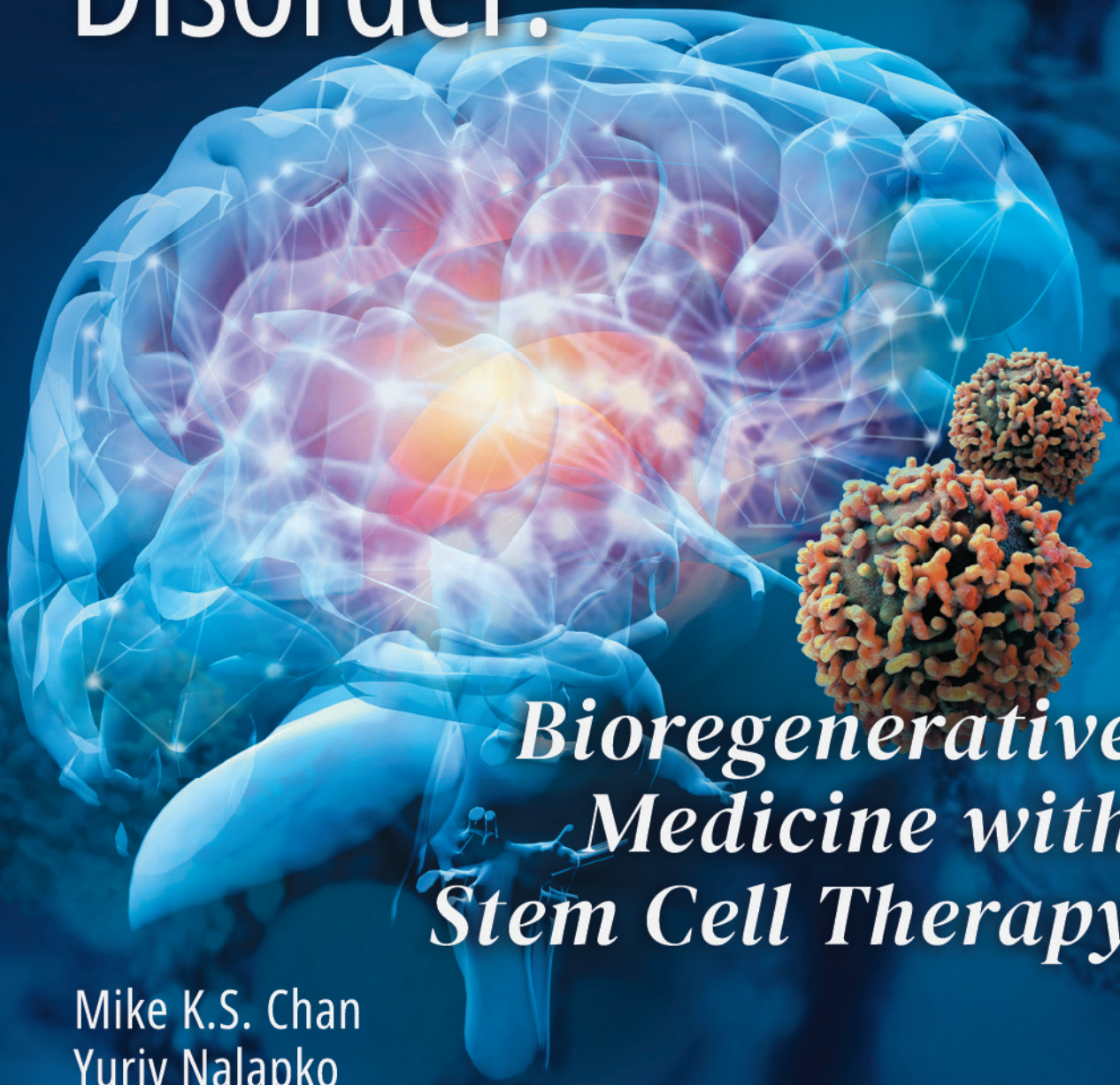
Autism Spectrum Disorder:



Bioregenerative Medicine with Stem Cell Therapy

Mike K.S. Chan
Yuriy Nalapko
Svetlana Yartseva
Michelle B.F. Wong

Autism Spectrum Disorder: Bioregenerative Medicine with Stem Cell Therapy is intended for a wide audience: parents of autistic children, regular and special education teachers, medical specialists, and for everybody who wants to learn about modern approaches to treat developmental diseases. This comprehensive monograph contains broad knowledge starting with a brief history of autism spectrum disorder, theories of occurrence and diagnosis, and reviewing modern behavioural, pharmacological, and bioregenerative therapeutic technologies.

