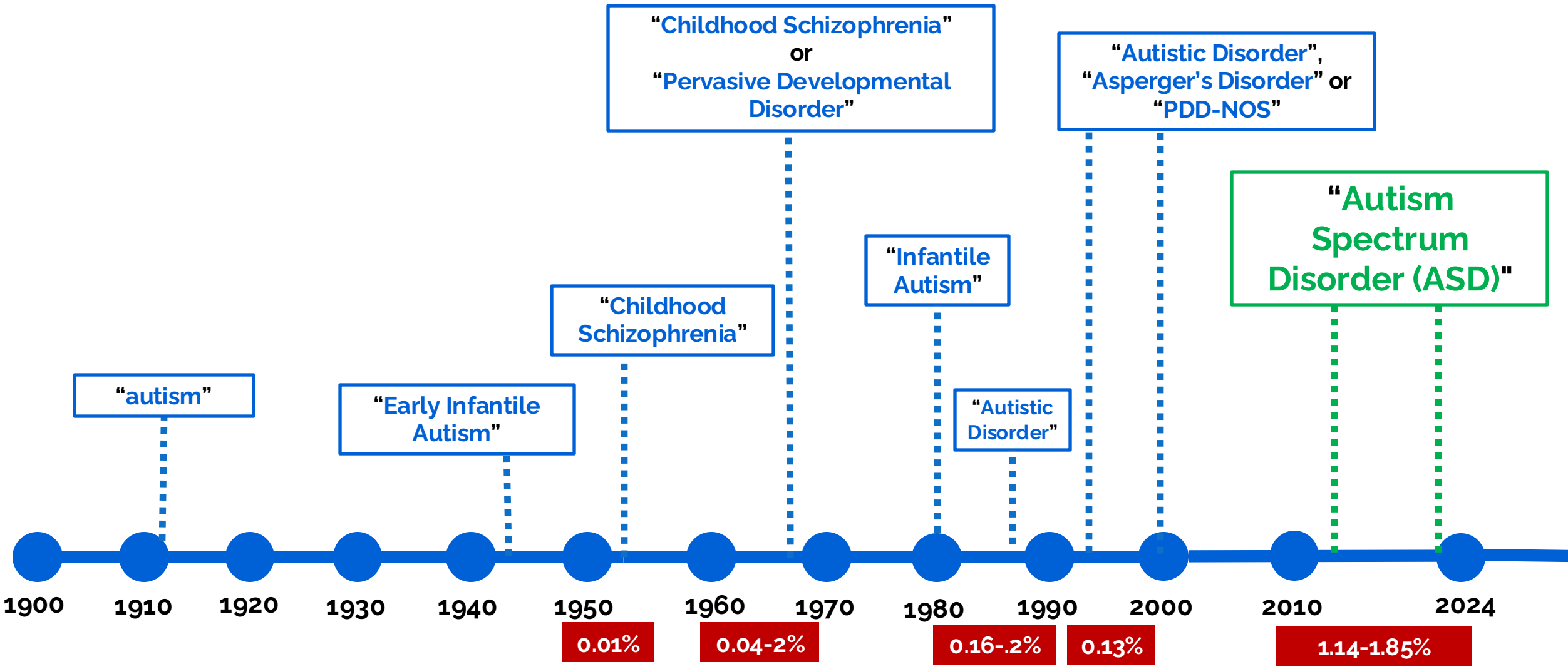
PARTE 3
Inflamación y TEA

PARTE 4
Avenidas de intervención e Investigación

Parte 1

Historia del Autismo

Una breve historia del autismo



1 de 100

1% En México

(2.78% en Estados Unidos)

2024

Trastorno del Espectro Autista (TEA)

