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Parkinson's Disease

Dr. Zoltan Mari, MD

May 14, 2024

SEMINAR INSTRUCTOR

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Cleveland Clinic Lou Ruvo Center for Brain Health

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Parkinson's Disease Overview & Update

Zoltan Mari, MD

KEEP MEMORY ALIVE

Supporting the Mission of the Cleveland Clinic Lou Ruvo Center for Brain Health

Cleveland Clinic

Disclosures

- Dr. Mari is a full-time staff and Ruvo Family (Endowed) Chair at Cleveland Clinic (CC) and is representing his own opinions and NOT that of CC
- Dr. Mari received (institutional) research support from: the National Institutes of Health, Michael J. Fox Foundation, Parkinson's Foundation, AbbVie, Eli Lilly, Cerevel
- Dr. Mari has served as a paid consultant for GB Sciences, Sanofi Genzyme, NeuroReserve, Neuonos, Sensory Cloud, Biogen, Kyowa Kirin, nQ Medical Inc, Supernus, ACADIA, and Global Kinetics Corporation
- Dr. Mari holds shares of Neunos, Inc. (Black Rock NeuroTechnology), Sensory Could, NeuroReserve, GB Sciences, and D&D PharmaTech (last 2 public)
- Dr. Mari is the immediate past Chair of the Telemedicine Study Group at the Movement Disorder Society (MDS), Chair of the Motor Features Working Group at the Parkinson Study Group (PSG), member of the Congress Site Selection Committee & Website Committee for the International Association of Parkinsonism & Related Disorders (IAPRD), and Associate Editor for the Parkinsonism & Related Disorders journal
- Dr. Mari is founder and CMO for both Neuraly, Inc. & Z NeuroSciences, LLC

OUTLINE

- What is Parkinson's Disease?
- Statistics & Public Health Impact of Parkinson's Disease
- Diagnosis, Causes, and Course of Parkinson's Disease
- Management of Parkinson's Disease
- The Importance of Multidisciplinary and Integrated Care Models

What is Parkinson's Disease?

- Parkinson's Disease is a progressive neurodegenerative "movement disorder", which is a clinico-pathological entity (disease) versus a group of specific diseases that share certain features (syndrome), such as "parkinsonism"
- Originally described (1817) as a disorder with only motor symptoms, it is now recognized as a "multisystem" disorder with manifestations in many domains, including motor and non-motor (such as neuropsychiatric, gastrointestinal, autonomic), with each PD patient's journey featuring a distinctly unique combination of many possible symptoms and subjective disabilities and challenges
- Traditionally diagnosed using clinical diagnostic criteria, with dopamine imaging as an optional "confirmatory" test, recently a synuclein-based assay has been suggested as a far more accurate diagnostic test, which is expected to fully replace the use of our currently "god standard" clinical diagnostic criteria

Parkinson's Disease Introduction: U.S. Statistics

- Prevalence:
  - ~ 1 million people (to reach 1.2M by 2030)
  - ~10M worldwide
- 1% of the population over 60 (likely more)
- Incidence per year: ~ 90,000 new cases
- Mean age of onset: 60 years
- US annual cost $52B (2017) – more since
- The fastest growing neurological disease
- Men are 1.5x more likely to have it

https://www.nature.com/articles/s41531-020-0117-1

Common Symptoms & Findings

- 4 cardinal features (motor):
  - **T**remor (rest)
  - **R**igidity
  - **A**kinesia (or bradykinesia)
  - **P**ostural Instability

- Non-motor symptoms
  - Speech abnormalities
  - Autonomic, sleep impairment
  - Cognitive, anxiety, depression
  - Pain symptoms, muscle mass↓
  - Anosmia

Illustration of the Parkinson disease by Sir William Richard Gowers from A Manual of Diseases of the System in 1886 (WikiMedia CommonNervouss)

Common Symptoms & Findings

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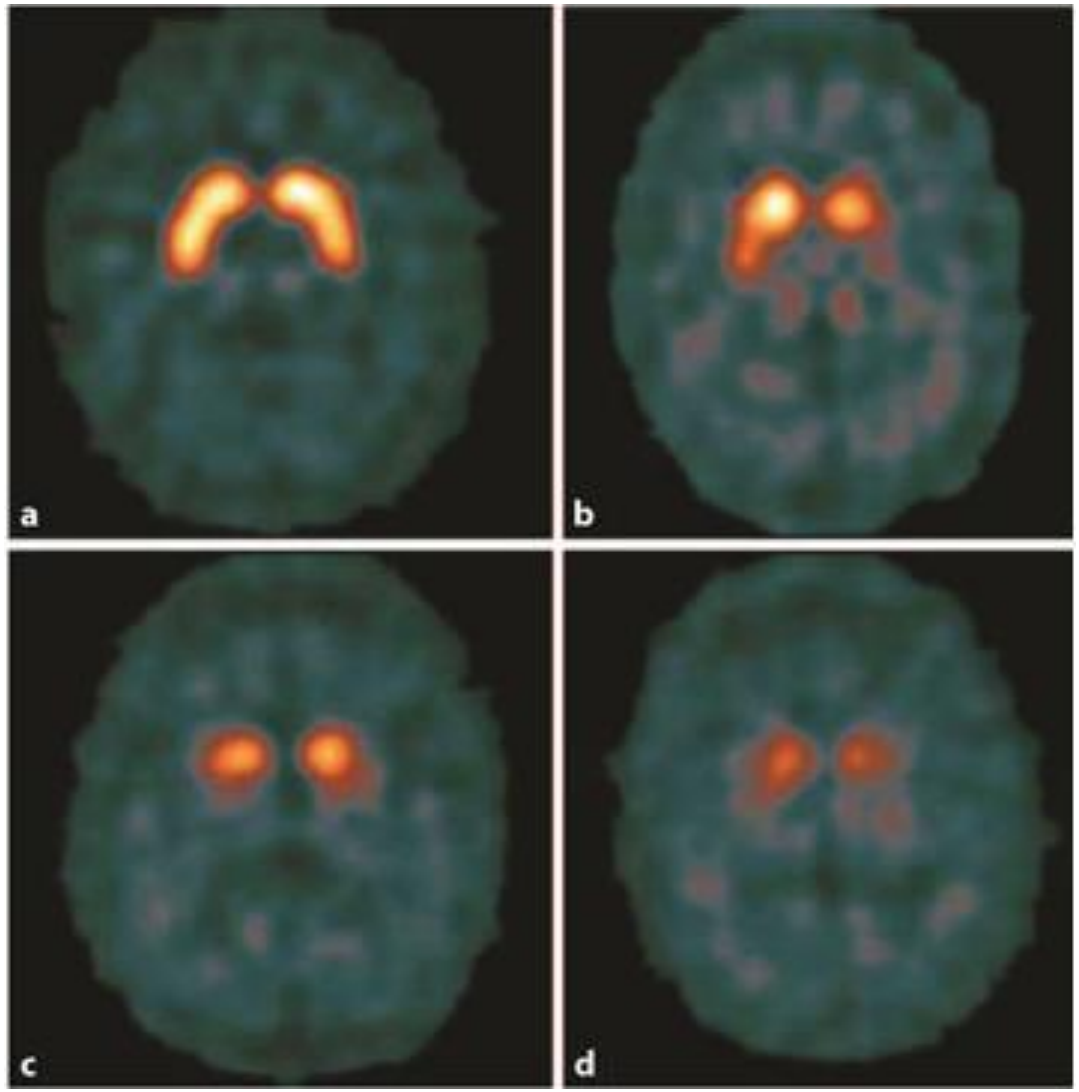
Typical Parkinson patient: video includes pre-DBS levodopa OFF & ON, as well as post-DBS clips

Non-Motor Features of PD

- Mentation, behavior & mood
  - Depression (up to 50%)
  - Dementia (bradyphrenia first, up to 40%)
  - Anxiety, panic attacks
- Autonomic
  - Orthostatic hypotension (from both PD + meds)
  - GI: gastroparesis (dose failure!), constipation
  - Other: skin (seborrhoea), sexual dysfunction

How do we make the diagnosis of PD?

- Neurological examination looking for the cardinal features (supplemented by observing a good response to dopamine replacement): UK BB – more recently MDS criteria
- Imaging can help confirm presynaptic dopaminergic deficits, either fluoro-DOPA PET scanning looking for dopamine storage or SPECT or PET scanning for the dopamine transporter. DaTscan is now approved in the US.
- Also approved is Syn-One Test



Normal and abnormal DaTscan SPECT images. a Normal DaTscan SPECT image. b–d Abnormal DaTscan images fall into at least one of the following three categories (all are considered abnormal): abnormal DaTscan SPECT image type 1(b), abnormal DaTscan SPECT image type 2 (c) or abnormal DaTscan SPECT image type 3 (d).

Randomized Controlled Trial > Neurodegener Dis. 2013;11(1):22-32. doi: 10.1159/000337351. Epub 2012 May 8.

Changes in clinical management and diagnosis following DaTscan SPECT imaging in patients with clinically uncertain parkinsonian syndromes: a 12-week follow-up study

Andreas Kupsch [1], Nin Bajaj, Frederick Weiland, Antonio Tartaglione, Susanne Klutmann, Ronald Copp, Paul Sherwin, Ann Tate, Igor D Grachev

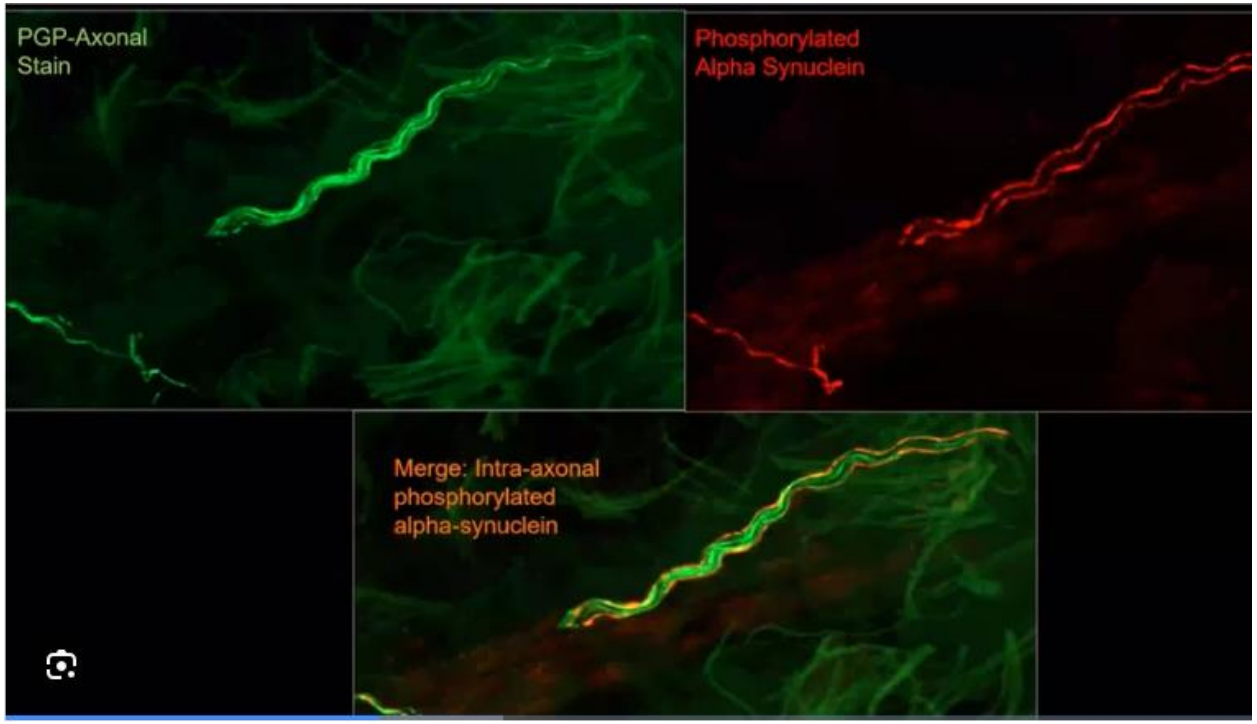
Affiliations + expand

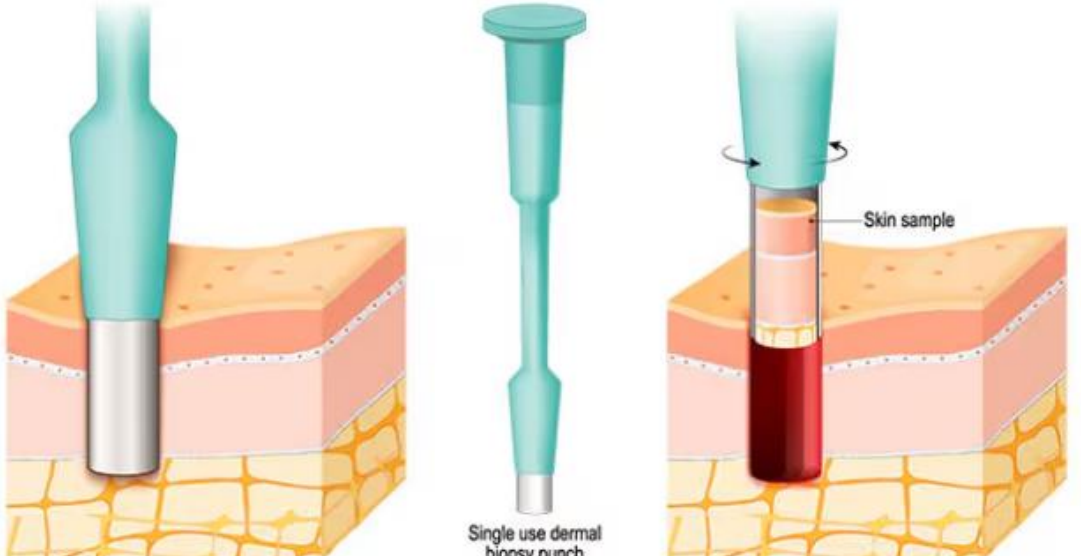
PMID: 22571977 DOI: 10.1159/000337351

Skin Biopsy

A skin biopsy test called the Syn-One Test can detect Parkinson's disease and related disorders by testing for the presence of P-SYN, an abnormal form of alpha-synuclein. P-SYN is present in the skin even before symptoms appear, so skin biopsies could improve early detection. A March 2024 study published in the Journal of the American Medical Association found that 93% of people with clinically confirmed Parkinson's disease tested positive for P-SYN in skin biopsies taken from the neck, knee, and ankle.