Why is this book different? It is grounded in recent data related to new conceptualizations of the occurrence of autism spectrum disorder which consider autism a mitochondrial disease. Thus, the authors explain core concepts. What is a mitochondrion? How does its damage appear and correlate to the signs and symptoms of autism? What kind of bioregenerative therapies have great therapeutic potential? Almost all therapeutic methods for autism spectrum disorder are discussed from the point of view of the evidence based medicine.

Bioregenerative technology is based on the restoration of damaged subcellular structures like mitochondria and brain cell peptides. Bioregenerative antioxidant therapy, hyperbaric oxygen therapy, transcranial magnetic/direct current stimulation, and mitochondrial organelles replacement therapy are key directions in the treatment of autism spectrum disorder.

Finally, new technology that holds high potential for the restoration of brain function is discussed—bioregenerative stem cell therapy. Because of the strong correlation between the symptoms of autism and changes in the brain, physicians have great tools to regulate such clinical symptoms through stem cell therapy. Most importantly, the authors have personal experience using the stem cell therapy with autism spectrum disorder patients. All facts are strongly supported by published scientific reviews and trials.

Prof. Dato' Sri Dr. Mike K.S. Chan is an author, educator and pioneer of Organotherapy, Cellular Therapy and Cell Membrane Therapy in Europe and Asia since the early 1980s. He founded and funded one of the world's largest research on Bio-Molecular and Bio-Regenerative Medicine in Switzerland and Germany, with a global presence in almost eighty countries. He has conducted more than 1,000 lectures, seminars, and symposiums worldwide in the fields of organotherapy, cell therapies, age reversal, regenerative medicine, and stem-cell therapies.

Dr. Yuriy Nalapko, MD, PhD, is an academician and practical medical doctor in Intensive Care, Antiaging, and Bioregenerative Medicine. An area of scientific interest is longevity and how a cell functions with different diseases. He is the author of many handbooks, monographs, and more than 300 articles. Based on his clinical experience, he proposed new approaches to reverse degenerative diseases of the brain, liver, and endocrine organs in different disorders. In Metabolic Disorder Therapy, he combines preventive, pharmacological, and bioregenerative approaches as a state-of-the-art paradigm to correct structural and functional malfunctions.

Dr. Svetlana Yartseva, MD, PhD, is a certified Endocrinologist and Internal Diseases Practitioner. A member of several international associations on the study of diabetes; the European Thyroid Association; and the European Association of Preventive, Regenerative, and Antiaging Medicine; she is a Consultant at numerous private clinics. Her main concern is treatment of patients with obesity, metabolic syndrome, and diabetes mellitus through modern pharmacological and non-pharmacological methods.

Prof. Dato' Sri Dr. Michelle B.F. Wong, ND, PhD is a scientist, researcher and educator actively involved in research and development (R&D) work on stem cells focusing on Fetal Precursor Stem-Cell Therapy. She is the Co-Founder/Senior Technical Advisor and Researcher in FCTI Inc., specialising in Stem cell Research and Bio-Regenerative Medicine for more than three decades. A prominent speaker, Michelle Wong presents at educational programs and global conferences on stem cells, cell therapy and biological medicine for physicians and healthcare practitioners worldwide.

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First and foremost, this book
is dedicated to families with autistic kids,
who demonstrate heroic patience and perseverance,
and whose strong spirit gives us the strength
and confidence to successfully
treat our little patients.