A. Trastorno de comunicación

B. Patrones estereotipados

C. Se manifiesta temprano

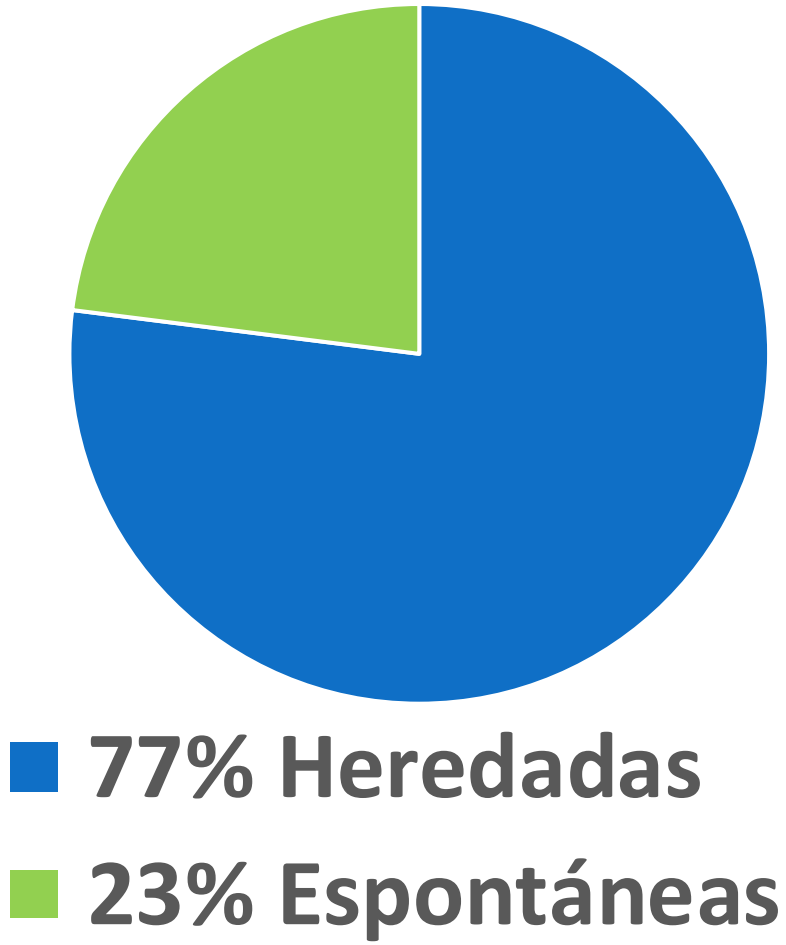
D. Presenta impedimentos significativos

E. No lo explica otro diagnóstico

Parte 2

Neurobiología del Autismo

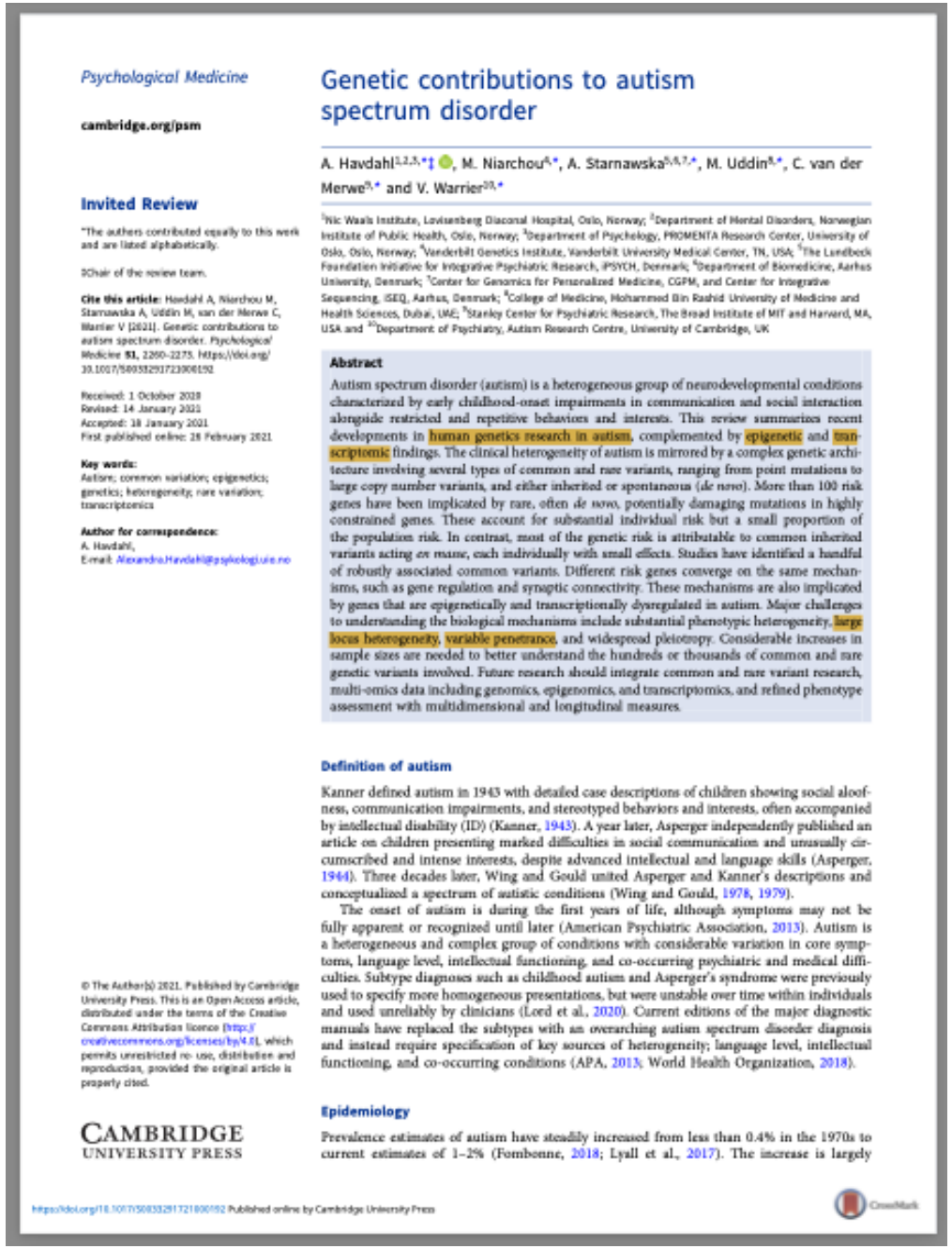
Variantes Genéticas Asociadas con el TEA



El Origen del Autismo

“La mayoría de los investigadores están de acuerdo en que el desarrollo del TEA **no está necesariamente determinado por un solo gen**, sino que es el resultado de una combinación de mutaciones en muchos genes, con un cierto grado de heredabilidad. Sin embargo, el TEA **no es un trastorno genético**. Una variedad de factores de riesgo, como una salud materna deficiente durante el embarazo y el parto, pueden desencadenar mutaciones que producen síntomas de TEA.”

- Chen et al., 2024 p. 8



Psychological Medicine

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Invited Review

*The authors contributed equally to this work and are listed alphabetically.

†Chair of the review team.

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Autism; common variation; epigenetics; genetics; heterogeneity; rare variation; transcriptomics

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Genetic contributions to autism spectrum disorder

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Abstract

Autism spectrum disorder (autism) is a heterogeneous group of neurodevelopmental conditions characterized by early childhood-onset impairments in communication and social interaction alongside restricted and repetitive behaviors and interests. This review summarizes recent developments in human genetics research in autism, complemented by epigenetic and transcriptomic findings. The clinical heterogeneity of autism is mirrored by a complex genetic architecture involving several types of common and rare variants, ranging from point mutations to large copy number variants, and either inherited or spontaneous (*de novo*). More than 100 risk genes have been implicated by rare, often *de novo*, potentially damaging mutations in highly constrained genes. These account for substantial individual risk but a small proportion of the population risk. In contrast, most of the genetic risk is attributable to common inherited variants acting *en masse*, each individually with small effects. Studies have identified a handful of robustly associated common variants. Different risk genes converge on the same mechanisms, such as gene regulation and synaptic connectivity. These mechanisms are also implicated by genes that are epigenetically and transcriptionally dysregulated in autism. Major challenges to understanding the biological mechanisms include substantial phenotypic heterogeneity, large locus heterogeneity, variable penetrance, and widespread pleiotropy. Considerable increases in sample sizes are needed to better understand the hundreds or thousands of common and rare genetic variants involved. Future research should integrate common and rare variant research, multi-omics data including genomics, epigenomics, and transcriptomics, and refined phenotype assessment with multidimensional and longitudinal measures.

Definition of autism

Kanner defined autism in 1943 with detailed case descriptions of children showing social aloofness, communication impairments, and stereotyped behaviors and interests, often accompanied by intellectual disability (ID) (Kanner, 1943). A year later, Asperger independently published an article on children presenting marked difficulties in social communication and unusually circumscribed and intense interests, despite advanced intellectual and language skills (Asperger, 1944). Three decades later, Wing and Gould united Asperger and Kanner's descriptions and conceptualized a spectrum of autistic conditions (Wing and Gould, 1978, 1979).

The onset of autism is during the first years of life, although symptoms may not be fully apparent or recognized until later (American Psychiatric Association, 2013). Autism is a heterogeneous and complex group of conditions with considerable variation in core symptoms, language level, intellectual functioning, and co-occurring psychiatric and medical difficulties. Subtype diagnoses such as childhood autism and Asperger's syndrome were previously used to specify more homogeneous presentations, but were unstable over time within individuals and used unreliably by clinicians (Lord et al., 2020). Current editions of the major diagnostic manuals have replaced the subtypes with an overarching autism spectrum disorder diagnosis and instead require specification of key sources of heterogeneity; language level, intellectual functioning, and co-occurring conditions (APA, 2013; World Health Organization, 2018).

Epidemiology

Prevalence estimates of autism have steadily increased from less than 0.4% in the 1970s to current estimates of 1–2% (Fombonne, 2018; Lyall et al., 2017). The increase is largely

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Genética, Ambiente y Autismo

1. Pesticidas y

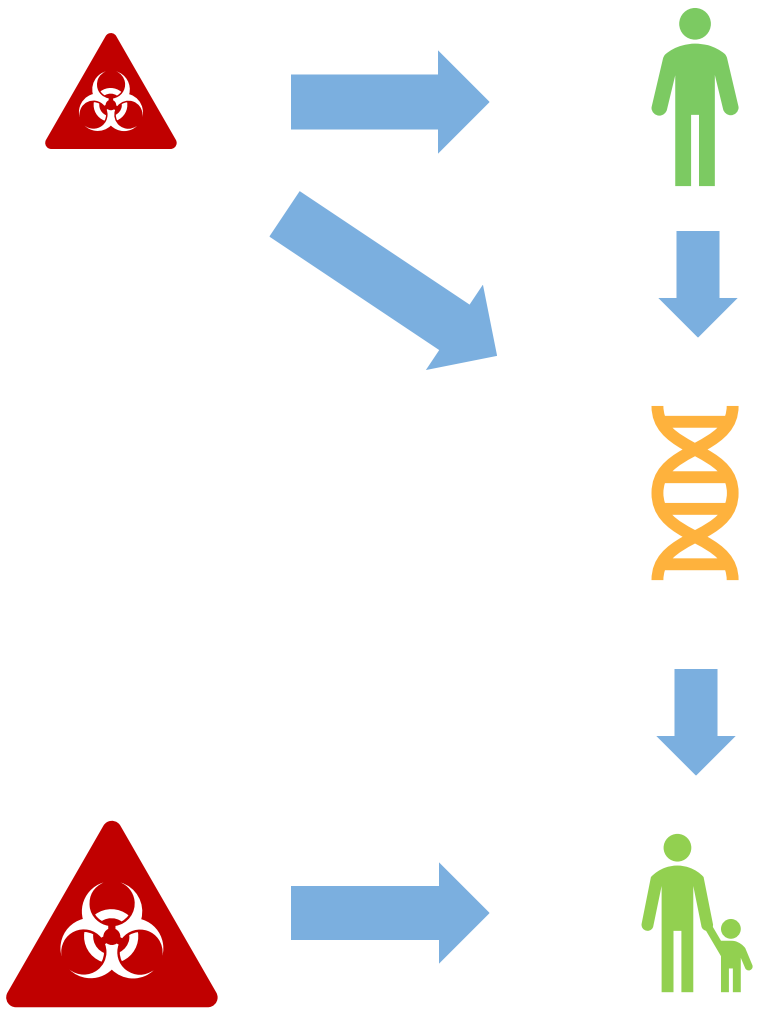
   Herbicidas
2. Medicamentos
3. Metales pesados
4. Microplásticos