https://www.parkinsonsecrets.com/blog/should-i-get-a-skin-biopsy-or-dat-scan-for-parkinsons-disease





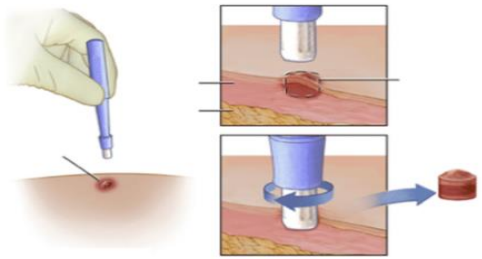
June 14, 2023

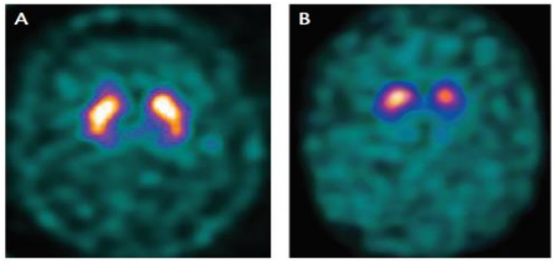
Do I Need A Skin Biopsy Or DaTscan For My Parkinson's Disease?

By Michael S Okun MD

A lot of buzz has been generated as a result of the emergence of skin biopsies and DaTscans as potential diagnostic tests for Parkinson's disease and for 'parkinsonisms.' The promise and the hope of these tests has been accompanied by 'confusion' as to 'what they are' and 'when to apply them.' In this month's parkinsonsecrets.com blog, I will review the **key information** persons with Parkinson's disease, family members and clinicians should all 'know' about these two diagnostic tests. Additionally, I will touch on the roles that these 'tests' may play in the future development of new therapies for Parkinson's disease.

SPOILER ALERT: If you already have a diagnosis of levodopa responsive Parkinson's disease, you may not require a skin biopsy or a DaTscan!





CLINICAL PROGRESSION OF PD

***** The pace of disease in *every* patient is *different***

PRE-CLINICAL STAGE

- REM Behavior Disorder
- Loss of smell
- Constipation, ED,
- low BP, urinary freq
- Biomarkers: Smell test, MIBG scan, SPECT, PET

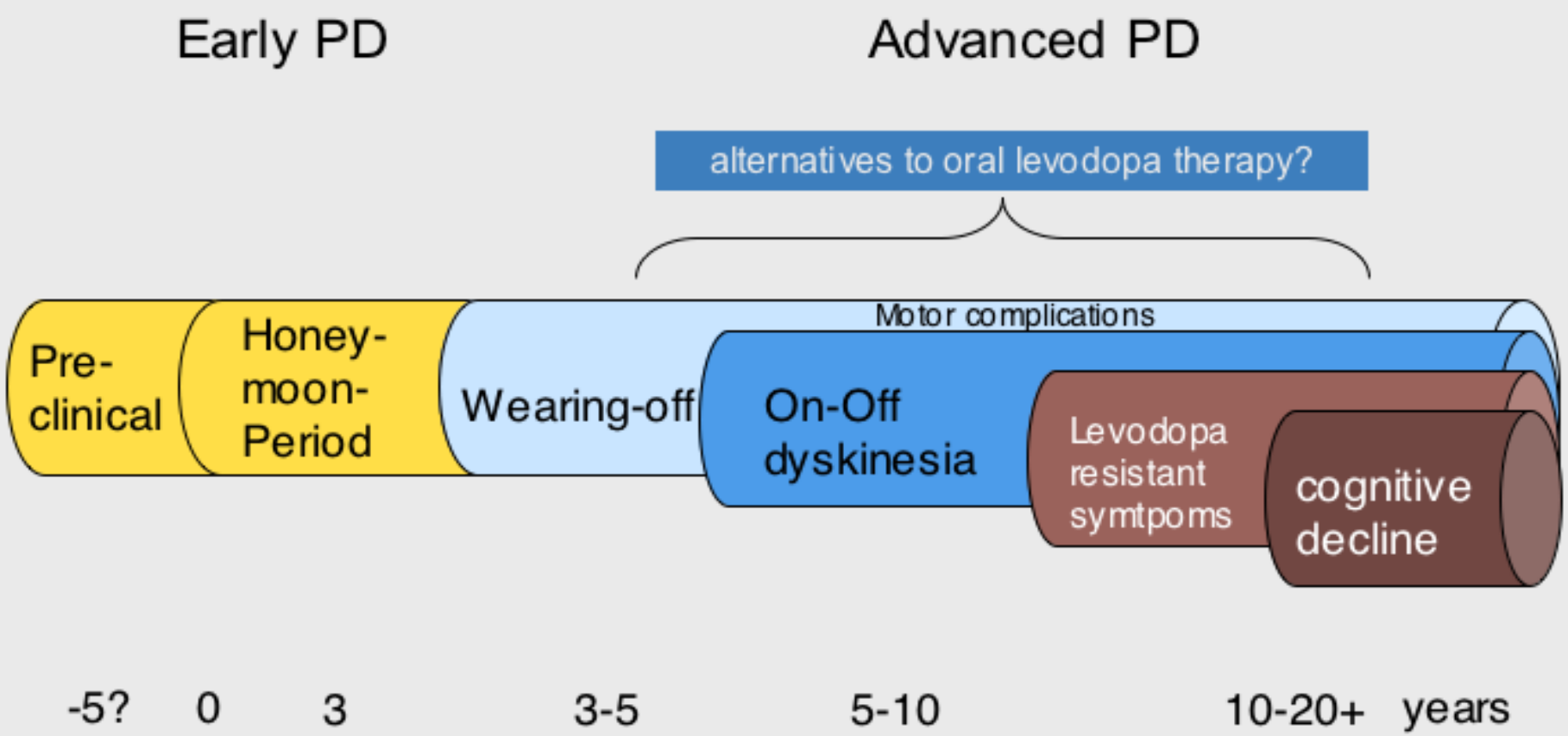
EARLY STAGE

- One sided symptoms
- Slowness
- Masked face
- (Stiffness) Rigidity
- Shaking (Resting tremor)
- Dystonia
- Shuffling gait

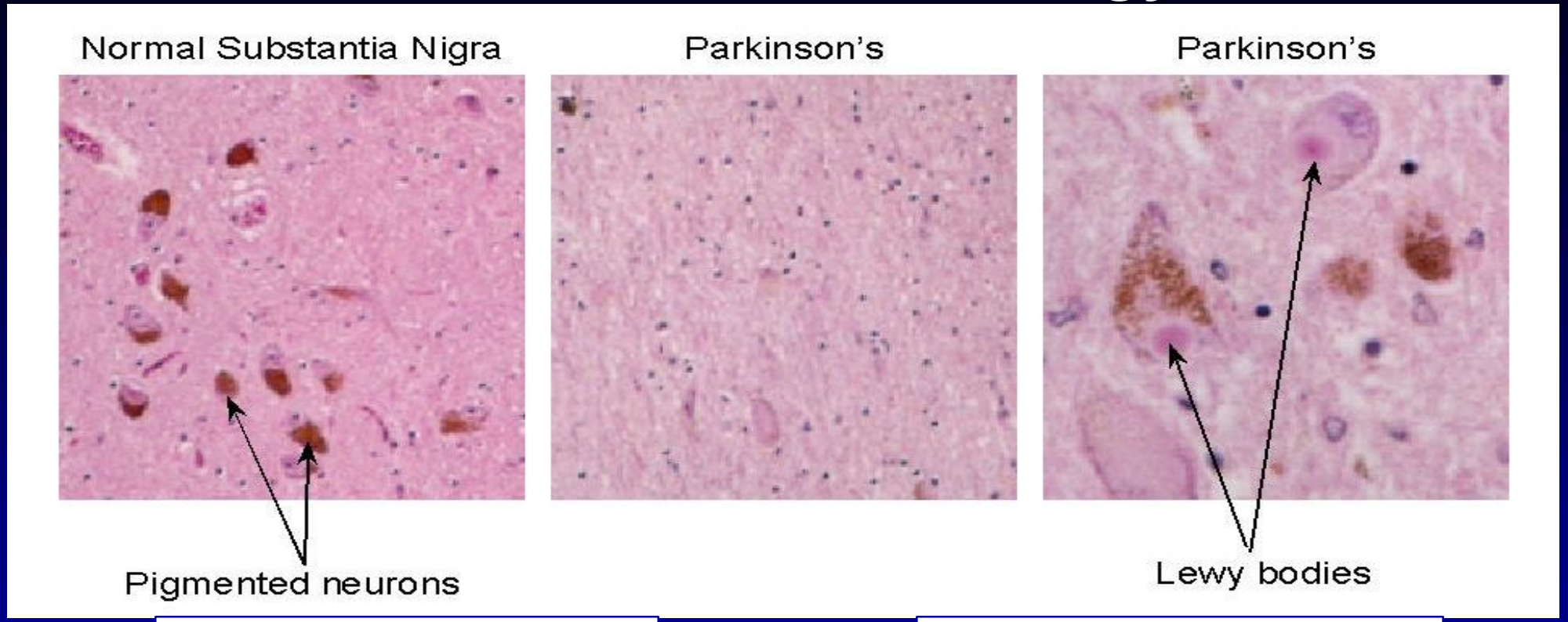
LATE STAGE

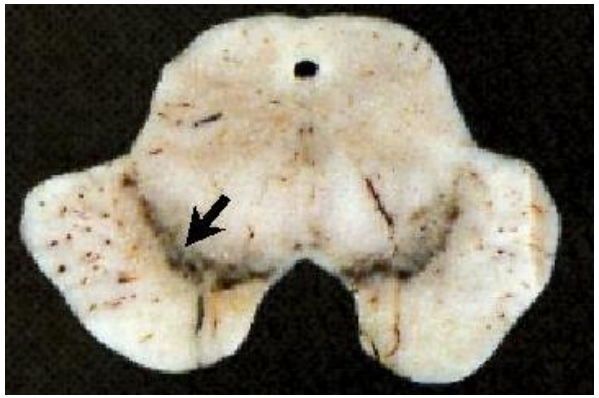
- Symptoms on both side
- Involuntary dancing movements
- Motor (On-off) fluctuations
- Speech problems
- Some difficulty in swallowing
- Poor memory (dementia)
- Hallucinations, confusion
- Falls
- Freezing of gait