Authors

TABLE OF CONTENTS

ABBREVIATIONS

5-HT	– 5-hydroxytryptamine, serotonin
AA	– arachidonic acid
ABA	– applied behavior analysis
ABC	– autism behavior checklist
ACTH	– adrenocorticotropin hormone
ADHD	– attention deficit/hyperactivity disorder
ADI-R	– autism diagnosis interview – revised
ADOS	– autism diagnostic observation schedule
AE	– adverse effect
ALA	– α-linolenic acid
AMPAR	– α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid
AMT	– artificial mitochondria transfer
AS	– Asperger syndrome

ASD – autism spectrum disorder
ASIEP – autism screening instrument for educational planning
ATP – adenosine triphosphate
BBB – blood-brain barrier
CARS – childhood autism rating scale
CAST – childhood Asperger syndrome test
CDC – Centers for disease control and prevention
CNS – central nervous system
CNV – copy number variation
CSF – cerebrospinal fluid
DCD – developmental coordination disorder
DHA – docosahexaenoic acid
DIR – developmental individual difference
DISCO – diagnostic interview for social and communication disorders
DLD – developmental language disorder
DLPFC – dorsolateral prefrontal cortex
DMSA – 2,3-dimercaptosuccinic acid
DNA – deoxyribonucleic acid
DSM – Diagnostic and statistical manual of mental disorders
DTI – diffusion tensor imaging
EAAT – equine-assisted activities and therapies
EEG – electroencephalography
ELISA – enzyme-linked immunosorbent assay
EPA – eicosapentaenoic acid
ESDM – early start Denver model
ETC – electron transport chain
EV – extracellular vesicle
FDA – Food and Drug Administration
fMRI – functional magnetic resonance imaging

FSC – fetal stem cell
GABA – gamma-aminobutyric acid
GARS – Gilliam autism rating scale
GcMAF – granulocyte macrophage activating factor
GSH – glutathione
GSSG – glutathione disulfide
GWAS – genome-wide association study
HBOT – hyperbaric oxygen treatment
HMD – heavy metals detox
HPLC – high-performance liquid chromatography
ICD – International classification of diseases
ID – intellectual disorder
IFN-gamma – interferon gamma
IL – interleukin
iPSC – induced pluripotent stem cell
IQ – intelligence quotient
MN – mirror neuron
MO – mitochondrial organelles, Mito Organelles
MRI – magnetic resonance imaging
MSC – mesenchymal stem cell
mtDNA – mitochondrial DNA
NAA – N-acetyl aspartate
NIBS – noninvasive brain stimulation
NMDA – N-methyl-D-aspartate
NMDAR – N-methyl-D-aspartate receptor
NOP – nanomized organopeptide
NSC – neural stem cell
PBS – Positive Behavior Support
PDD – pervasive developmental disorders

PDD-NOS – pervasive developmental disorder not otherwise specified
PECS – picture exchange communication system
PLA 2 – phospholipase A 2
POMC – proopiomelanocortin
PUFA – polyunsaturated fatty acid
RAADS-R – Ritvo autism Asperger diagnostic scale – revised
RDI – relationship developmental intervention
ROS – reactive oxygen species
RRB – restricted, repetitive behavior
RT – responsive teaching
rTMS – repetitive TMS
SCQ – social communication questionnaire
SNV – single nucleotide variation
SPCD – social pragmatic communication disorder
SSRI – selective serotonin reuptake inhibitor
STAT – screening tool for autism in toddlers and young children
tDCS – transcranial direct current stimulation
TEACCH – treatment and education of autistic and related communication handicapped children
THR – therapeutic horseback riding
TMS – transcranial magnetic stimulation
TNF-α – tumor necrosis factor-α
TNT – tunneling nanotube
ToM – theory of mind
WHO – World Health Organization

PREFACE

Autism spectrum disorder (ASD) is a widespread developmental disorder. This condition is characterized by three core deficits: impaired communication, impaired reciprocal social interaction and restricted, repetitive, and stereotyped behavioral patterns. The presentation of these impairments varies in range and severity and often changes with the acquisition of other developmental skills.