Contaminantes

Salud natal y prenatal

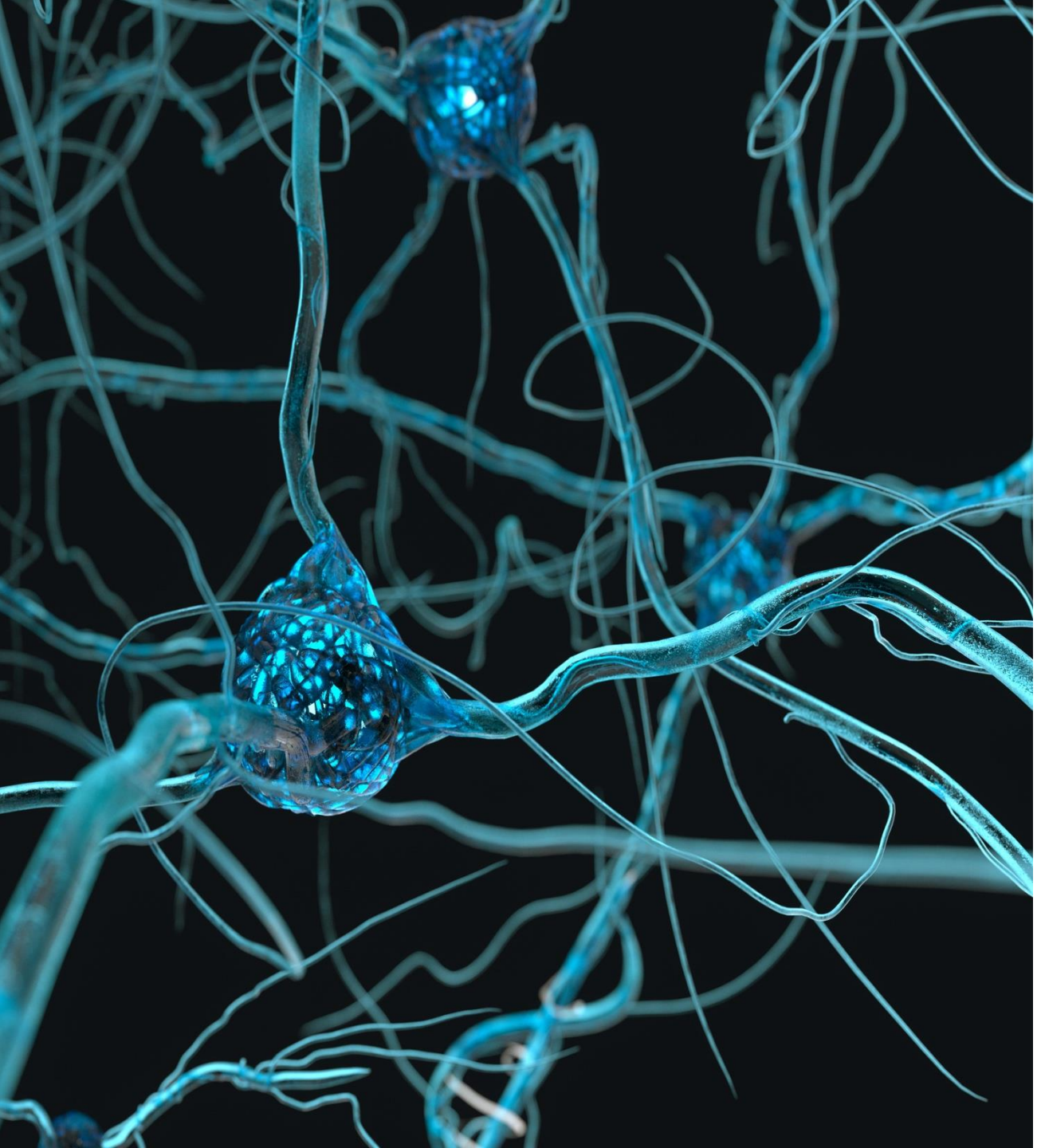
Autismo

El origen del autismo



1. Inflaman el sistema nervioso, resultando en síntomas del TEA
2. Distorsionan el código genético
3. Se repite del ciclo con aumento en cantidad y variedad de contaminantes

Organofosforados

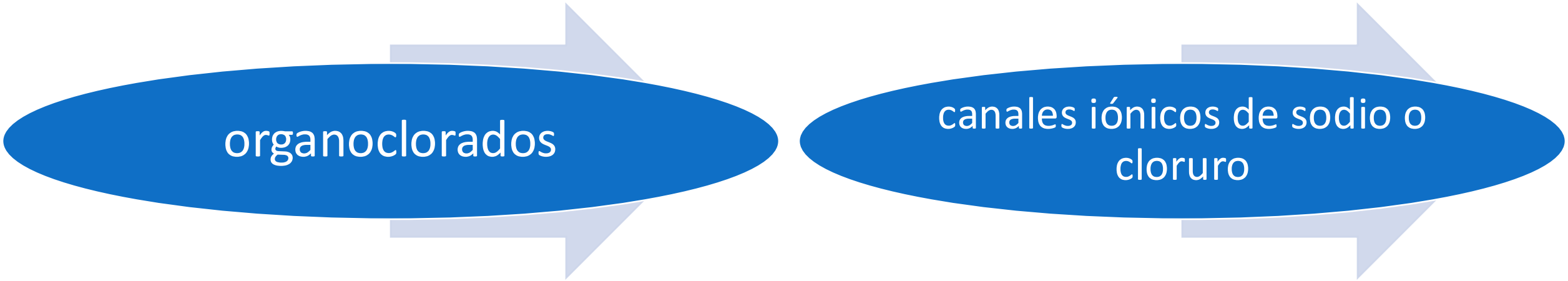


Efectos de los Organofosforados

1. Sobre estimulación del sistema nervioso.

2. Disfunciones en plasticidad sináptica.

Organoclorados



Organoclorados

Se han vinculado con cáncer, obesidad, síndrome metabólico, trastornos reproductivos, deficiencia de vitamina D, función cognitiva, diabetes tipo 2 y disrupción endocrina.

Glifosato

Se han vinculado con cáncer, disrupción endocrina, salud intestinal, alteraciones en el microbioma, daño hepático y renal.