Progression of Parkinson‘s disease

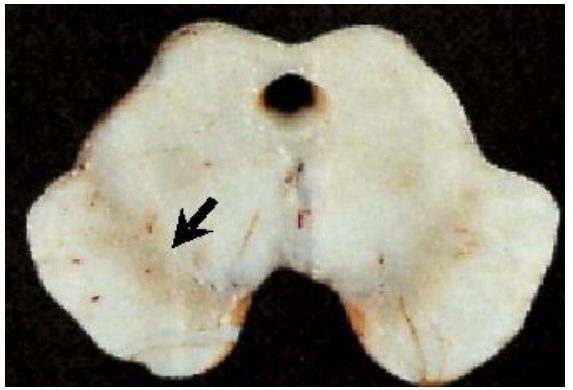


PD Basics: Pathology



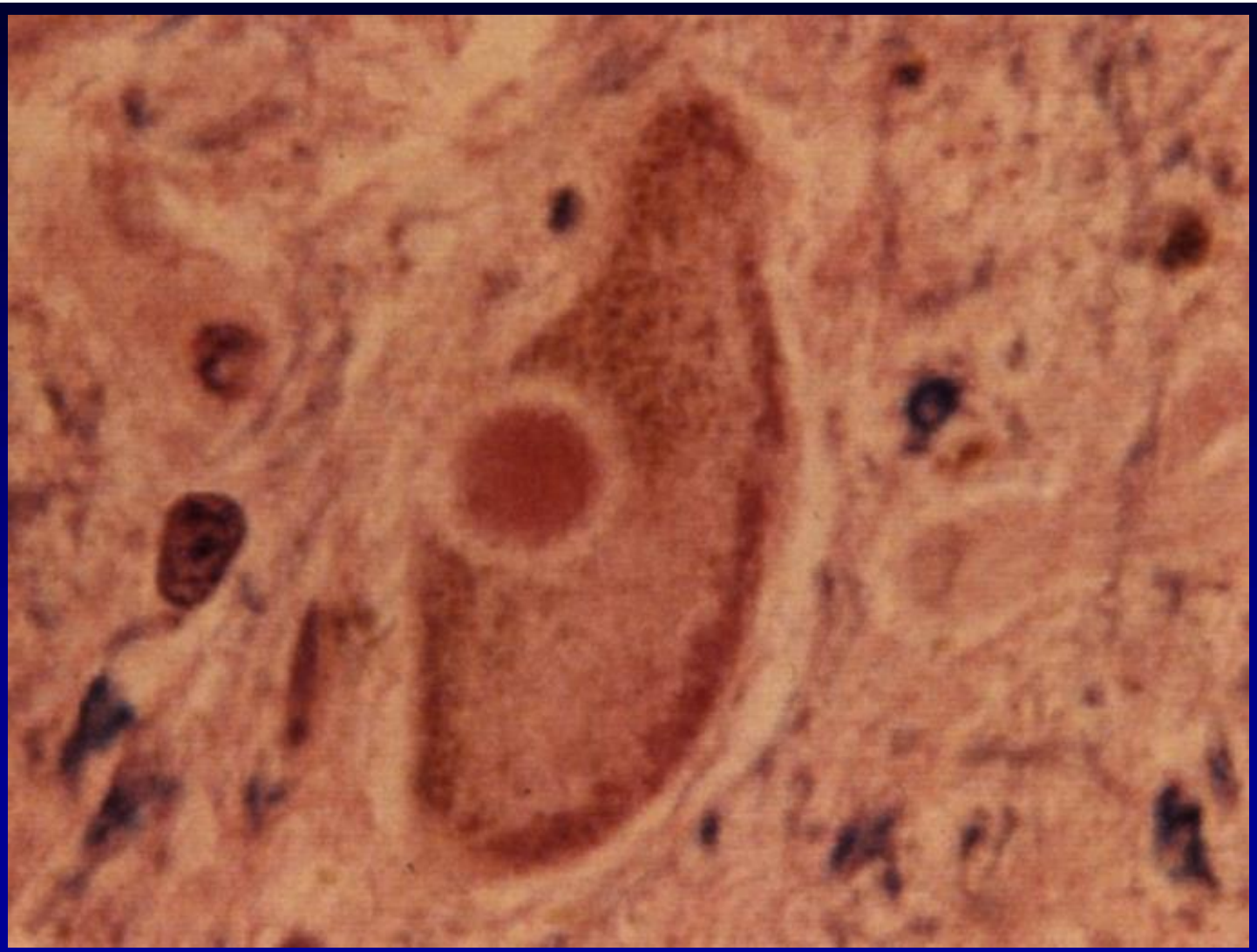


Normal: Nigra (pigment)



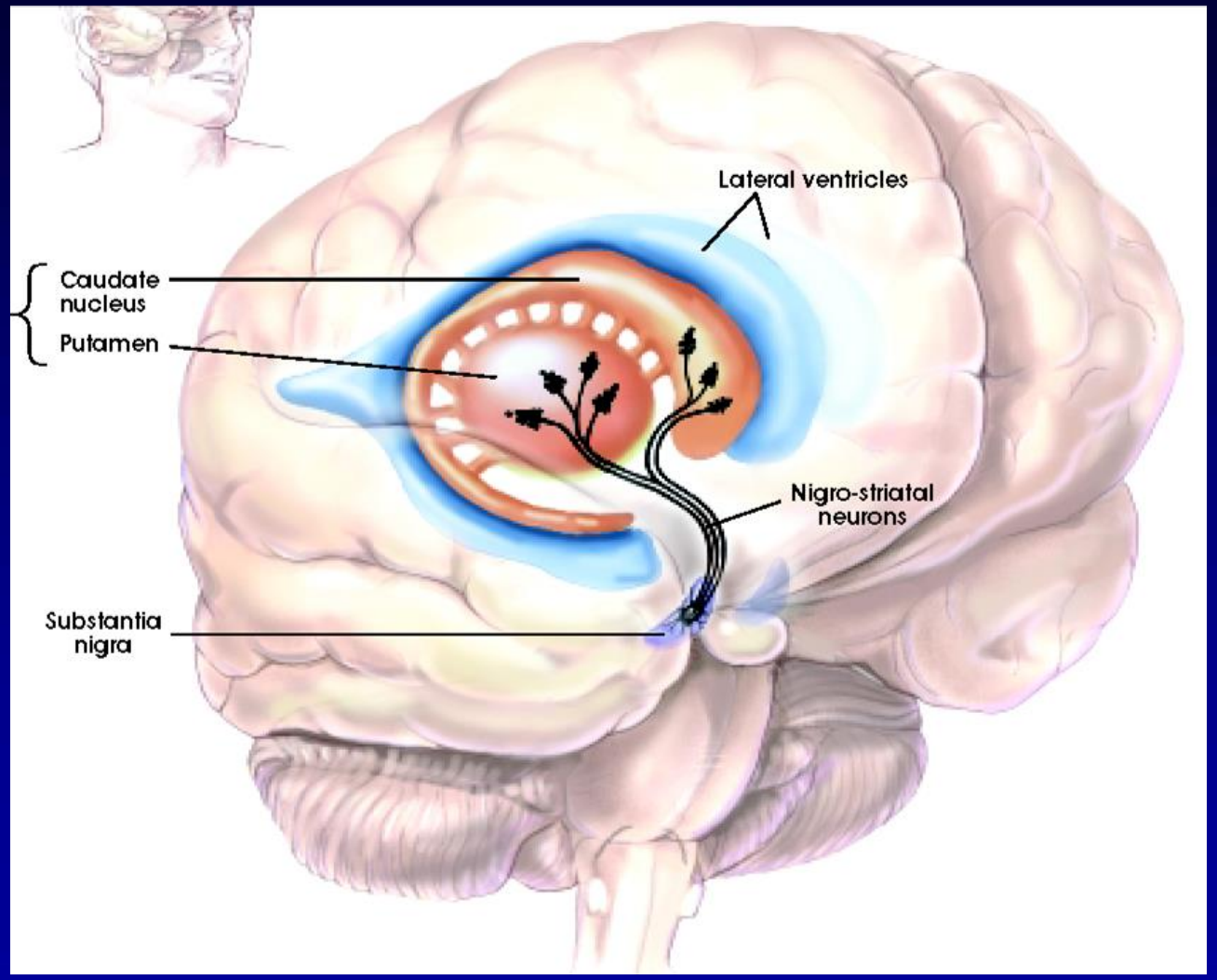
Parkinson: Nigra (lost pigment)