The occurrence of ASD is attributed to many theories and a unified scientific understanding is lacking. There are many studies mentioning plenty of risk factors for ASD. Presently, the concept includes genetically and clinically heterogeneous psychic disorders united by impaired social interaction. This is a group of heterogeneous complex disorders of nervous system development with the key damage occurring to the brain mitochondria.

Chapter One describes the definition of ASD, its theories and risk factors. A clear understanding of this knowledge will help us arrive at correct diagnoses of ASD and to learn modern medical and biological techniques for its treatment.

In Chapter Two, key diagnostic features and ASD classifications are reported. Here we review some of the neuroanatomical alterations reported in ASD, with a particular focus on well-validated findings of the different signs and symptoms. We ask what these observations tell us about normal and abnormal brain development. Here, we advocate the concept that mitochondria are an integral part of diverse cellular functions and are susceptible to many insults in ASD. Besides brain lesions, other organs and systems may be damaged in ASD, causing co-morbid disorders.

Finally, Chapter Three is devoted to the different approaches in treating patients with ASD including behavioral, pharmacological, and bioregenerative strategies. Special attention is given to the promise of modern bioregenerative mitochondrial replacement, neuropeptide restoration, and stem cell therapy. Medicinal and natural antioxidants, as well as detoxification, are also described in this section.

This book is based on the reviews of recent publications, scientific data, and our personal experience in treating patients with ASDs. We advocate for the use of integrative pharmacological and bioregenerative technology. The use of bioregenerative therapies is becoming increasingly popular due to the minimally invasive nature of these procedures and their high efficacy. Stem cell therapy and cell peptide prescription are gaining interest due to its ability to stimulate and accelerate tissue and organ healing processes. We discuss techniques for maintaining the structural integrity and functional capability of brain tissue in ASD.

The authors hope that this comprehensive analysis of the occurrence, development, and treatment of ASD in different age groups will foster the development of the most effective and timely treatments for these patients. This review will spur interest in the study of the bioregenerative medicine in autism. The ultimate goals of this work are to understand the role of mitochondrial damage, its bioregenerative restoration and stem cell replacement for this disorder. As well as in our previous books, we aim to open the new horizons of the bioregenerative medicine in non-communicable diseases.

Authors

INTRODUCTION

Autism spectrum disorder (ASD) is a prominent developmental disorder. This condition is characterized by three core deficits: impaired communication, impaired reciprocal social interaction, and restricted, repetitive, and stereotyped patterns of behaviors or interests. The presentation of these impairments is variable in range and severity and often changes with the acquisition of other developmental skills. The term “spectrum” refers to the wide range of clinical symptoms and levels of impairment that can occur in ASD individuals.

ASD occurs across all racial, ethnic, and socioeconomic groups and is highly prevalent in all populations. It affects tens of millions of individuals worldwide and treatment and support costs are high. In the United States, the incidence of ASD is 1 in 68 children. The prevalence of ASD appears to be on the rise (a 10-fold increase over 40 years) and is partially explained by improved diagnosis.

Given this extreme etiological diversity, it is reasonable to hypothesize that a brain perturbation can precipitate the behavioral hallmarks of ASD. There are

options for therapeutic approaches that might enhance efficacy in addressing core symptoms. One possibility is to focus on mitochondrial function, which is integral to many disease pathways. In addition to its well-known role as the “powerhouse of the cell,” as they produce the bulk of the cellular energy, mitochondria are critically involved in cellular metabolism, intracellular calcium signaling, generation of reactive oxygen species, and apoptosis in both innate and adaptive immunity. Mitochondria carry out the synthesis of glycine, the ligand of glycine receptors. These receptors mediate inhibitory neurotransmission in the matured central nervous system (CNS). However, they are highly expressed in the embryonic brain and mediate excitatory neurotransmission and are therefore believed to promote cortical interneuron migration and the generation of excitatory neurons.

