

# Dementia in Parkinson's Disease: Clinical Presentation and Considerations in Diverse Rural Communities

Karen Torres, PsyD, ABPP  
Clinical Neuropsychologist  
Assistant Professor of Neurology

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# Learning Objectives

- The rapid growth of Parkinson's disease
- Complexity of clinical presentation
- Neuropsychological Functioning
  - Cognitive Impairment
  - Mood symptoms
- Environmental risk factors for PD
- Individuals in rural/agricultural communities at increased risk
  - Example in the Hispanic-Latino Community

# Parkinson's Disease (PD)

PD is the 2<sup>nd</sup> leading neurodegenerative disease after Alzheimer's disease<sup>1</sup>

- One million living with PD in the U.S (8.5 million worldwide)
  - 90,000 new cases per year in the U.S
  - Fastest growing neurological disorder, worldwide (est. 12 million by 2030)
- Age of onset 40-70
  - More typically >60
- Insidious progression over the course of 5-20 years
- Men > Women (1.5x)
- Most cases do not have a familial link
  - 5-15% associated with genetic mutations

# Parkinson's Disease (PD)

- Environmental exposures can increase risk
  - Occupational exposure to pesticides is associated with an approximately 50% increased risk of PD <sup>2, 3</sup>
- Caucasian and Hispanic-Latino communities are twice as likely to develop PD
  - One study found an incidence rate per 100,000 was highest among Hispanic-Latino communities, followed by Caucasians.
- A large meta analyses of Latin American countries found a prevalence of 472/100,000 vs. 315/100,000 globally <sup>4</sup>

PD is highly heterogeneous in its presentation and progression; individuals can vary dramatically in symptom profile, symptom severity, and rate of decline.



# Clinical Features of PD – It's Complex!

- **Motor Symptoms**

- Tremor
  - Resting tremor
  - Asymmetric
- Bradykinesia
  - Lack of initiation
  - Masked facies (hypomimia)
  - Micrographia
  - Hypophonia
- Rigidity

- **Cognition**

- Cognitive Impairment
- Dementia

- **Autonomic Disturbance**

- Orthostatic hypotension
- Constipation
- Erectile dysfunction
- Decreased/loss sense of smell
- Swallowing difficulties

- **Sleep Symptoms**

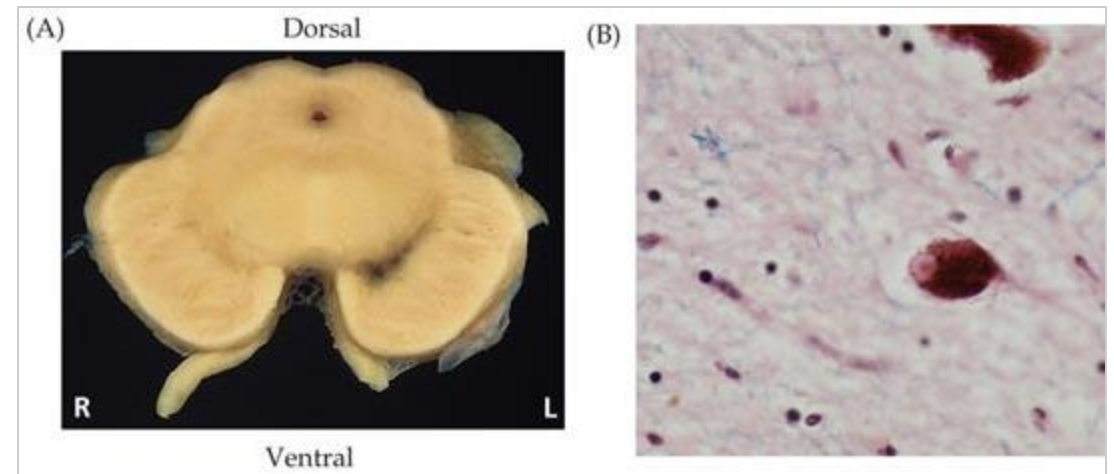
- REM sleep behaviors
  - Active movements in sleep

- **Mood**

- Depression, anxiety, apathy, impulse control difficulties

# Neuropathology of Parkinson's Disease

- Parkinson's disease (PD) is an idiopathic neurodegenerative disorder caused by the loss of dopaminergic neurons in the substantia nigra pars compacta (appears pale)
- Lewy body accumulations in the involved nuclei of the basal ganglia, cortical regions
  - Principal protein is  $\alpha$ -synuclein



# Prodromal Stage

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## *Autonomic*

Smell

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REM sleep behaviors

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Erectile dysfunction

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## *Psychiatric*

Depression

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Anxiety

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## *Cognitive*

Mild Cognitive  
Impairment (20-30% at  
time of diagnosis of PD)