Cellular Pathology of Parkinson's disease: the Lewy Body

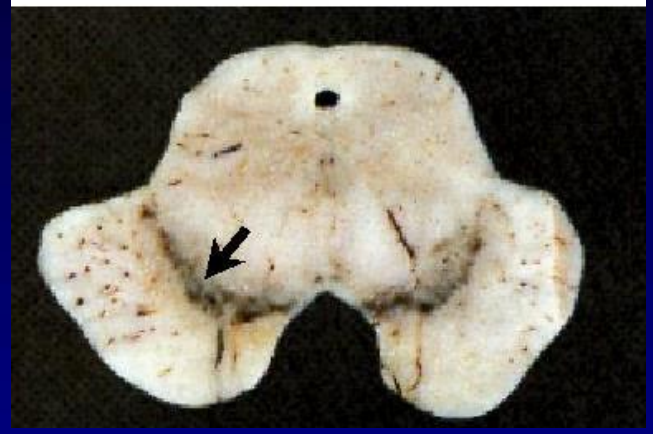


Lang & Lozano 1998

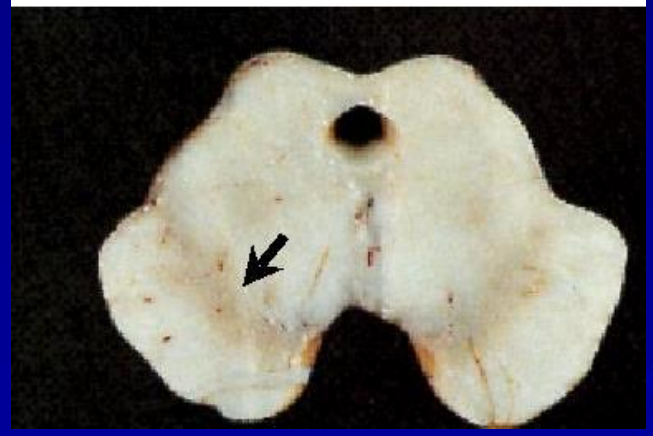
Gross Pathology of Parkinson's Disease



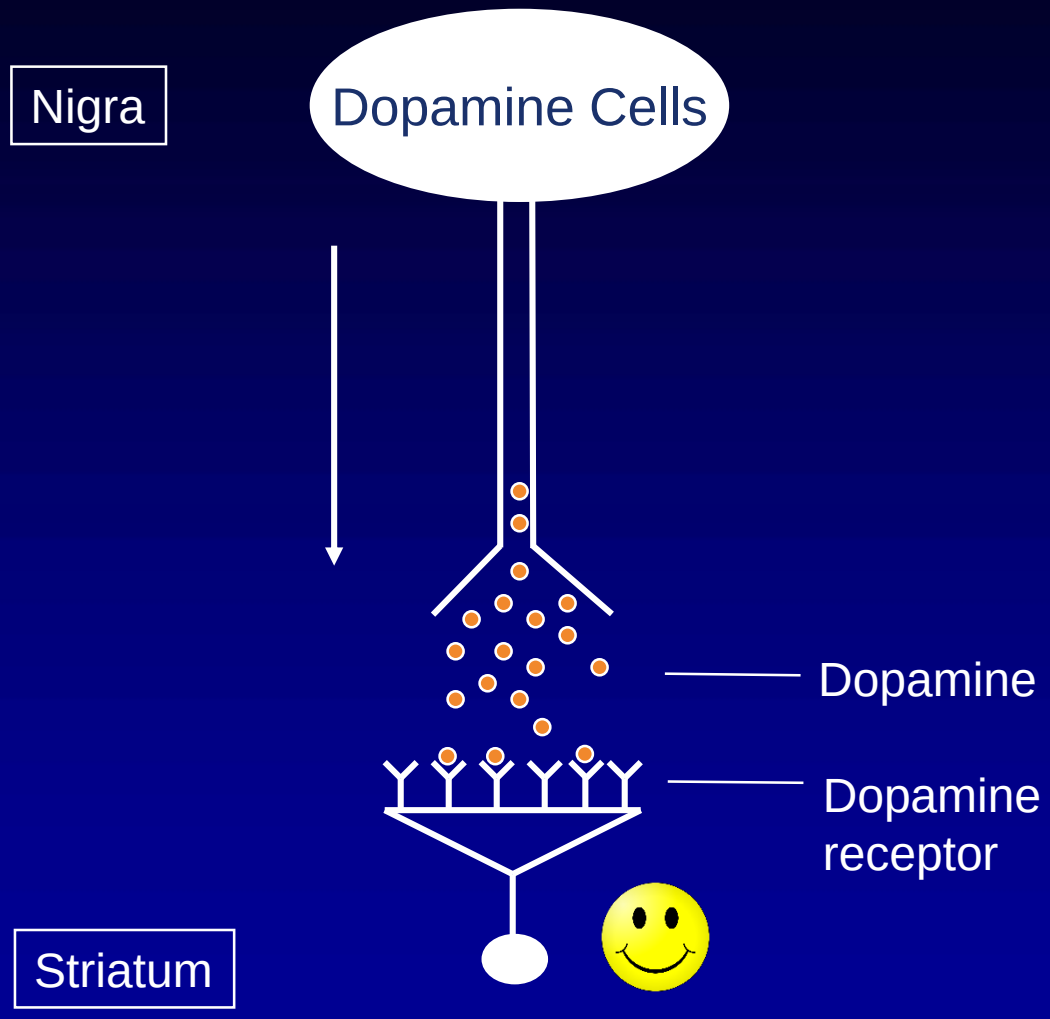
Normal



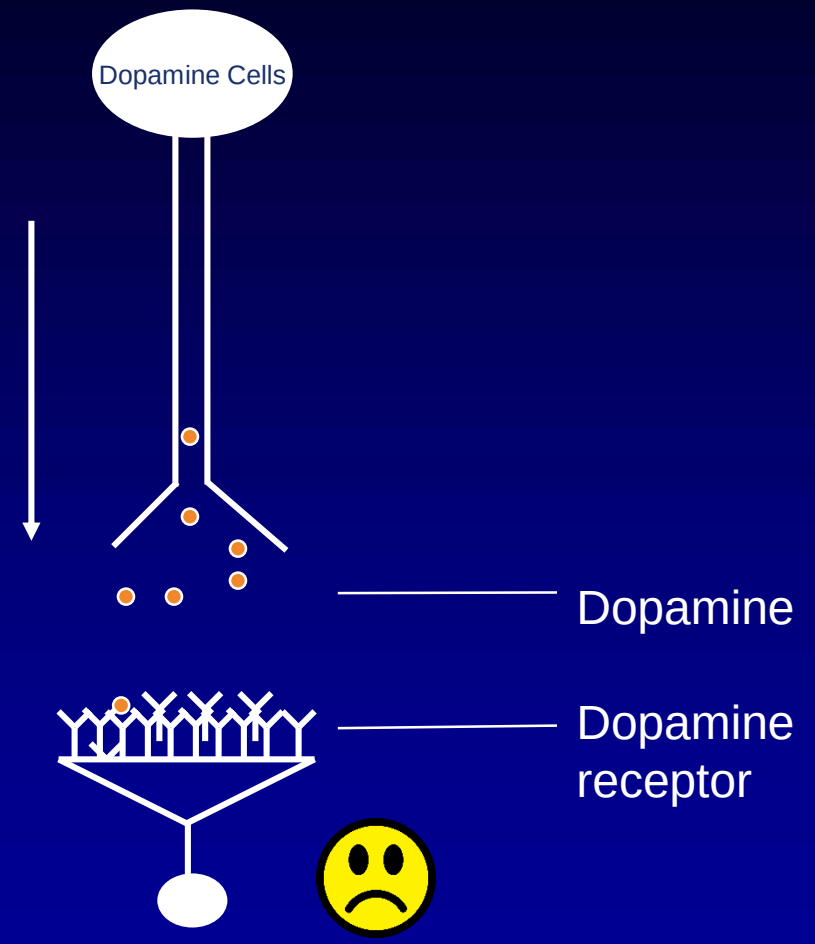
Parkinson's



Neuropharmacology of PD

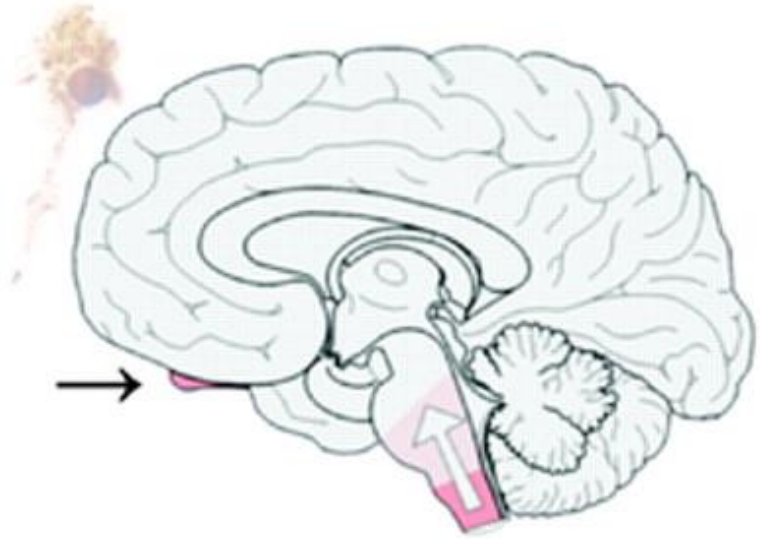


Normal State

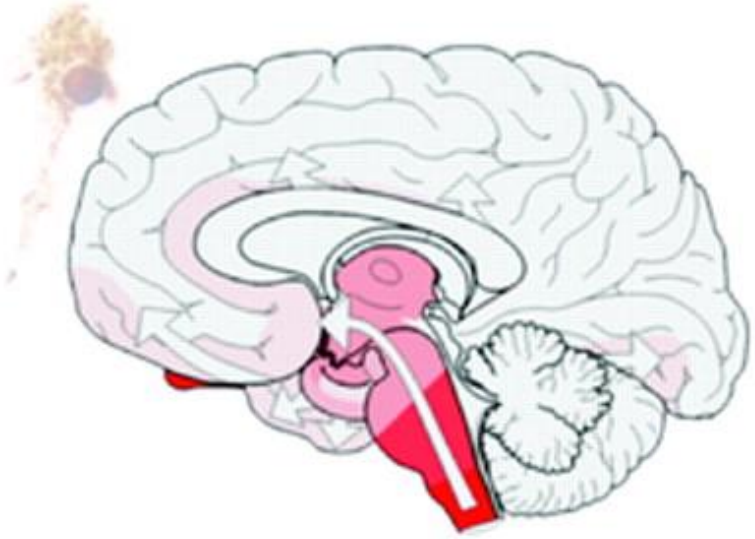


Parkinson's Disease

Braak Staging of PD



Braak Parkinson's disease stages 1 & 2
PRECLINICAL



Braak Parkinson's disease stages 3 & 4
CLINICAL PARKINSON'S DISEASE

Braak Parkinson's disease stages 5 & 6
COGNITIVE IMPAIRMENT

Olanow, C. W. et al. Neurology 2009;72:S1-S136

AMERICAN ACADEMY OF NEUROLOGY

What Causes Parkinson's Disease?

*Genetics *Age *Environment

Pathogenesis

Oxidative Stress | Mitochondria Dysfunction | Inflammation | Apoptosis Cascade | Protein Aggregation

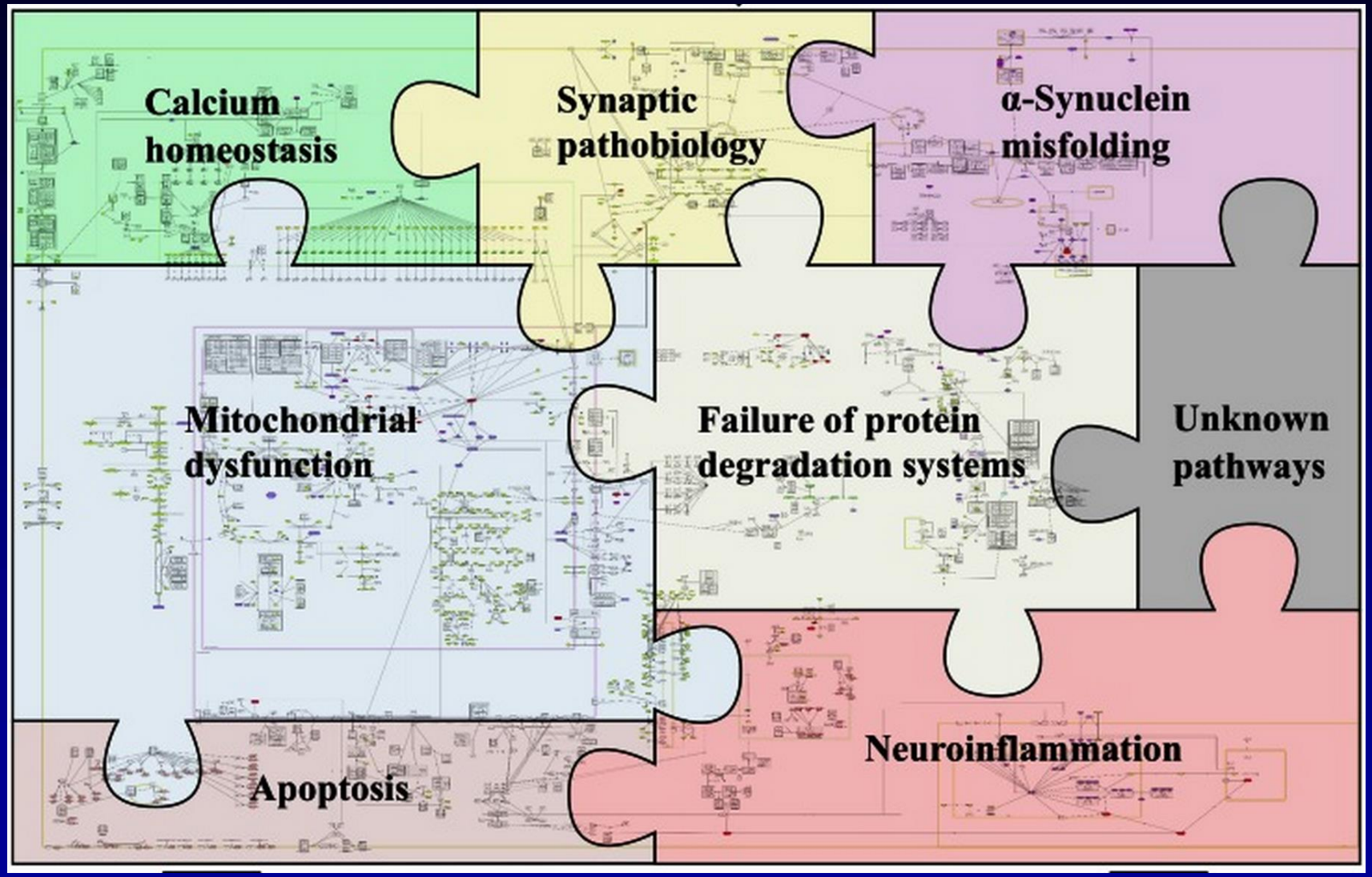
Parkinson's Disease

"Parkinson's Disease: The Life Cycle of the Dopamine Neuron", The New York Academy of Sciences, 2003

Parkinson's Disease Risk Factors

- Age
- Twins: concordance 75% for MZ, 22% for DZ by fluoro-dopa PET
- Positive family history
- Environmental factors: rural, pesticides
- Protective factor: smoking, coffee drinking

Biological systems involved in PD

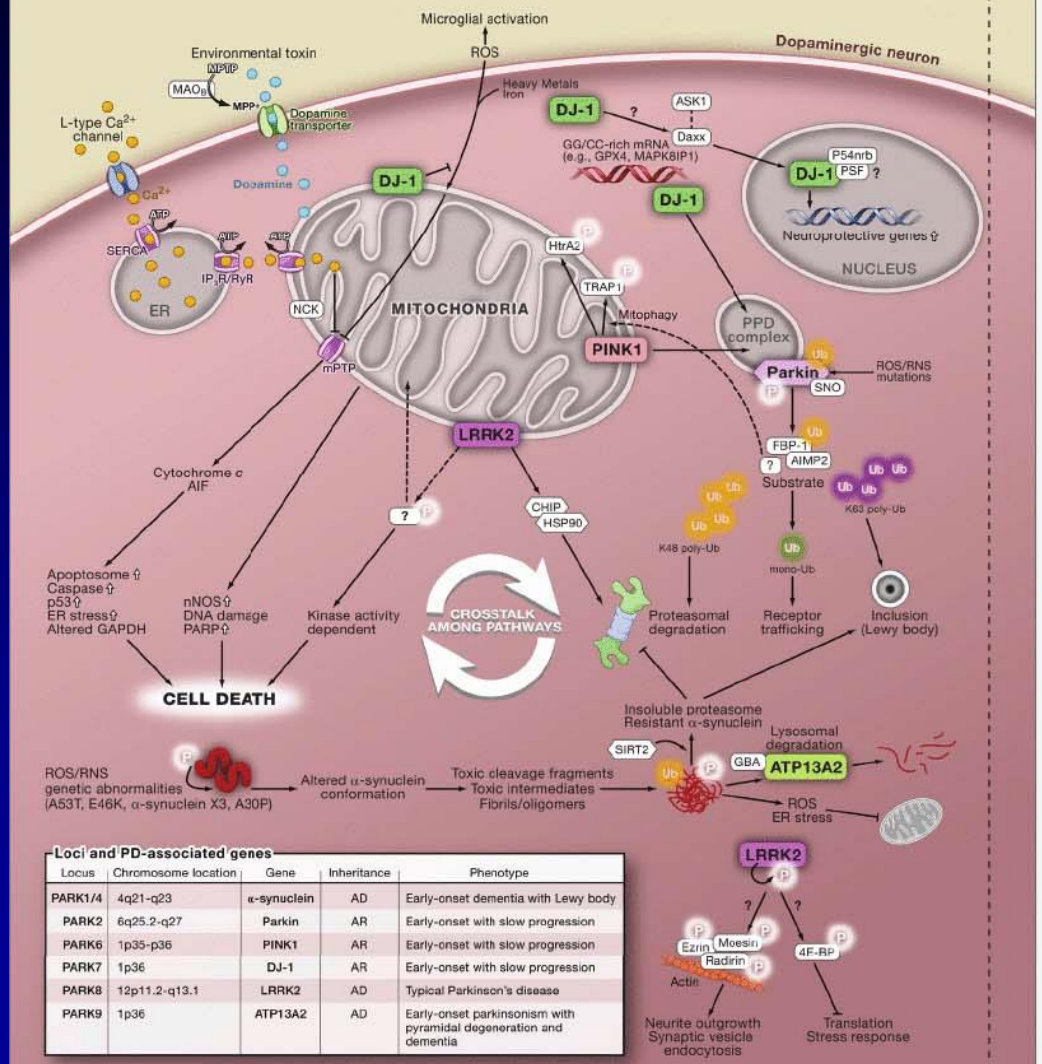


Fujita KA, Ostaszewski M, Matsuoka Y, Ghosh S, Glaab E, Trefois C, et al. Integrating pathways of parkinson's disease in a molecular interaction map. *Mol. Neurobiol.* 2014;49:88-102

SnapShot: Pathogenesis of Parkinson's Disease

Cell

Joo-Ho Shin, Valina L. Dawson, and Ted M. Dawson
NeuroRegeneration and Stem Cell Programs, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA



Loci and PD-associated genes

Locus	Chromosome location	Gene	Inheritance	Phenotype
PARK1/4	4q21-q23	α-synuclein	AD	Early-onset dementia with Lewy body
PARK2	6q25.2-q27	Parkin	AR	Early-onset with slow progression
PARK6	1p35-p36	PINK1	AR	Early-onset with slow progression
PARK7	1p36	DJ-1	AR	Early-onset with slow progression
PARK8	12p11.2-q13.1	LRRK2	AD	Typical Parkinson's disease
PARK9	1p36	ATP13A2	AD	Early-onset parkinsonism with pyramidal degeneration and dementia

See online version for legend and references.