Mitochondria play a vital role in CNS. The brain has high energy demands, consuming approximately 20% of calories while accounting for only 2% of total body weight. Additionally, the brain demands a significant amount of adenosine triphosphate (ATP) to maintain ionic gradients essential for neurotransmission and plasticity. In addition, mitochondria are involved in the proliferation, differentiation and maturation of neural stem cells, formation of dendritic processes, developmental and synaptic plasticity and cell survival and death. Thus, it is not surprising that multiple lines of evidence in both human and animal models support the role of mitochondrial dysfunction in the etiology of ASD.

These mitochondrial defects are found in 5% of ASD patients, which is 500 times higher than the rate found in the general population (≈0.01%). In addition, several genes known to regulate mitochondrial function have been clearly identified as autism-risk genes (Cheng et al., 2017). Also, it has a high prevalence of abnormal metabolic biomarkers, suggesting that as many as 30% of children with ASD may experience metabolic abnormalities. However, metabolic and mitochondrial dysfunction may not exist in all patients with ASD.

ASD has broad and heterogeneous manifestations, as it is associated with many possible etiological factors (both genetic and environmental). In most cases, ASD etiology remains undefined.

Beyond the core behavioral symptoms, ASD is increasingly shown to affect the gastrointestinal, immune, hepatic, and endocrine systems. Typical co-morbidities include neurologic, psychiatric, and physical conditions. These neurologic comorbidities include epilepsy, sleep disorders, sensory abnormalities, and delays and/or deficits in motor function. Many psychiatric conditions like depression, anxiety, and attention deficit/hyperactivity disorder and somatic issues such as chronic gastrointestinal disturbance are also associated with ASD. The rate of non-ASD developmental diagnoses is around 85%.

The hope is that early diagnosis and correct target therapy (pharmacological, behavioral as well as bioregenerative) will improve overall quality of life of such individuals and reduce the severity of ASD symptoms.

Chapter 1.

BASICS OF AUTISM SPECTRUM DISORDER

Sometimes it is the people no one can imagine anything of who do the things no one can imagine.

—ALAN TURING,

creator of the first computer used to break codes during WW II

1.1. BRIEF HISTORY OF ASD

In 1943, the American psychiatrist Leo Kanner used the term “early infantile autism” to describe children who lacked interest in other people. This infantile autism was identified as an “inborn autistic disturbance of affective contact.”

In 1944, an Austrian pediatrician, Hans Asperger, independently described another group of children with similar behaviors, but milder severity and higher intellectual abilities. Since then, his name has become assigned to a higher functioning form of autism, Asperger syndrome (AS). Asperger syndrome was described as the behavioral characterization of individuals with difficulties in communication and social interaction.

Since then, many researchers have tried to identify the definition and diagnostic criteria for autism. In 1980, the American Psychiatric Association recognized autism as a distinct category in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-3) and introduced it as infantile autism.

The term “autism spectrum” was introduced by the English psychiatrist Lorna Wing in the 1970s (Wing, 1997). The term implied the existence of an array of diseases accompanied by the disorders in communication, social interaction, social understanding, and imagination. In 1981, Wing restarted her research and formally named Asperger syndrome. A few years later, in 1989, the first diagnostic criteria for Asperger syndrome were proposed, and the 10th Revision of the International Classification of Diseases (ICD-10) was the first major classification system that recognized the disease (1993).

Finally, in 1994, Asperger syndrome was introduced in DSM-IV, as a specific entity along with autistic disorder, both included within the classification of pervasive developmental disorders (PDD).

Although Asperger syndrome was introduced as a discrete diagnostic category in the DSM-IV, almost twenty years later, its diagnostic label has been removed in the DSM-5 where it was reassigned to a more general category of ASD.

During this period, researchers were focused on the diagnostic measures of Asperger syndrome and its differences with high-functioning autism. In 2013, the DSM-5 removed the diagnostic category of Asperger syndrome, and the World Health Organization (WHO) followed a similar approach in ICD-11, which comes into effect in 2022.

The definition and diagnosis of these disorders has been broadened over the years to include milder forms of autism (see *Definitions and classifications*).

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