Metales pesados

1. Glifosato puede afectar síntesis de melatonina

2. La melatonina permite la creación de la proteína PIN1

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REVIEW

Journal of Neurochemistry JNC

Is autism a PIN1 deficiency syndrome? A proposed etiologica role for glyphosate

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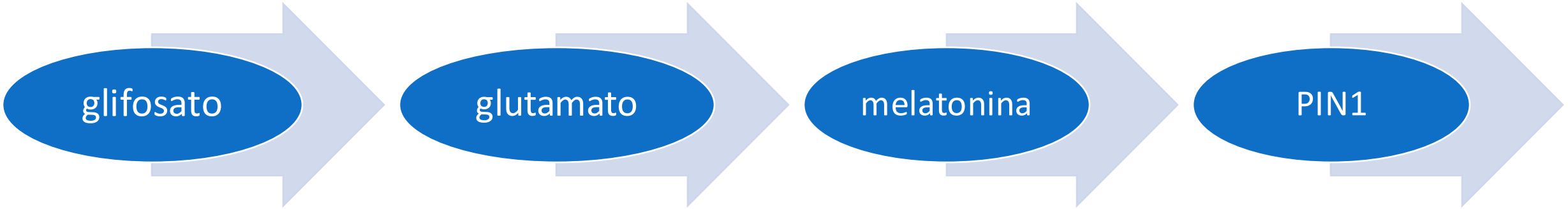
Abstract

Autism is a neurodevelopmental disorder, the prevalence of which has increased
matically in the United States over the past two decades. It is characterized by
reotyped behaviors and impairments in social interaction and communication. In
paper, we present evidence that autism can be viewed as a PIN1 deficiency syndro
Peptidyl-prolyl cis/trans isomerase, NIMA-Interacting 1 (PIN1) is a peptidyl-prolyl
trans isomerase, and it has widespread influences in biological organisms. Bro
speaking, PIN1 deficiency is linked to many neurodegenerative diseases, whe
PIN1 over-expression is linked to cancer. Death-associated protein kinase 1 (DA
strongly inhibits PIN1, and the hormone melatonin inhibits DAPK1. Melatonin
ciency is strongly linked to autism. It has recently been shown that glyphosate expo
to rats inhibits melatonin synthesis as a result of increased glutamate release from
cells and increased expression of metabotropic glutamate receptors. Glyphosate'
hibition of melatonin leads to a reduction in PIN1 availability in neurons. In this pa
we show that PIN1 deficiency can explain many of the unique morphological feat
of autism, including increased dendritic spine density, missing or thin corpus callos
and reduced bone density. We show how PIN1 deficiency disrupts the functio
of powerful high-level signaling molecules, such as nuclear factor erythroid 2-rel
factor 2 (NRF2) and p53. Dysregulation of both of these proteins has been linke
autism. Severe depletion of glutathione in the brain resulting from chronic expo
to oxidative stressors and extracellular glutamate leads to oxidation of the cyst
residue in PIN1, inactivating the protein and further contributing to PIN1 deficie

Abbreviations: (Ser/Thr), (serine/threonine); AD, Alzheimer's disease; ALS, amyotrophic lateral sclerosis; AMPA, α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid; ARD, a
repeat domain; AREs, antioxidant response elements; ASDs, autism spectrum disorders; BTBR, Black and Tan Brachyury; Ca2+/CaM, calcium/calmodulin; CAM, cell adhesion m

Glifosato y Autismo



-Seneff, Kriakopolous, Nigh, 2024

La evidencia

1. Se han publicado estudios cuestionando la relación entre el glifosato y el autismo

2. Estas publicaciones son por parte de Bayer, productor de glifosato

LETTER

REPLY TO REEVES AND DUNN:

Risk for autism in offspring after maternal glyphosate exposure

Kenji Hashimoto[a,1] and Bruce D. Hammock[b,c]

Autism spectrum disorder (ASD) is a developmental disorder that affects communication and behavior. Environmental factors, including exposures to synthetic chemicals (i.e., the herbicide glyphosate) during pregnancy, might increase the risk for ASD (1). A population-based case-control study in California showed that the risk of ASD was associated with the use of glyphosate (odds ratio = 1.16) as well as other pesticides (odds ratio = 1.10 to 1.13) (2). We report that maternal exposure to the formulated glyphosate (0.098%) causes ASD-like behaviors in juvenile murine offspring (3). Furthermore, exposure to formulated glyphosate during pregnancy caused differential expression of microRNAs and antioxidant-related genes in the brain of rodent offspring (4, 5).

Reeves and Dunn (6) point out that ASD-like behaviors after maternal exposure to formulated glyphosate might stem from other ingredients in the formulated herbicides such as Roundup Maxload (48% [wt/vol] glyphosate potassium salt, 52% other ingredients including water and surfactant). Furthermore, they point out that the body weight of pregnant mothers exposed to formulated glyphosate was lower than that of the control group, indicating that the mothers exhibit signs of malnutrition (6). They also point out that a quaternary amine surfactant may affect the outcome in offspring after maternal exposure to formulated glyphosate. Collectively, they address the possibility that the ingredients in Roundup Maxload may contribute to ASD-like behaviors in offspring after maternal exposure to formulated glyphosate.

In contrast, we recently reported that maternal exposure to pure glyphosate (0.098%, Sigma-Aldrich Co., Ltd) caused ASD-like behaviors in male juvenile offspring (7). Body weight of pregnant mothers exposed to pure glyphosate was not different from the control group, indicating that pure glyphosate did not affect the body weight of pregnant mothers (7). Thus, it is possible that the ingredients included in the Roundup Maxload can decrease the body weight of mothers during exposure to formulated glyphosate (3). In addition, a recent study using pure glyphosate showed that maternal exposure to glyphosate during pregnancy caused neurobehavioral alterations (i.e., development of reflexes, motor activity, and cognitive function) (8). Taken together, it is likely that maternal exposure to a high dose of glyphosate without ingredients could cause behavioral abnormalities such as ASD-like behaviors in offspring. Importantly, we know that the rodent data do not necessarily translate to humans. Further research is needed to identify the molecular mechanisms of maternal glyphosate exposure in ASD etiology.

The urinary levels of glyphosate in some occupationally exposed subjects varied from 0.26 μg/L to 73.5 μg/L, and the urinary levels in environmentally exposed subjects ranged from 0.16 μg/L to 7.6 μg/L (9). A prospective birth cohort study showed that >90% of pregnant women had detectable levels of glyphosate (0.1 μg/L) in the urine, and that these levels were correlated significantly with shortened pregnancy lengths (10). Finally, a further cohort study on measurement of blood (or urine) levels of glyphosate and its major metabolite aminomethylphosphonic acid in pregnant mothers who have their offspring with or without ASD is of great interest.

1 L. A. Sealey et al., Environmental factors in the development of autism spectrum disorders. Environ. Int. **88**, 288–298 (2016).
2 O. S. von Ehrenstein et al., Prenatal and infant exposure to ambient pesticides and autism spectrum disorder in children: Population based case-control study. BMJ **364**, l962 (2019).
3 Y. Pu et al., Maternal glyphosate exposure causes autism-like behaviors in offspring through increased expression of soluble epoxide hydrolase. Proc. Natl. Acad. Sci. U.S.A. **117**, 11753–11759 (2020).

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Metales pesados

1. Aluminio
2. Cadmio
3. Cromo
4. Cobre
5. Plomo
6. Arsénico
7. Manganeso
8. Mercurio

Ftalatos

Exposición prenatal ha sido asociada con cambios negativos en comportamiento social.

Parte 3

Inflamación y Autismo

Evidencia de inflamación en el TEA

1. Presencia excesiva de ciertas **citoquinas proinflamatorias** (como IL-1β, IL-6, TNF-α)

2. Respuesta inmune disfuncional (**inflamación**)

pharmaceuticals

MDPI

Review

Inflammation and Neuro-Immune Dysregulations in Autism Spectrum Disorders

Dario Siniscalco [1,2,3,*], Stephen Schultz [4], Anna Lisa Brigida [2] and Nicola Antonucci [5]

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check for updates

Abstract: Autism Spectrum Disorder (ASD) is characterized by persistent deficits in social communication and interaction and restricted-repetitive patterns of behavior, interests, or activities. Strong inflammation states are associated with ASD. This inflammatory condition is often linked to immune system dysfunction. Several cell types are enrolled to trigger and sustain these processes. Neuro-inflammation and neuro-immune abnormalities have now been established in ASD as key factors in its development and maintenance. In this review, we will explore inflammatory conditions, dysfunctions in neuro-immune cross-talk, and immune system treatments in ASD management.