SnapShot: Pathogenesis of Parkinson's Disease

Cell

Joo-Ho Shin, Valina L. Dawson, and Ted M. Dawson
NeuroRegeneration and Stem Cell Programs, Johns Hopkins University School of Medicine, Baltimore, MD 21205, USA

Parkinson's disease (PD) is the most common movement disorder characterized by death of dopaminergic neurons in the substantia nigra pars compacta. Sophisticated genetic analysis has revealed several PD-associated genes including those encoding α-synuclein, parkin, PINK1, DJ-1, LRRK2, and ATP13A2 (see table). Diverse environmental factors in conjunction with genetic risk factors lead to PD pathogenesis, although the exact mechanisms are still under investigation. This SnapShot summarizes the roles that proteins encoded by PD-associated genes play in both common and divergent mechanisms of PD pathogenesis.

Death of Dopaminergic Neurons

Unlike most other brain neurons, the dopaminergic neurons of the substantia nigra use L-type Ca^{2+} channels (containing a distinctive Cav 1.3 pore-forming subunit, Cacna1d) for pace making, resulting in increased ATP consumption and Ca^{2+} influx. Ca^{2+} enters the endoplasmic reticulum (ER) via a high-affinity smooth ER Ca^{2+} (SERCA) pump. Ca^{2+} flows back into the cytoplasm through inositol trisphosphate receptors (IP_3R) and ryanodine receptors (RyR) forming a high-concentration Ca^{2+} zone near mitochondria. Mitochondria in dopaminergic neurons take up Ca^{2+} via the Ca^{2+} uniporter, whereas mitochondrial Ca^{2+} efflux is mediated by the Na^+/Ca^{2+} exchanger (NCX). Mitochondria are a key target in PD and impaired mitochondria contribute to death of dopaminergic neurons. Due to their unique Cav 1.3 calcium channels, dopaminergic neurons are more vulnerable to environmental toxins, such as 1-methyl-4-phenyl-1,2,3,6 tetrahydropyridine (MPTP). MPTP selectively kills dopaminergic neurons by blocking the activity of mitochondrial complex I and has been used to create animal models of PD. Once monoamine oxidase B (MAOB) metabolizes MPTP into 1-methyl-4-phenyl-pyridium (MPP^+), MPP^+ is transported and concentrated in mitochondria by the dopamine transporter. This leads to inhibition of mitochondrial complex I, eventual depolarization of the mitochondrial membrane, and opening of the mitochondrial permeability transition pore (mPTP). This results in release of mitochondrial cell death effectors such as cytochrome *c* and apoptosis-inducing factor (AIF). Ultimately death of the dopaminergic neuron ensues through various cell death cascades including caspase-dependent and caspase-independent pathways, ER stress, neuronal nitric oxide synthase (nNOS) activation, DNA damage, poly(ADP-ribose) polymerase (PARP) activation, and GAPDH modification. Microglial activation also contributes to the demise of dopaminergic neurons.

PD Genes and Molecular Pathogenesis

Autosomal-Dominant

α-Synuclein: Even though the physiological function of α-synuclein is still unclear, numerous studies indicate its association with membranes, synaptic vesicle recycling, dopamine neurotransmission, and lipid interactions. This protein is the major structural component of Lewy bodies, which are the pathological hallmark of PD. Identification of genetic abnormalities in the *α-synuclein* gene have implicated the protein encoded by this gene in the pathophysiology of PD. Native unfolded or altered α-synuclein monomers with genetic modifications (A53T, E46K, A30P) form toxic intermediates such as oligomers and fibrils, which eventually form Lewy bodies. Triplication of the *α-synuclein* gene (X3) can also lead to PD due to the simple increase in the steady-state level of wild-type α-synuclein. In sporadic PD, reactive oxygen species (ROS), reactive nitrogen species (RNS), and aging play a role in the aggregation of α-synuclein. ROS/RNS production, disruption of macroautophagy, mitochondrial dysfunction, and proteasome inhibition can also be triggered by mutant or aggregated α-synuclein. The histone deacetylase, Sirtuin2 (SIRT2), may also contribute to PD pathogenesis.

LRRK2: LRRK2 consists of diverse domains, including a leucine-rich repeat, a Roc GTPase domain, a COR (C-terminal of Ras) domain, a kinase domain, and a WD40-repeat. LRRK2 is localized in the cytoplasm and is associated with membranous structures including mitochondria, the ER, and synaptic vesicles. Familial mutants of LRRK2 result in a gain of function and neuronal toxicity that is kinase dependent and regulated by the chaperones CHIP and HSP90. These chaperones control LRRK2 levels through the ubiquitin-proteasome system. Moesin is a putative LRRK2 substrate. Functional studies implicate LRRK2 in neurite outgrowth and the endocytosis of synaptic vesicles. 4E-BP is a putative LRRK2 substrate, suggesting that LRRK2 is involved in translation. Data suggest that LRRK2 may also regulate mitochondrial function. Disease-causing mutations in human LRRK2 consistently cause α-synuclein pathology.

Autosomal-Recessive

PINK1: Little is known about the function of PINK1, other than that it is thought to be a mitochondrial kinase that acts upstream of parkin in the PD pathogenesis cascade. Disease-causing mutations in PINK1 may lead to a loss of function. The mitochondrial chaperone, TRAP1, and the serine protease, HtrA2, are putative PINK1 substrates that play important roles in regulating mitochondrial function and mitochondrial-dependent cell death pathways. PINK1 may also physiologically regulate Ca^{2+} efflux from the mitochondria via NCK.

DJ-1: DJ-1 is a molecular chaperone with multiple functions. Disease-causing mutations in DJ-1 may lead to a loss of function. DJ-1 regulates ROS levels by acting as an atypical peroxiredoxin-like peroxidase and also modulates RNA metabolism and gene transcription. In addition, DJ-1 may bind to Daxx/apoptosis signal-regulating kinase1 (ASK1) and inhibit ASK1 activity and cell death. DJ-1 is also involved in a Parkin-PINK1-DJ-1 (PPD) complex that promotes the degradation of unfolded proteins.

Parkin: Mutations in the *parkin* gene and posttranslational modifications to the protein, such as phosphorylation or S-nitrosylation (SNO) by ROS/RNS, block parkin's ability to function as an E3 ubiquitin ligase. This leads to the accumulation of its substrates, including AIMP2, FBP-1, and others, which are somehow involved in mitochondrial dysfunction and neuronal toxicity. Parkin uses poly-K48 ubiquitin linkages to promote degradation of its substrates. Parkin also uses poly-K63 ubiquitin linkages or mono-ubiquitination to regulate intracellular signaling, receptor trafficking, and the formation of inclusions. Parkin acts downstream of PINK1 in genetic models and appears to play a role in the clearance of mitochondria by autophagy (mitophagy).

ATP13A2: ATP13A2 is a large lysosomal P-type ATPase. Although functional studies are at a very early stage, it is thought that ATP13A2 and the PD susceptibility gene encoding β-glucocerebrosidase (GBA) may be involved in the clearance of α-synuclein aggregates.

Abbreviations

AD, autosomal-dominant; AR, autosomal-recessive; 4E-BP, eukaryotic initiation factor 4E (eIF4E)-binding protein; PSF, pyrimidine tract-binding protein-associated splicing factor; p54nrb, p54 nuclear RNA-binding protein; GPX4, glutathione peroxidase 4; MAPK8IP1, mitogen-activated protein kinase 8-interacting protein 1; Ub, ubiquitin.

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Therapeutic Goals

- Address expectations
- Treatment goals are greatly customized – patients vary with regards to what's important to them
- Currently available treatments are all symptomatic
- Many potential confounds, educate
- Dynamic entity, continuously reassess
- Don't ignore non-motor treatments
- Don't ignore non-pharmacological treatments
  - Talk to the patient/patient centeredness
  - Multi-disciplinary teams
  - Importance of the care partner/biopsychosocial approach
  - Care away from the hospital: telehealth, wearables

Parkinson Disease Treatments

- Symptomatic
  - All currently approved treatments for PD
  - Help improve symptoms: the appearance and impact – without affecting the underlying causes of disease
  - Symptomatic and disease modifying efficacy may not be exclusionary of each other

- “Disease modifying” (not a synonym of “neuroprotective”)
  - There is no known proven or approved neuroprotective or disease modifying treatments – only symptomatic at this point
  - Disease modifying means altering any aspect of the disease in a lasting manner (e.g. after removing the intervention)
  - This may be through neuroprotection, but could be through a number of other mechanisms
  - Endpoints capturing disease modification technically more feasible than actual neuroprotection in human clinical trials
  - However, showing disease modification may imply underlying neuroprotection depending on the purported MoA

Management Options

- Supportive therapies! Multidisciplinary care!
  - PT
  - OT
  - SLP
  - Clinical mental health therapy/counseling
  - Social work
  - Music therapy, dance therapy, tai chi, yoga, rock steady boxing, Teracycle, etc
- Oral neurotransmitter replacement and modulation
  - C/L
  - Dopamine agonists
  - Anticholinergics
  - Extenders (MAO-B inhibitors, COMT inhibitors)
  - Others (amantadine)
- Advanced treatments
  - Surgical ablation and deep brain stimulation
  - Intestinal levodopa suspension (Duopa pump)
  - Inhaled C/L
  - SQ Apokyn
- Experimental/pending approval in US
  - Neuroprotection
  - SQ Pumps
  - Gene therapy
  - Stem cells
  - tDCS
  - Cannabis – CBD oil (NO! THC)

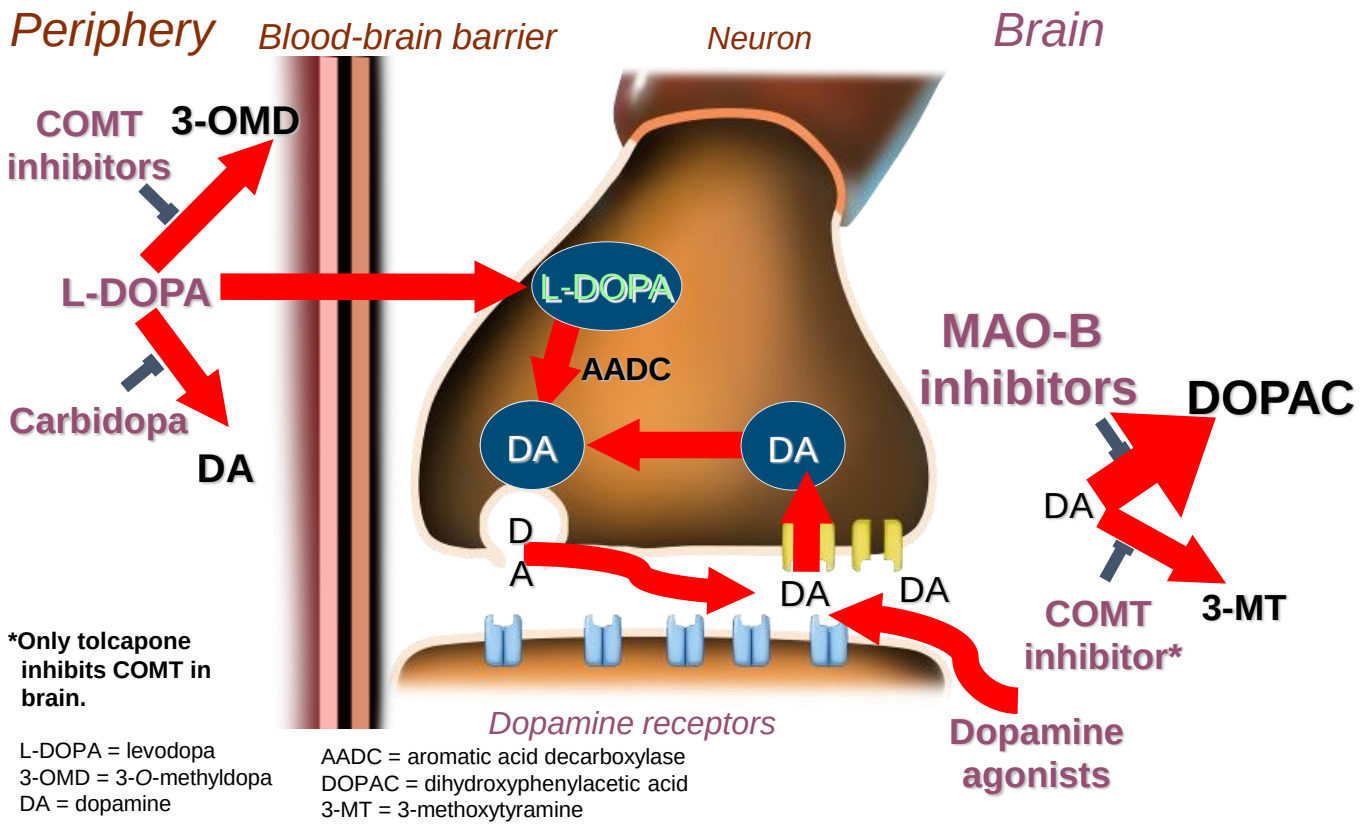
Summary 2: Pros and Cons of Available FDA-Approved Monotherapy

AGENT	PROS	CONS*
MAO B Inhibitors	Effective Once-daily dosing AE profile similar to that of placebo	Potential drug interactions
Carbidopa/ Levodopa	Highly effective Rapid onset of action	Motor fluctuations and dyskinesia are common with long-term use
Dopamine agonists	Effective Delays start of L-dopa Low risk of motor complications	Neuropsychiatric AEs Somnolence warning Agonist-specific AEs
Amantadine	Beneficial for tremor Antiparkinsonian effects	Cognitive AEs Anticholinergic AEs Withdrawal effects

*This does not represent a complete listing of safety information. Please see product prescribing information.
FDA = Food and Drug Administration; L-dopa = levodopa.