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Executive Dysfunction

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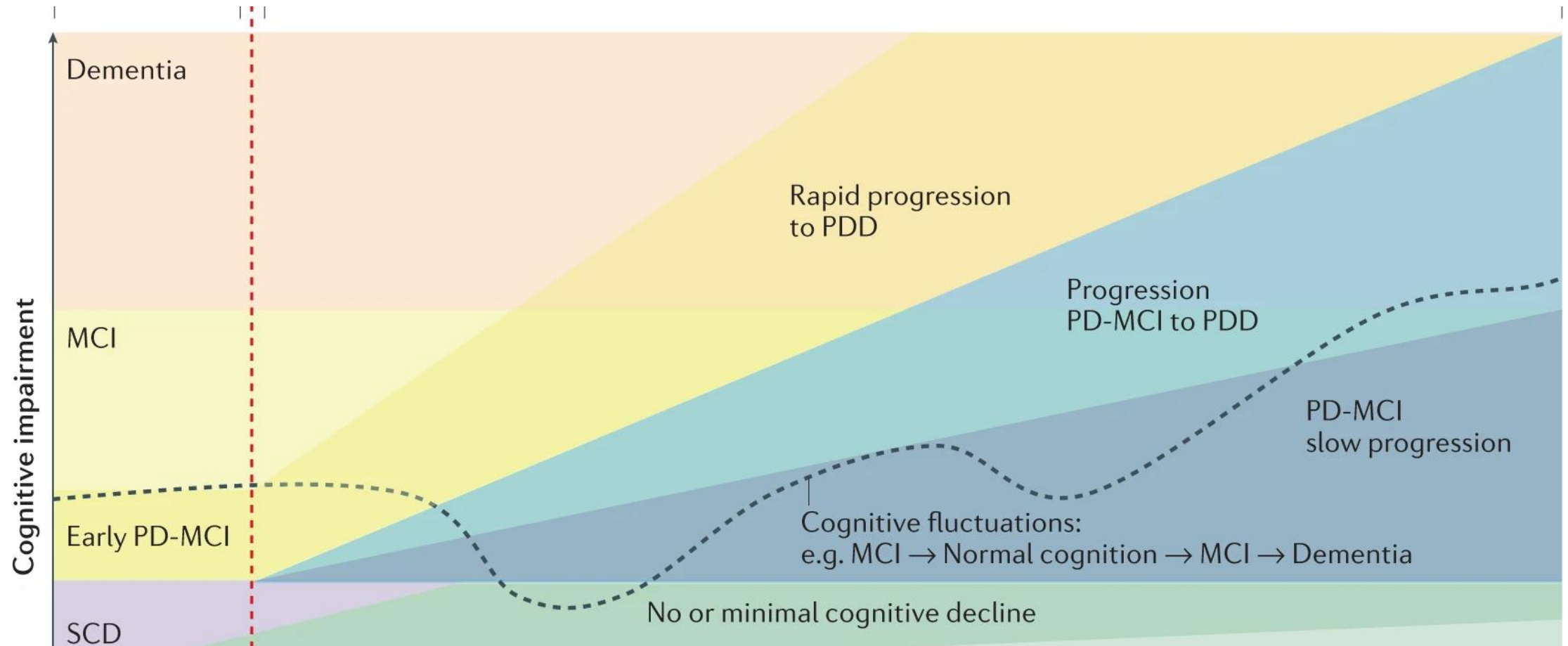
Symptoms present decades before onset of motor symptoms

- 60% of the neurodegeneration of the dopaminergic system has depleted by the time motor symptoms begin