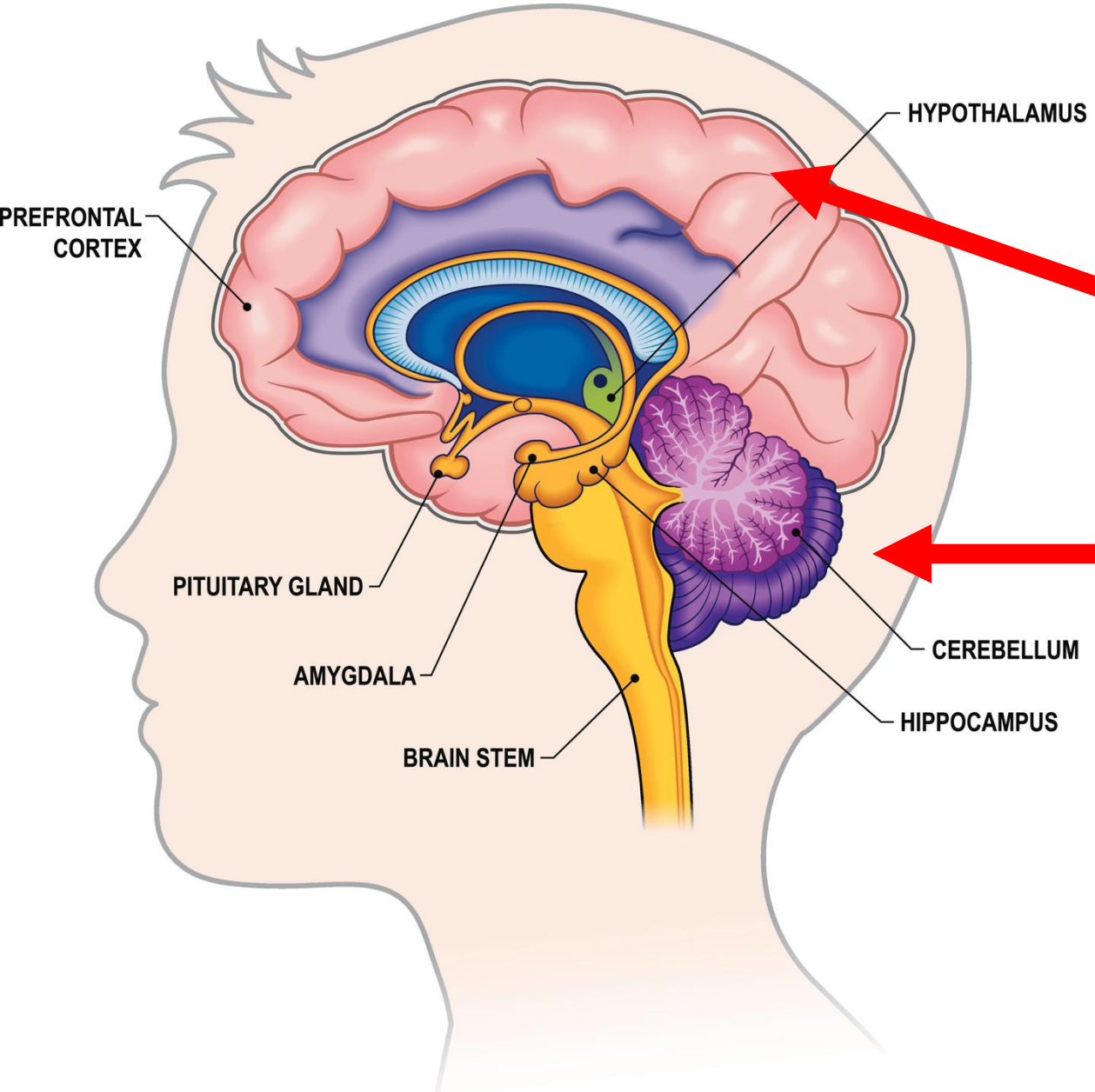
Keywords: autism; monocytes; macrophages; inflammation; neuro-immune system

1. Autism Spectrum Disorder (ASD)

Autism Spectrum Disorder (ASD) is defined by the Diagnostic and Treatment Manual for Mental Disorders, Fifth Edition (DSM-5) and is characterized by persistent deficits in social communication interaction and restricted-repetitive patterns of behavior, interests, or activities. These symptoms begin in early childhood, and produce clinically significant developmental impairment [1]. The DSM-5 combined the previously separate subtypes of ASD listed in the Diagnostic and Statistical Manual for Mental Disorders, Fourth Edition (DSM-4). Autistic disorder, Asperger syndrome, pervasive developmental disorder-not otherwise specified (PDD-NOS), and childhood disintegrative disorder are now combined into one diagnosis of ASD. Two of the prominent clinical features of ASD are inflammation and neuro-immune system dysregulation [2–4].

In a 2013 review article, we summarized environmental factors which could contribute to ASD pathogenesis through epigenetic modifications [5]. Since the publication of that review, additional articles have continued to add to the evidence of epigenetic modifications in ASD. Some of these epigenetic modifications include DNA methylation, epigenetic proteins, gene polymorphisms associated with variation in diet, histone modifications, and microRNA (miRNA) dysregulation [6–10].