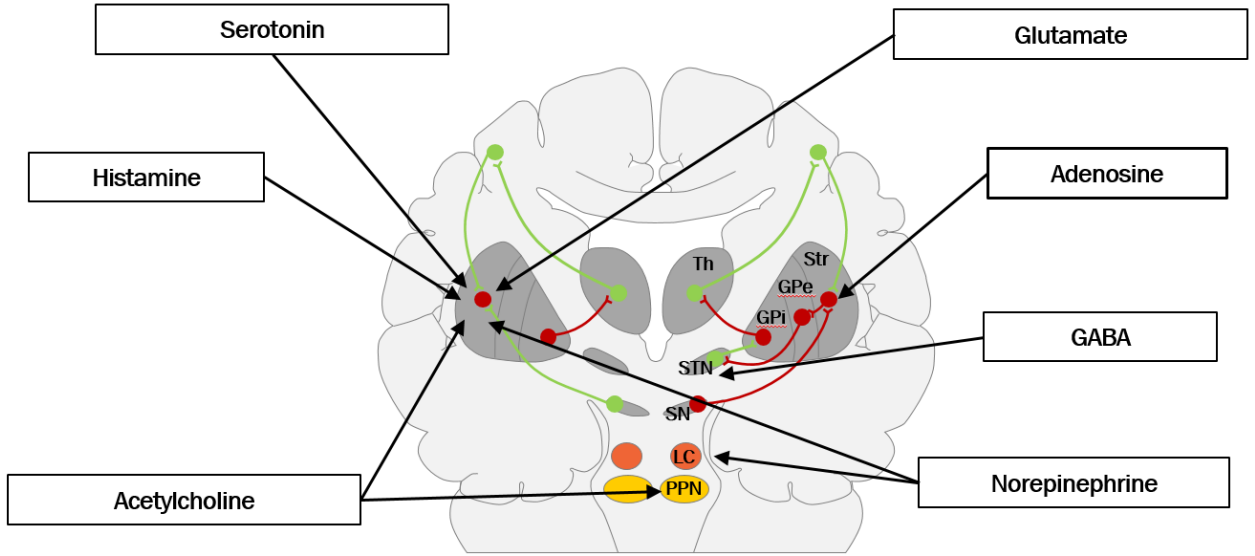
AZILECT Prescribing Information. 05/09.
Olanow et al. *Neurology*. 2001;56(11 suppl 5):S1-S88.
Jankovic. *Neurology*. 2002;58(4 suppl 1):S19-S32.

Sites of Action of PD Drugs



Within the basal ganglia, multiple nondopaminergic neurotransmitters/neuromodulators play a role in motor function

- Adenosine and GABA influence the indirect pathway
- Acetylcholine, glutamate, histamine, norepinephrine, and serotonin modulate the basal ganglia neuronal network
- These neurotransmitters help regulate the basal ganglia network with the dopaminergic system
- Nondopaminergic receptors serve as targets for modern PD treatments



Adjuvant Treatments for PD

- Istradefylline – novel adenosine A_{2a} receptor antagonist

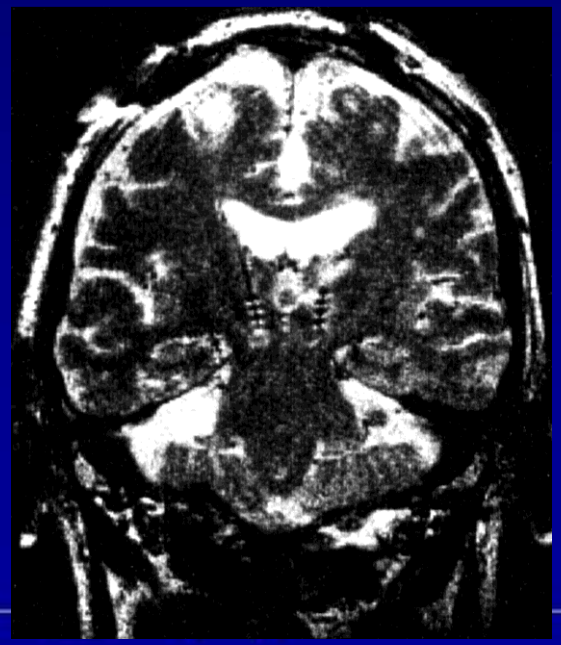
- COMT-inhibitors
  - Opicapone
  - Entacapone
  - Tolcapone
- MAO-B inhibitors
  - Selegiline
  - Rasagiline
  - Safinamide

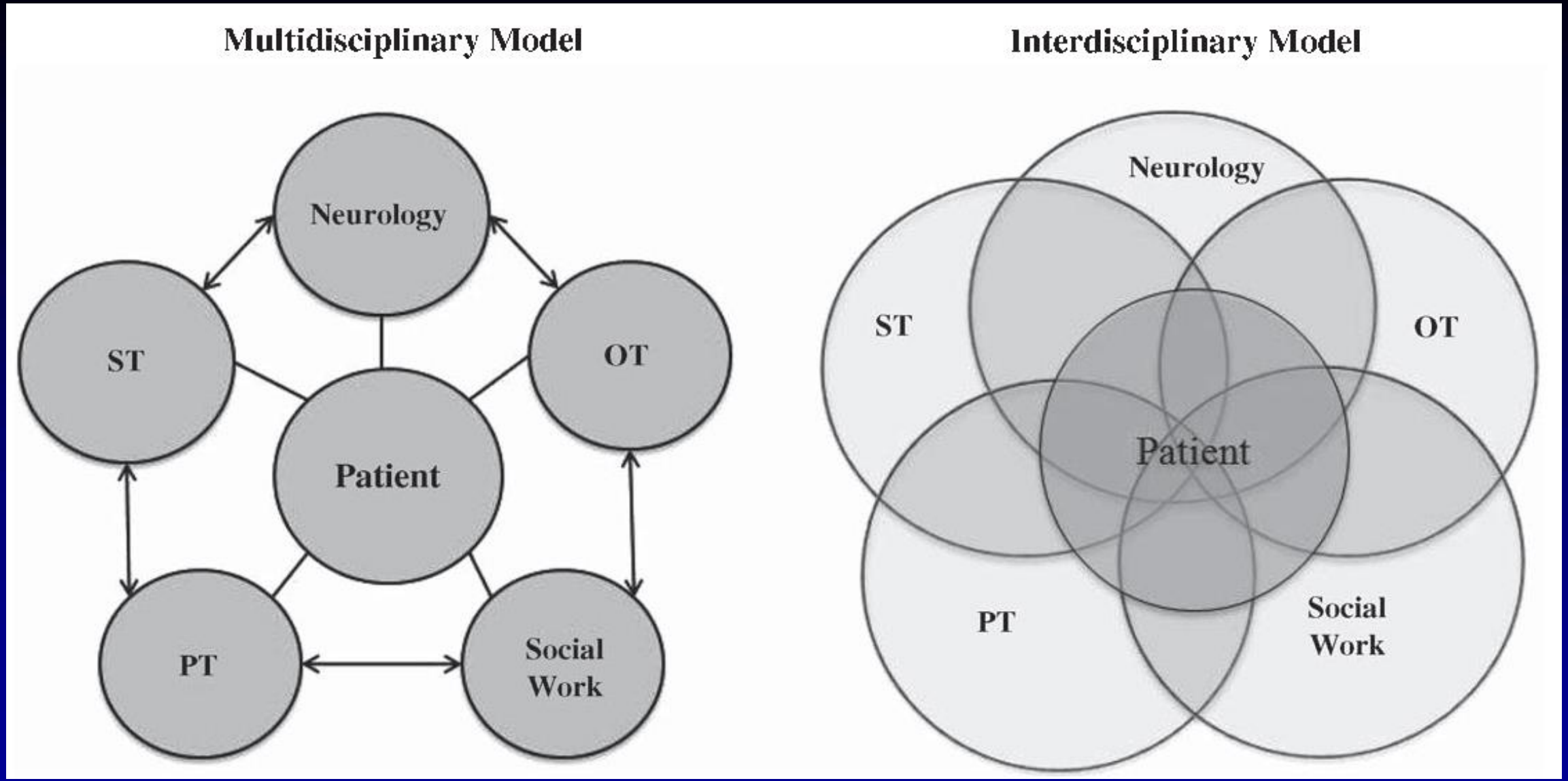
- Amantadine may be adjuvant besides monotherapy – new long-acting formulations have become available

- “Rescue” options
  - Inhaled carbidopa/levodopa
  - SQ injectable apomorphine
  - Sublingual film apomorphine

Surgical Treatments for PD

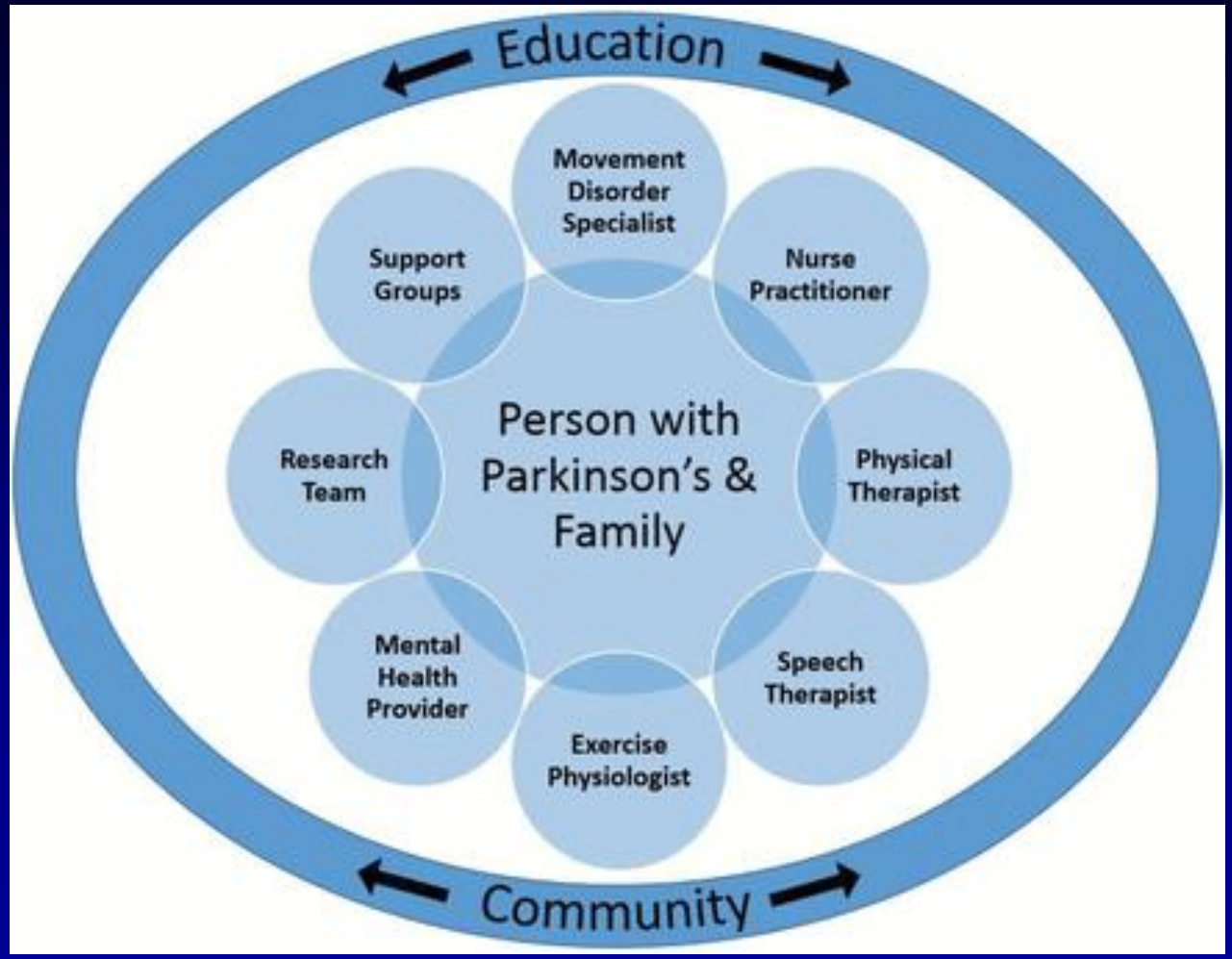
- MRgFUS
- Deep Brain Stimulation
  - Activa device system from Medtronic
  - Infinity device system from St. Jude
  - Vercise device system from Boston Scientific
- Levodopa-carbidopa intestinal gel infusion pump





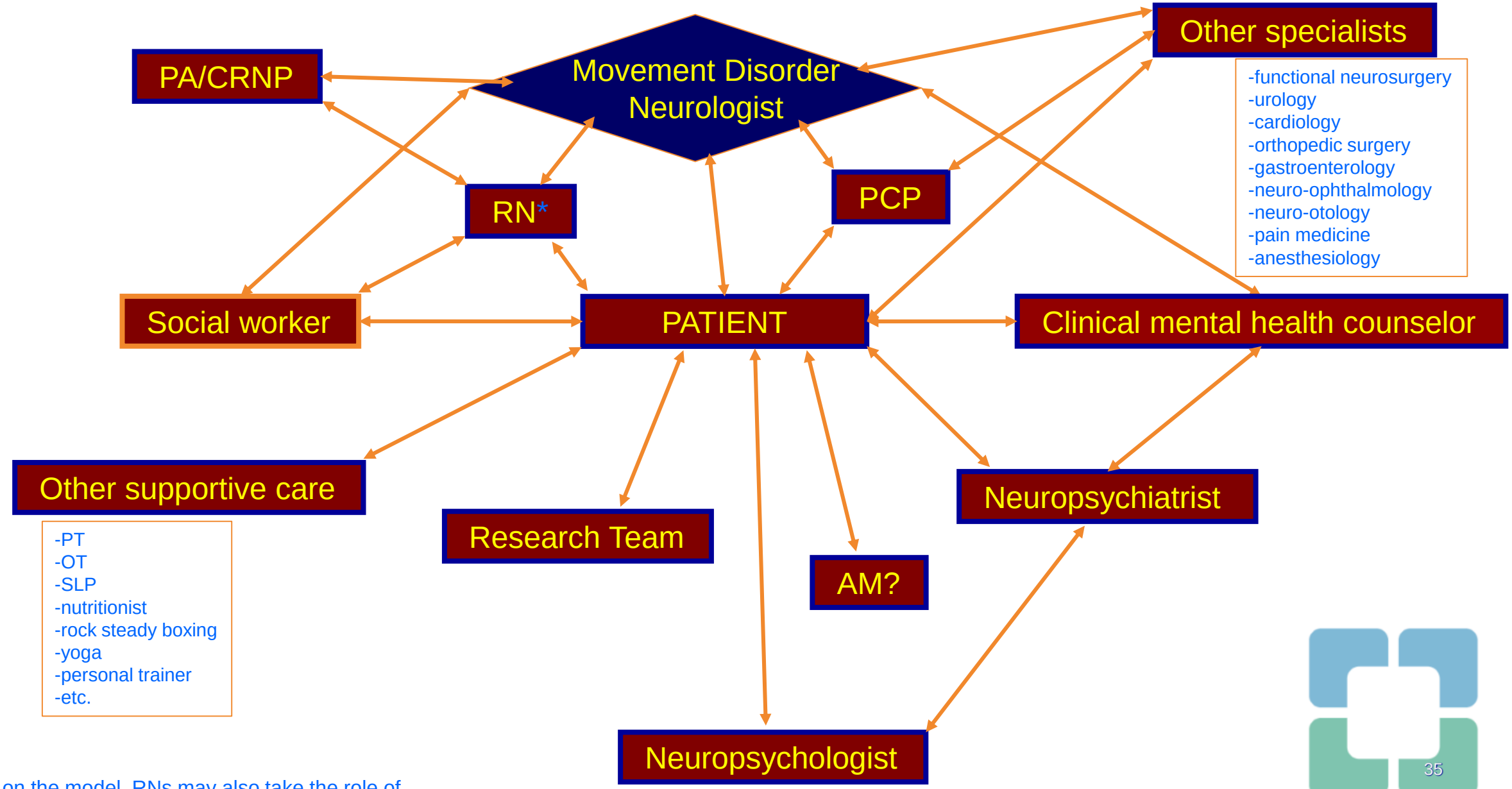
The integrative care of Parkinson's disease: a systematic review.
Lindsay Penny Prizer, Nina Mikelashvili Browner
Published 2012 in Journal of Parkinson's disease
DOI:10.3233/JPD-2012-12075

Implementation of an Integrative Holistic Healthcare Model for People Living with Parkinson's Disease



Implementation of an Integrative Holistic Healthcare Model for People Living with Parkinson's Disease
Ingrid Pretzer-Aboff, PhD Allen Prettyman, PhD
The Gerontologist, Volume 55, Issue Suppl_1, 1 June 2015, Pages S146–S153,
https://doi.org/10.1093/geront/gnv004
Published: 16 May 2015

Multi-Disciplinary Care in PD



*depending on the model, RNs may also take the role of patient educator, research coordinator, and are key toward the PDNS model

Parkinson's Foundation Center of Excellence (COE)

- Recognizes medical facilities with specialized, multi-disciplinary teams providing evidence-based PD care.
- Each center is required to meet rigorous care, professional training, research, community education and outreach criteria.
- As of 2022, there are **51** COEs in the GCN.
  - **37** located in the U.S.
  - **14** located internationally

Parkinson's Foundation
CENTER OF EXCELLENCE

Designation Criteria

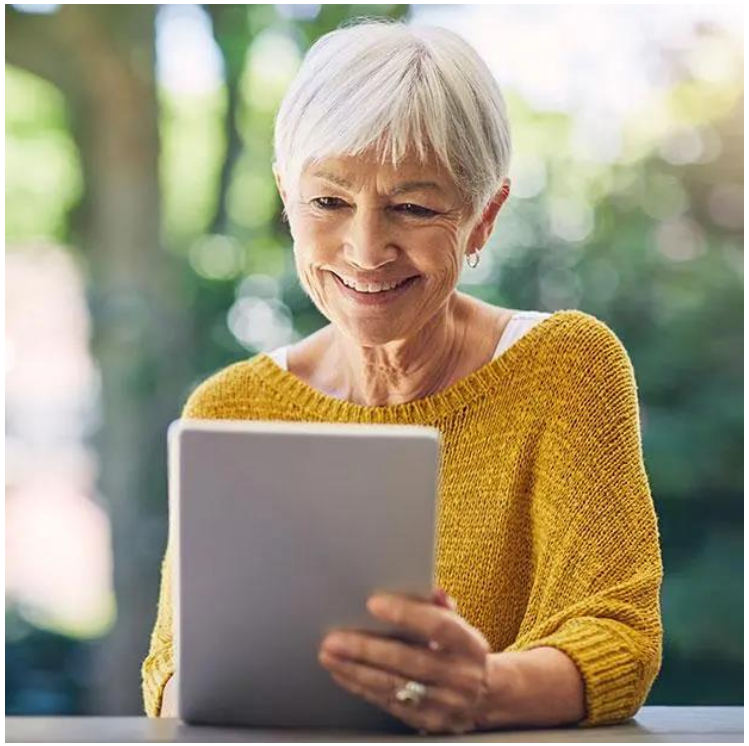
- **Care**
- **Education**
- **Community Outreach**
- **Research**

Care

- Care must be **coordinated, comprehensive** and provided by an **interdisciplinary team**
- Provide **expert care across the full spectrum of patient issues**, including motor, non-motor (particularly neuropsychiatric and cognitive symptoms)
- Provide all people with Parkinson's **equal access to services**

Education

- Provided at the center for:
  - Health care professionals
  - People with PD
  - Care partners



- Ensure that clinicians are knowledgeable about all evidence-based treatments for Parkinson’s Disease

- Educational events for people with PD and families, to provide current information about Parkinson’s disease (such as an annual symposia or lunch and learn programs)

- Provide resources - such as newsletters, fact sheets, etc.

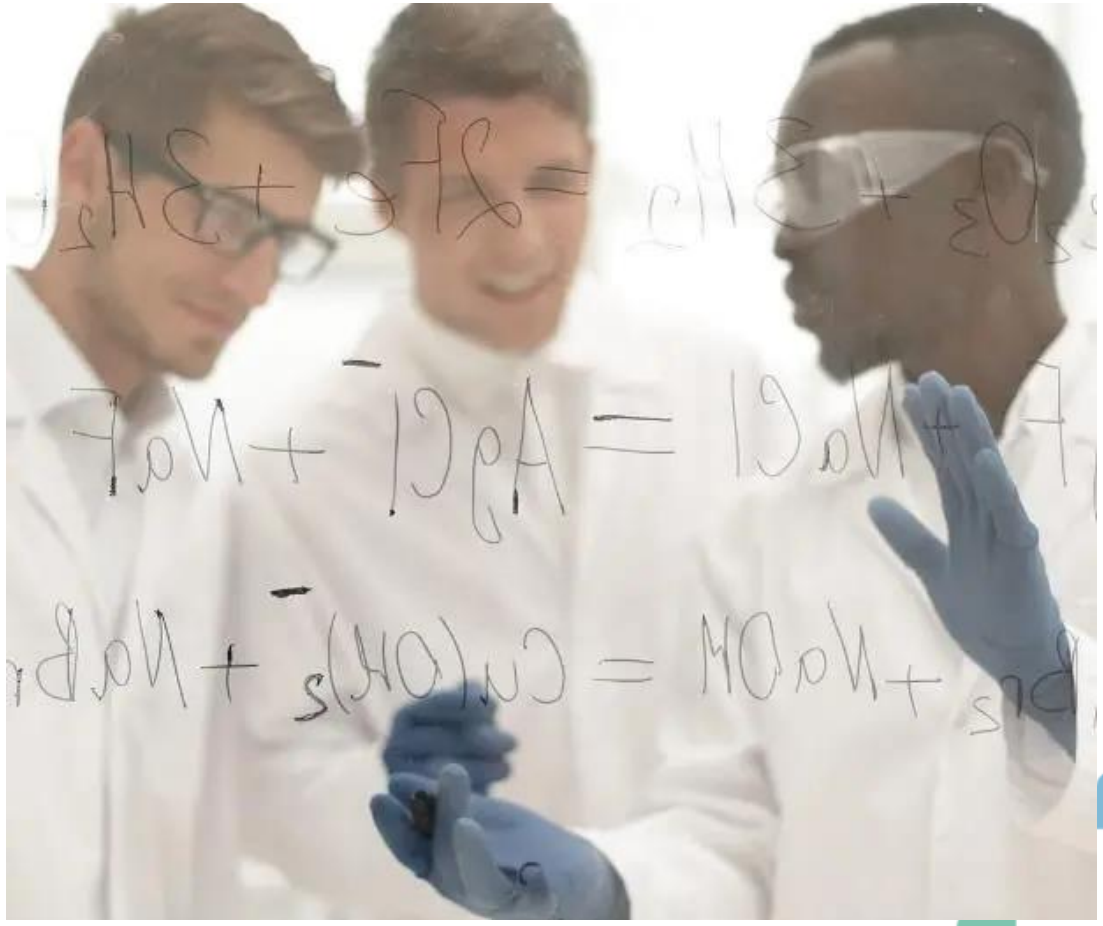
Community Outreach

- Provided outside of the Center for:
  - Healthcare professionals
  - People with PD
  - Care Partners
- Facilitation of community events
- Provide resources for alternative wellness programs, support groups, exercise programs, etc.
- Provision of disease-specific community provider education and training

Research

Centers must:

- Conduct research relevant to Parkinson's
- Publish findings from this research in peer-reviewed journals



U.S. COEs - 2022

AZ—Barrow Neurological Institute
CA—University of Southern California
CA—University of California, San Francisco
CA—University of California, San Diego Movement Disorder Center
CO—University of Colorado Movement Disorders Center
DC—Medstar Georgetown University Hospital
FL—University of Florida Center for Movement Disorders and Neurorestoration
FL—University of Miami
FL—University of South Florida Parkinson's Disease and Movement Disorders Center
GA—Medical College of Georgia, Augusta University
GA—Emory University
IA—University of Iowa
IL—Northwestern University Movement Disorders Center
IL—Rush University Medical Center
IN—Indiana University School of Medicine
KS—University of Kansas Medical Center
MD—Johns Hopkins Parkinson's Disease and Movement Disorders Center
MA—Massachusetts General Hospital
MA—Beth Israel Deaconess Medical Center
MN—Struthers Parkinson's Center
NH—Dartmouth Hitchcock Medical Center
NY—Mount Sinai Beth Israel
NY—Columbia University Irving Medical Center
NY—Marlene and Paolo Fresco Institute for Parkinson's and Movement Disorders at NYU Langone Medical Center
NY—University of Rochester Medical Center
NC—Duke Health Movement Disorders Center
NC—University of North Carolina at Chapel Hill School of Medicine
NV—Cleveland Clinic Lou Ruvo Center for Brain Health
OH—Cleveland Clinic Center for Neurological Restoration
OR—Oregon Health and Science University Parkinson Center
PA—University of Pennsylvania Movement Disorder Center
PA—Jefferson Health’s Comprehensive Parkinson’s Disease and Movement Disorder Center
SC—Medical University of South Carolina
TN—Vanderbilt University Medical Center
TX—Baylor College of Medicine
UT—University of Utah
VA—Virginia Commonwealth University

International COEs – 2022

Australia
Victorian Comprehensive Parkinson's Program

Canada
McGill Parkinson Program
University of Western Ontario, London Health Sciences Centre
University of Calgary
University of Alberta
Pacific Parkinson's Research Centre, University of British Columbia
Toronto Western Hospital Movement Disorders Center

Germany
Philipps University

Israel
Tel Aviv Sourasky Medical Center

Netherlands
Nijmegen Parkinson Center

Singapore
Singapore National Neuroscience Institute

Taiwan
National Taiwan University Hospital, Center for Parkinson & Movement Disorders

United Kingdom
Kings College Hospital
Derby Hospitals NHS Foundation Trust and The University of Nottingham