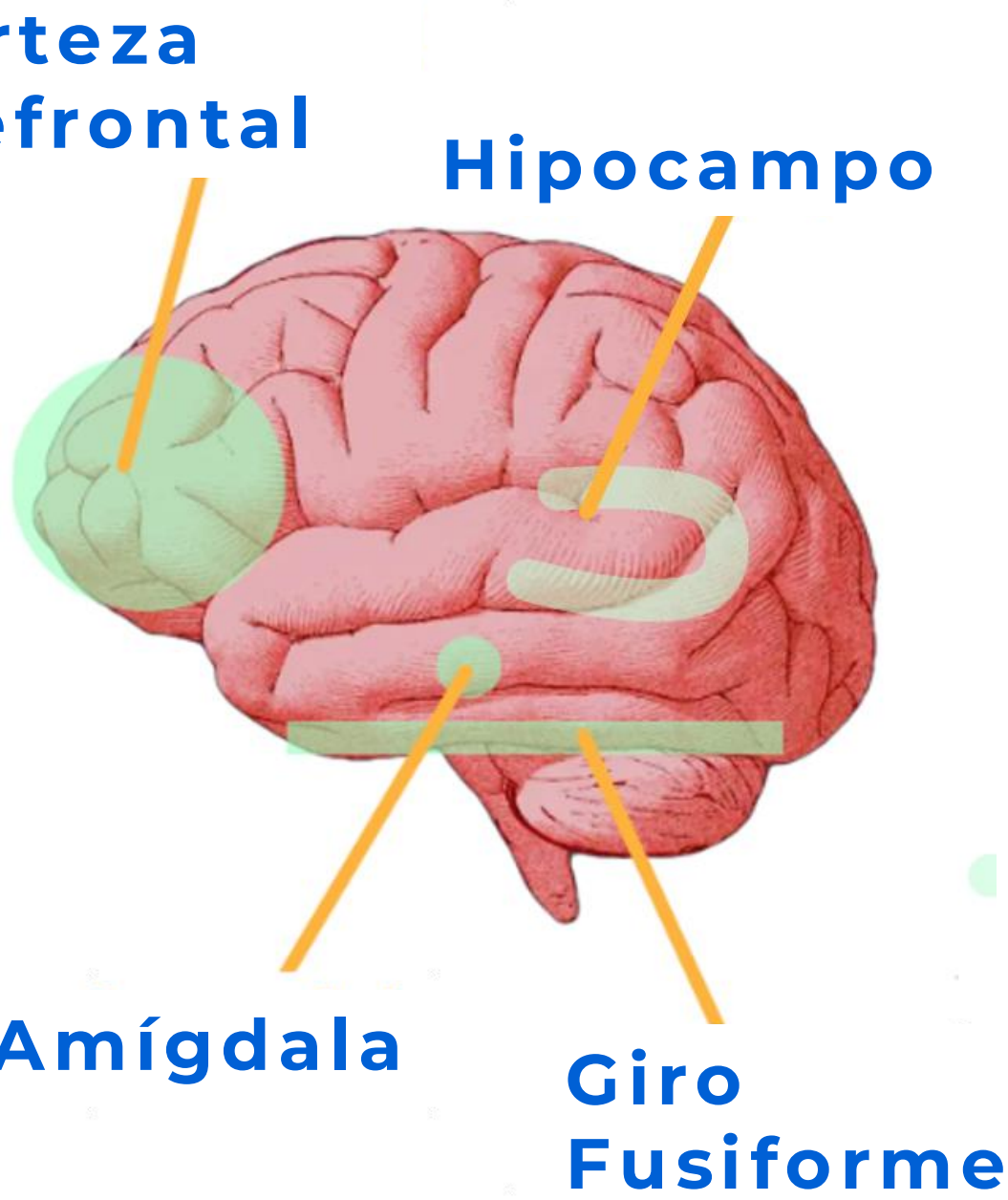
Áreas de neuroinflamación



- Corteza cerebral
- Cerebelo

Otras áreas afectadas

- Comportamiento
- Cognición



Parásitos y autismo

- La presencia de parásitos puede causar neuroinflamación.
- La neuroinflamación es la causa de los síntomas de autismo.

Parte 4

Un Enfoque Integral

Un enfoque integral

- Manejo de inflamación
- Manejo de parásitos
- Terapia adecuada

Dieta antiinflamatoria

1. Dieta antiinflamatoria
2. Eliminación de contaminantes
3. Suplementos antiinflamatorios
4. Reducir exposición a contaminantes

Combatiendo parásitos

1. Seguimiento del microbioma

2. Entorno libre de contaminantes

Terapias adecuadas

1. Terapias conductuales
2. Mucha y variada actividad física
3. Terapias biomédicas

En resumen

1. Un acercamiento comprensivo es necesario

2. Todos tenemos que estar informados

3. Prevención y política publica son importantes

Explora Tu Potencial A.C.

¡Ha sido un placer estar con ustedes!

Para servicios y entrenamiento:

656-346-0358

juarezetp@gmail.com

TalleresdeAutismo.com

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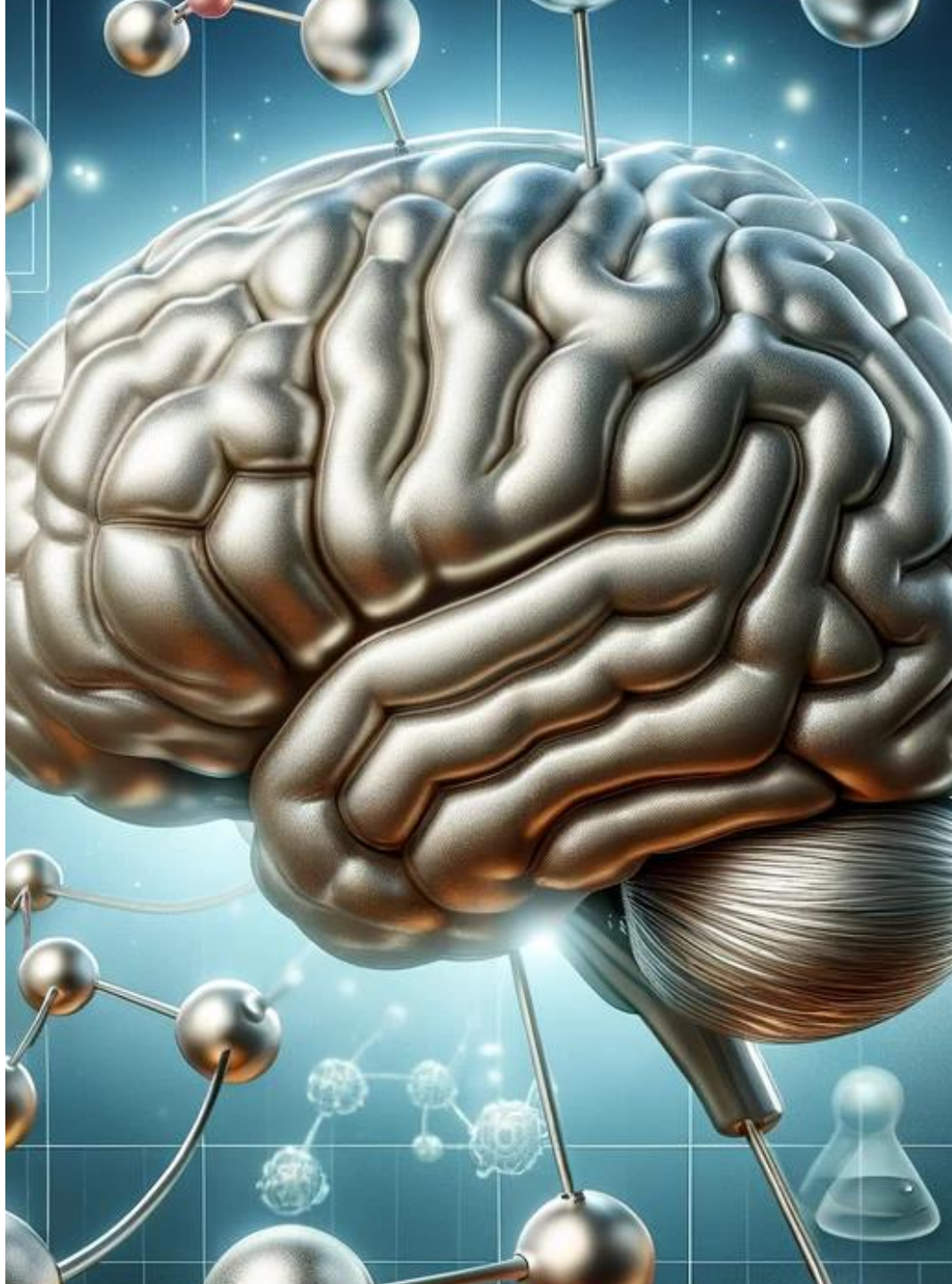
Pre and Postnatal Risk Factors