COMMITMENT TO QUALITY: PD & Movement Disorders @CCLRCBH

Multiple recognitions as a Center of Excellence

Parkinson's Foundation
CENTER OF EXCELLENCE

Director: Zoltan Mari, MD FAAN

LBDA
LEWY BODY DEMENTIA ASSOCIATION
RESEARCH CENTERS OF EXCELLENCE

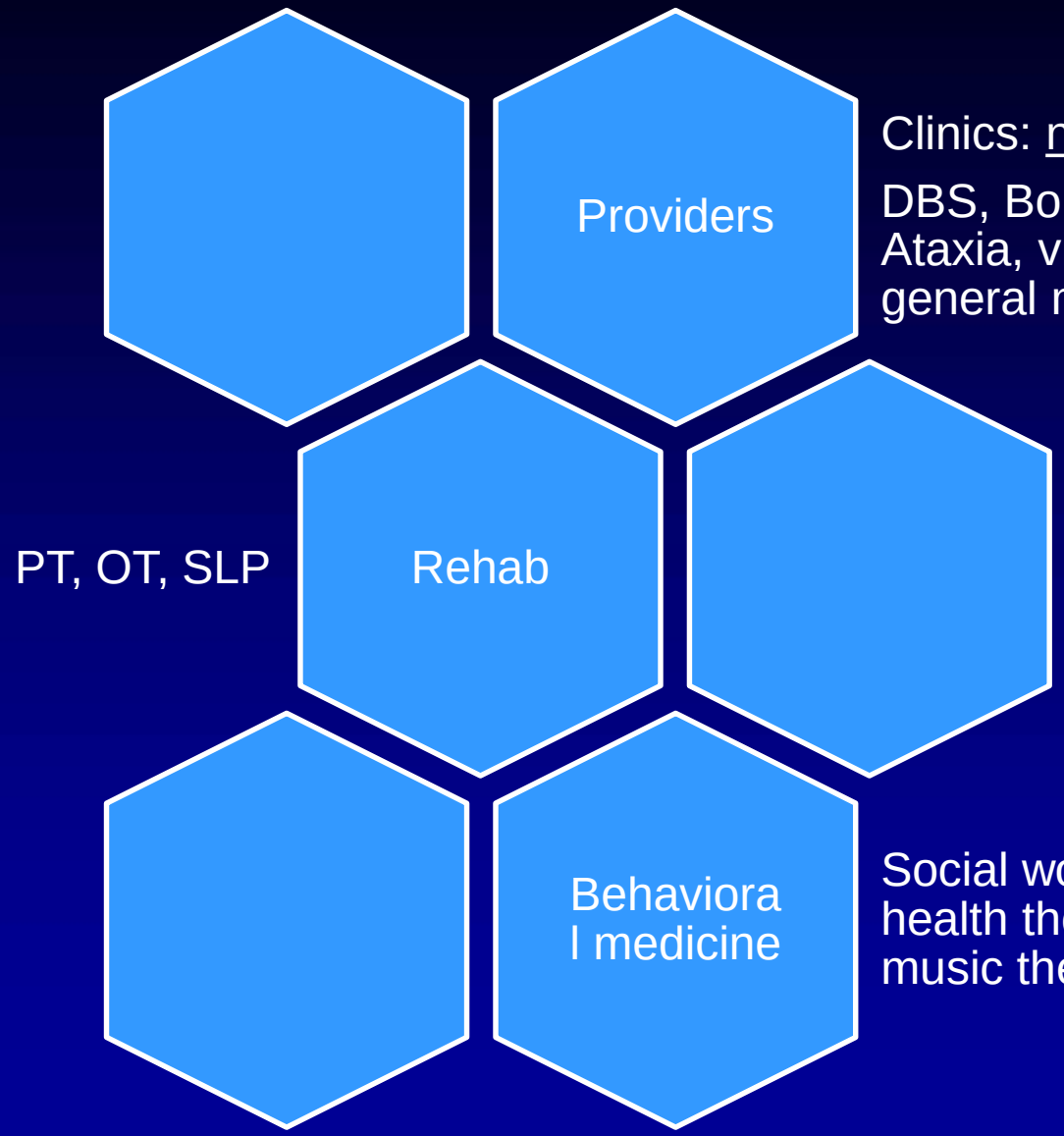
Currently: accreditation on hold/re-application under way (formerly Co-PIs: MS/ZM, then PI: ZM)

curePSP | CENTER OF CARE

Currently: just re-accredited (director: ZM)

HDSA CoE: application under way (Dr. Oguh)

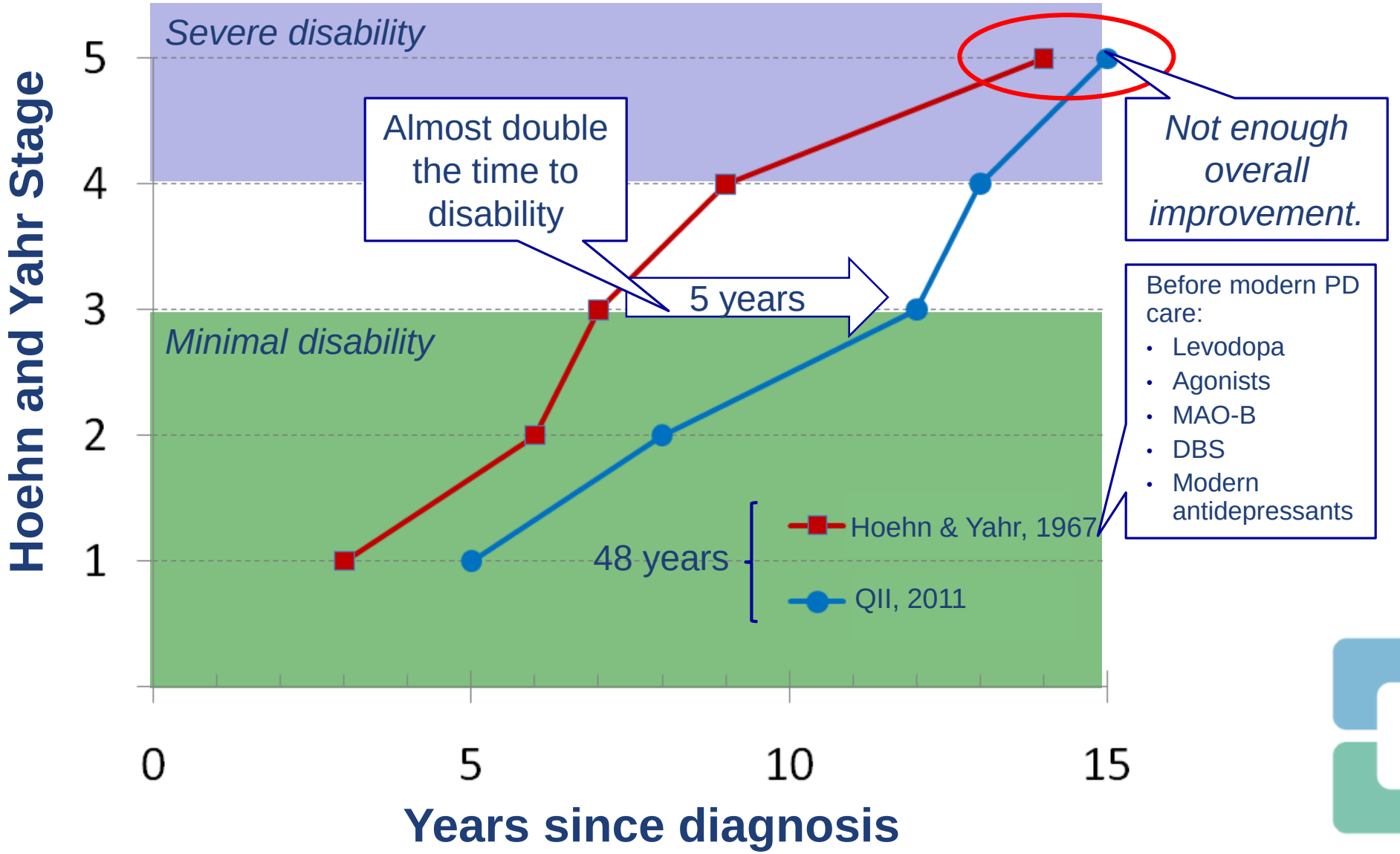
Clinical Domain: PD & Movement Disorders at the CCLRCBH



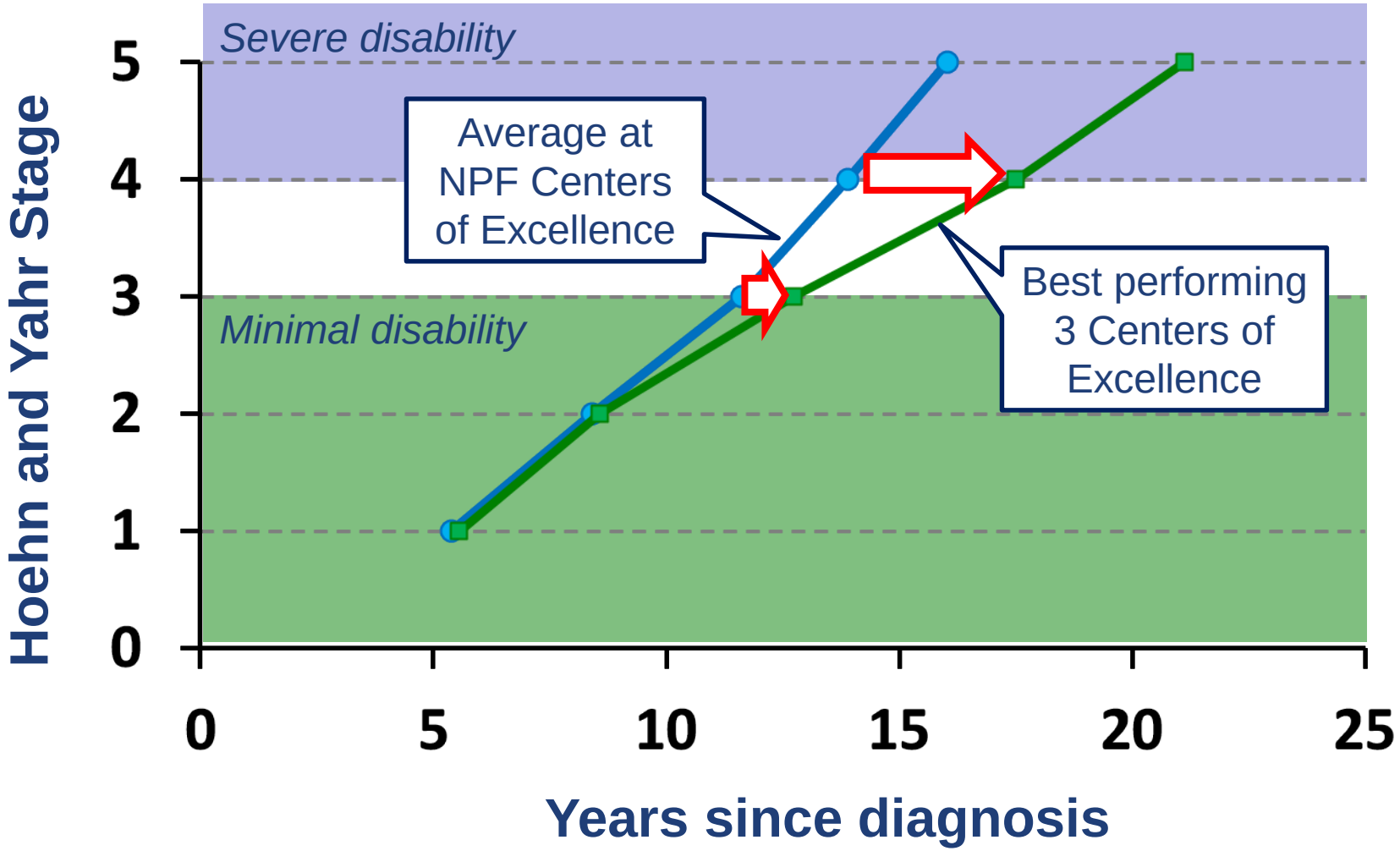
Additional services:

- Nursing
- Patient education
- Library
- Outreach
- Research
- Imaging
- Neuropsychology
- Neuropsychiatry
- Genetics counseling

Dramatic Gains from Treatment



Further Gains Are Still Possible



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- Nature: Incidence of Parkinson disease in North America: https://www.nature.com/articles/s41531-022-00410-y

Cleveland Clinic

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Creating an Age-Friendly Health System & Dementia-Friendly Community in Nevada

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UNLV

UNLV SCHOOL OF DENTAL MEDICINE

UNLV SCHOOL OF PUBLIC HEALTH

UNLV HOWARD R. HUGHES COLLEGE OF ENGINEERING

UNLV SCHOOL OF NURSING

UNLV HEALTH

UMC UNIVERSITY MEDICAL CENTER

WILLIAM BEE RIRIE CRITICAL ACCESS HOSPITAL AND RURAL HEALTH CLINIC

Mission Pines Nursing & Rehabilitation Center

St. Joseph Transitional Rehabilitation Center

Cleveland Clinic

Nathan Adelson HOSPICE

NEVADA SENIOR SERVICES

alzheimer's association

Southern Nevada SLP SENIOR LAW PROGRAM

VMSN Volunteers in Medicine of Southern Nevada

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