1. Poor **maternal health**
2. Complications in **childbirth**
3. Exposure to **contaminants** before and after birth

Environmental Risk Factors

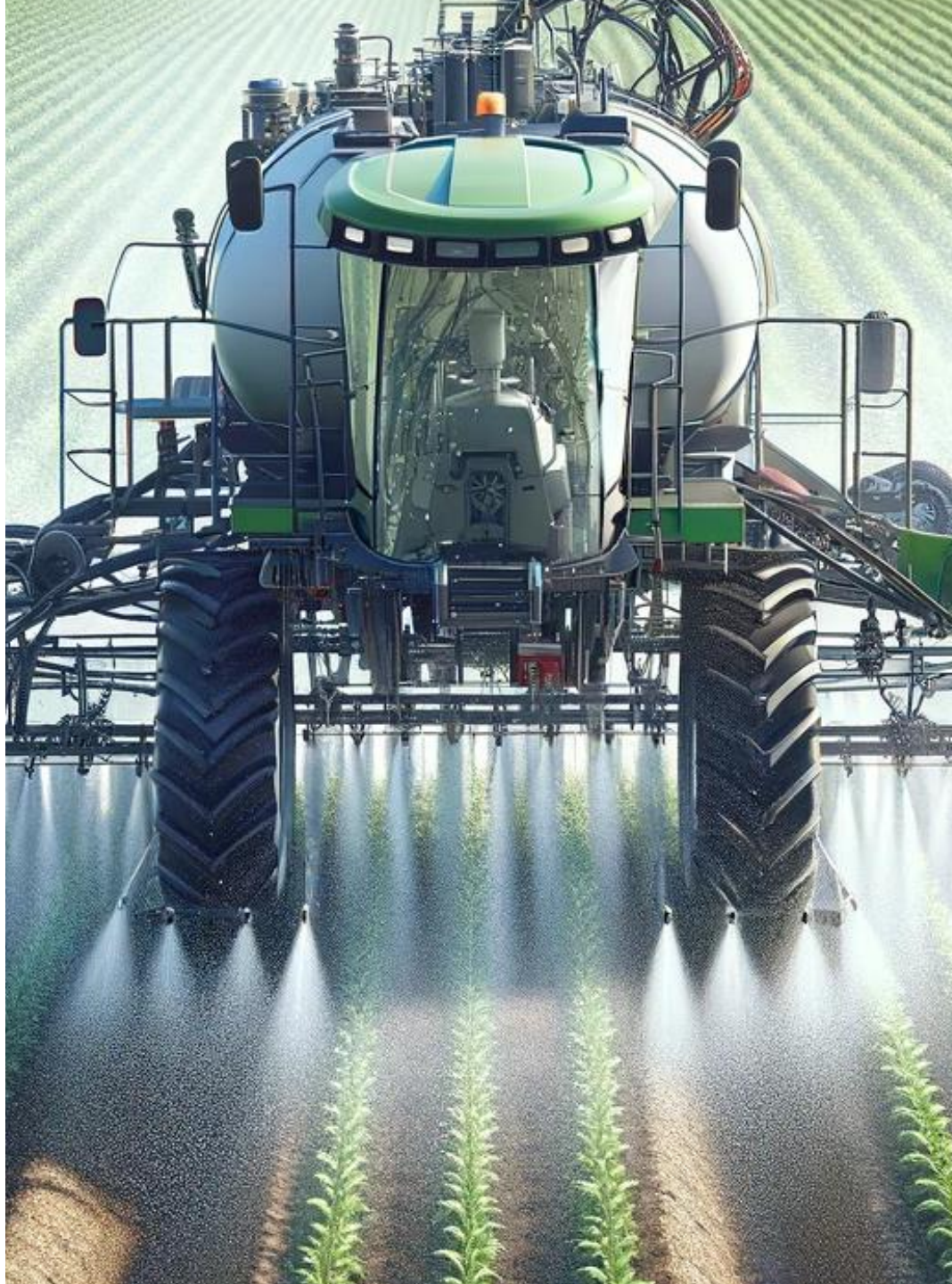
- Heavy metals including **aluminum, cadmium**, **chromium**, **copper**, **lead**, **arsenic, manganese** and **mercury** have found in significantly higher concentrations in individuals diagnosed with ASD (Aschner et al., 2024; Akyuzlu et al., 2014; Ding et al., 2023; Seneff et al., 2012).

- Relationships between **phthalates** and ASD, and other disorders, have been documented through multiple studies (Jeddi et al., 2016). xs



Environmental Risk Factors

Significant relationships have been found between exposure to herbicides like **glyphosate** and pesticides like **organophosphorus** and **organochlorine** (Chen et al., 2024; Pu et al., 2020).



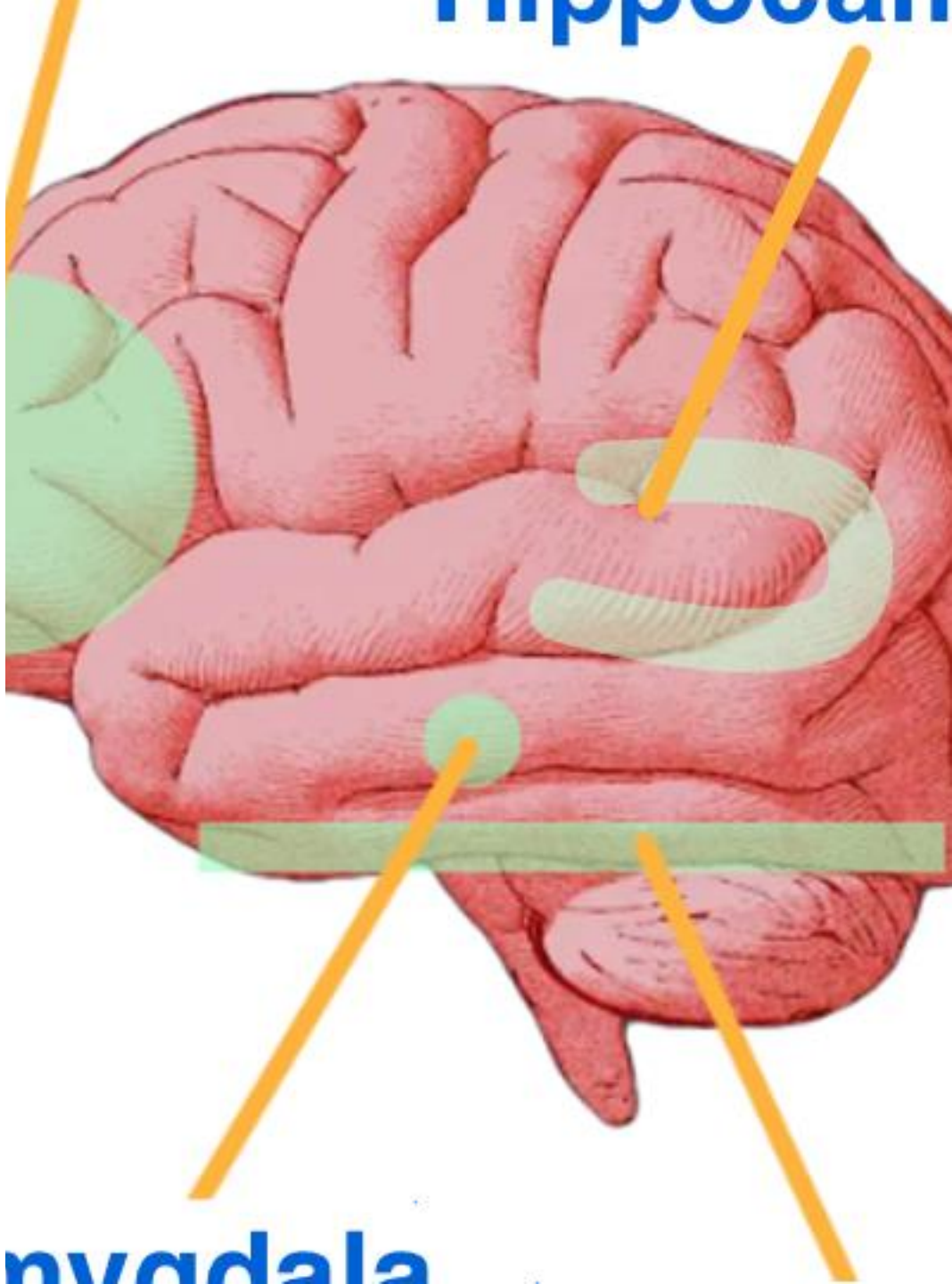
Resulting Differences in Neurological Functioning

Review and learn in detail specific brain structures responsible for:

- **Speech and Language**
- **Other Social Communication Skills**

Some Key Brain Areas

- Hippocampus
- Prefrontal Cortex
- Amygdala
- Fusiform Gyrus



The Hippocampus

Cognitive Mapping

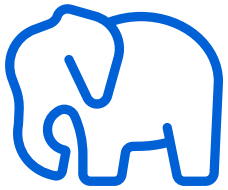
Affordance Perception

Model-Based Planning

The Prefrontal Cortex

Decision Making

Personality and Behavior Regulation



Working Memory

Attention and Concentration

The Amygdala

Key Emotional Processor

Fight or Flight Response

Emotional Memory

Social Interactions

Decision Making

The Fusiform Gyrus

Face Recognition

Reading

Connectivity

Stay informed

- Keep up with the **most current research** on ASD.
- Understand how ASD affects **individuals and those around them**.
- Understand the **wide-ranging** impacts of ASD.

Section III

Current Definition / Diagnostic criteria of ASD (DSM-5-TR)

Terminology

- Autism Spectrum Disorder or (ASD) according to definition in **DSM-5-TR** (APA, 2022)

- Corresponding ICD-11 code is **6A02** (WHO, 2018)

Criteria A

“**Persistent deficits**" in **three areas** of social communication including:

1. **Reciprocity**
2. Comprehension and use of **nonverbal communication**
3. Difficulties establishing and maintaining **social relationships**

Criteria B

At least two of four stereotypical behaviour patterns:

1. **Stereotyped or repetitive** movements
2. Insistence on **sameness**
3. Highly restricted, **fixated interests**
4. **Hyper/hypo reactivity** to specific stimuli

Criteria C, D and E

The observed symptoms :

C. Occurred **early in development**

D. “Cause clinically **significant impairment** in social, occupational, or other important areas of current functioning,” (APA, 2022)

E. Are **not explained by other categories of disability** such as intellectual disability and global developmental delay.

Additional Specifications:

Diagnosis must specify if:

- There are accompanying **intellectual and language impairments**.
- The condition is associated with a known **medical** or **genetic** condition or **environmental** factor
- The condition is associated with another **neurodevelopmental**, **mental**, or **behavioral** problem.

Section IV

Identification and Evaluation of Communication Impairments in ASD

ASD in Early Infancy (0-36 months)

Early Motor Impairments

- Delayed developmental milestones **(C)**
- Slow or stagnant development of gross and fine motor skills **(C)**
- Prone to accidents **(C)**

Early Signs of Social Communication Impairments

- Lack of response to name **(A2)**
- Slow vocabulary development **(A1)**
- Delayed speech or symbolic communication **(A1)**
- Limited reciprocal interactions **(A3)**
- Limited attention to people's faces **(A2)**
- Difficulty establishing nonverbal communication **(A2)**

Repetitive and Restricted Behaviors and Sensory Sensitivities

- Repetitive movements **(B1)**
- Strong adherence to routines **(B2)**
- Intense interests in specific objects **(B3)**
- Over- or under-reactivity to sensory input **(B4)**
- Unusual sensory interests **(B4)**

ASD and Early Childhood (3-6 years)

Social Communication Challenges

- Difficulty with peer interactions **(A1)**
- Limited use of gestures and facial expressions **(A2)**
- Delayed or unusual speech development **(A1)**

Repetitive and Restricted Behaviors

- Repetitive play patterns **(B1)**
- Insistence on sameness **(B2)**
- Intense focus on specific interests **(B3)**

Sensory Sensitivities

- Over- or under-reactivity to sensory stimuli **(B4)**

- Unusual sensory interests **(B4)**

Impact on Learning and Development

- Difficulty with imaginative play **(A3)**
- Challenges in following instructions **(A2)**
- Emotional regulation difficulties **(A3, D)**

Speech and Language Evaluation Tools for Early Infants and Young Children

Standardized Assessment Tools for Early Infants and Young Children

- Social Communication Questionnaire (SCQ; Rutter et al., 2003)
- MacArthur-Bates Communicative Development Inventories (CDI; Fenson et al., 1994)
- Preschool Language Scale, Fifth Edition (PLS-5; Zimmerman et al., 2011)
- Mullen Scales of Early Learning (MSEL; Mullen, 1995)

Non-Standardized Assessment Tools for Infants and Young Children

- Language Sample Analysis
- Interviews and Observations with parents and primary caregivers
- Developmental Observation Checklist System

ASD and the School Age (6-12 years)

Early Developmental History

- Delayed language development **(C)**
- Co-morbidities **(D)**
- Associated with known medical or genetic conditions **(E)**

Behavioral Patterns and Sensory Sensitivities

- Repetitive movements **(B1)**
- A strong adherence to routines and Intense interests **(B2, B3, B4)**
- Rigidity in thinking **(B2)**
- Hyper- or hypo-reactivity to sensory input **(B4)**
- Unusual sensory interests **(B4)**

Signs of Social Communication Impairments in School Environments

- Struggles with conversational skills **(A1)**
- Difficulties with attention and joint attention skills**(A3)**
- Difficulty with social interactions . **(A2, C)**
- Difficulty making friends **(A3)**

Speech and Language Evaluation Tools for School-Age Children

Standardized Assessment Tools for School-Age Children

- Clinical Evaluation of Language Fundamentals, Fifth Edition (CELF-5; Wig et al., 2013)
- Peabody Picture Vocabulary Test, Fifth Edition (PPVT-5; Dunn, 2019)
- Test of Narrative Language, Second Edition (TNL-2; Gillam & Pearson, 2017)

Criterion-Referenced and Non-Standardized Assessment Tools for School-Age Children

- Dynamic Assessment of Language Learning
- Educator and Parent Questionnaires
- Play-Based Assessments

ASD During the Teen Age (13-17 years)

Social Communication Challenges

- Limited back-and-forth conversations **(A1)**
- Difficulty understanding social cues **(A1, A2)**
- Challenges in making and maintaining friendships **(A3)**

Repetitive and Restricted Behaviors

- Strong adherence to routines **(B2)**

- Intense interests or preoccupations **(B3)**

Sensory Sensitivities

- Over- or under-reactivity to sensory input **(B4)**
- Stereotypical, repetitive and restrictive behaviors **(B1)**
- Seeking sensory input **(B4)**

Impact on Academic and Daily Life

- Difficulty with executive functioning **(A1, A2, A3)**
- Social isolation **(D)**
- Heightened anxiety and stress **(D)**

Speech and Language Evaluation Tools for Teenagers

Standardized Assessment Tools for Teenagers

- Clinical Evaluation of Language Fundamentals, Fifth Edition (CELF-5; Wig et al., 2013)

- Test of Narrative Language, Second Edition (TNL-2; Gillam & Pearson, 2017)

- Social Language Development Test – Adolescent (SLDT-A; Bowers et al., , 2008)

Criterion-Referenced and Non-Standardized Assessment Tools for Teenagers

Language Sample Analysis

Observations of Peer Interactions

Self, Educator and Parent Questionnaires

ASD and Adulthood (18+ years)

Social Communication Challenges

- Difficulty with social interactions **(A1)**
- Maintaining relationships **(A3)**
- Literal interpretation of language **(A1, A2)**

Repetitive and Restricted Behaviors

- Repetitive routines and rituals **(B2)**
- Focused interests **(B3)**
- Repetitive movements **(B1)**

Sensory Sensitivities

- Sensitivities to sensory stimuli **(B4)**
- Limited exposure to social and otherwise learning activities **(B4)**

Impact on Daily Life and Employment

- Low employment rates **(D)**
- Difficulties with social communication in the workplace**(D)**
- Difficulty understanding and following workplace rules**(D)**

Speech and Language Evaluation Tools for Adults

Standardized Assessment Tools for Adults

- Assessment of Functional Living Skills (AFLS; Partington & Mueller, 2013)

- Communication Checklist - Adult (CC-A; Bishop, 2013)

Criterion-Referenced and Non-Standardized Assessment Tools for Adults

Narrative Analysis

Workplace Communication Observations

Functional Communication Assessments

Considerations for Assessment Design

Select your assessment tools **carefully**.

Stay up to date about the **physical and mental wellbeing** of the person and family members.

Refer as needed while providing adequate support.

Considerations for Selection of Interventions

Select interventions grounded in **rigorous research**.

Learn to **adapt and individualize** interventions for each individual.

Use a **variety** of interventions depending on individual need.

It was our pleasure!

TruePotentialCE

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