# Neuropsychological Functioning

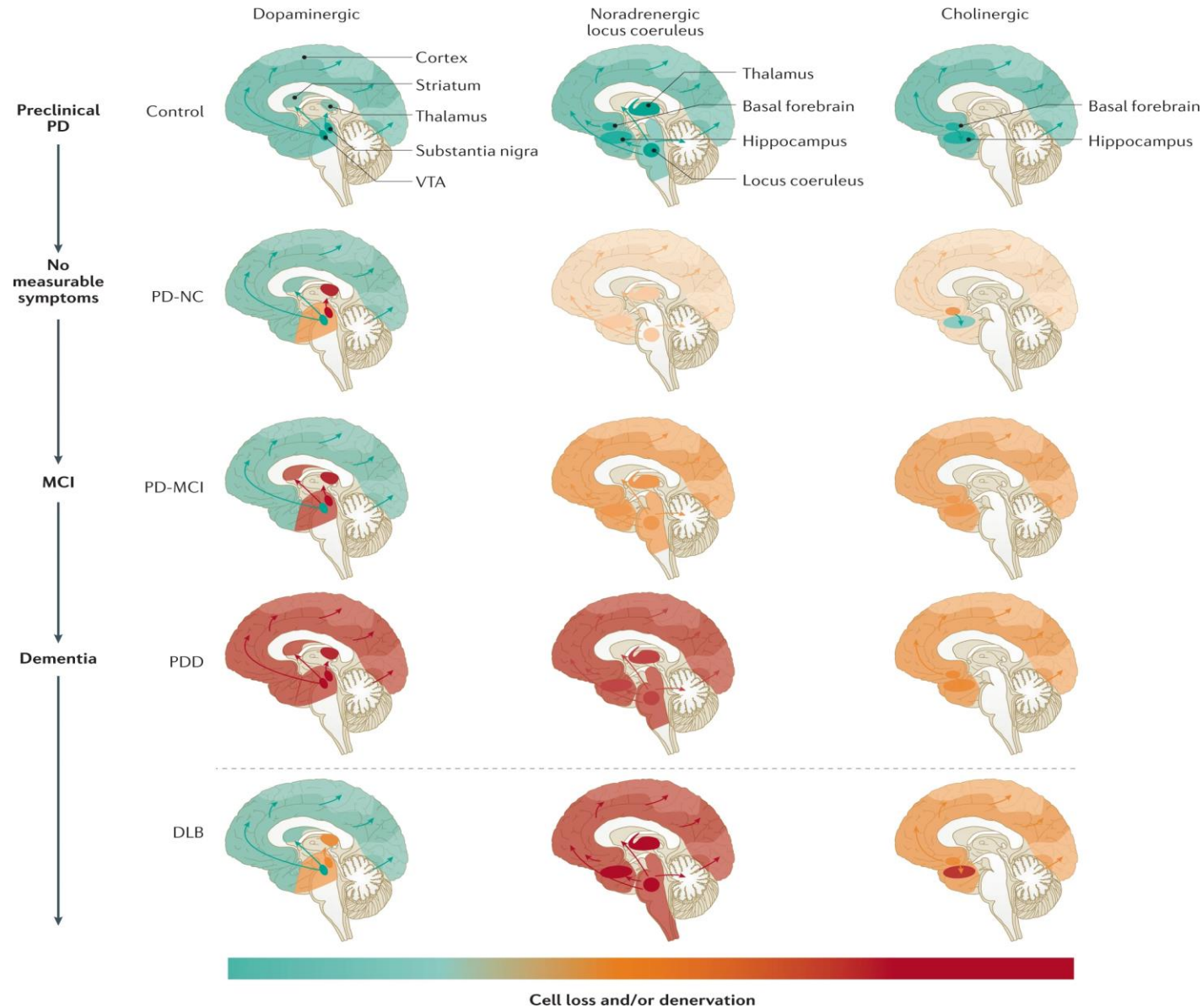
# The cognitive spectrum and the heterogeneity of progression of cognitive impairment in Parkinson disease <sup>5</sup>



# Cognitive Impairment Present at Diagnosis

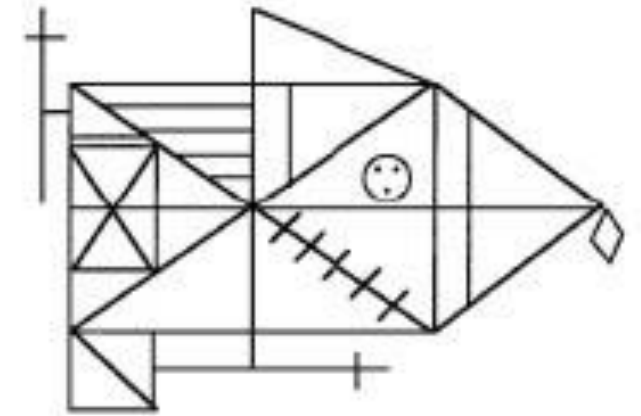
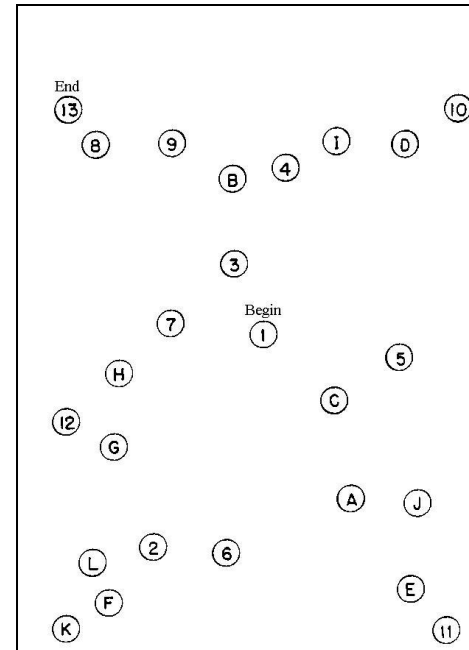
- For approximately 20-30 percent of people diagnosed with PD, by the time they are diagnosed, 60% of their dopamine has been depleted in the substantia nigra
- Cognitive domains most impacted:
  - Executive functioning
    - Cognitive efficiency
    - Organization
    - Mental flexibility
- Prodromal cognitive deficits
  - Not likely to be captured on neuropsychological evaluations traditional scores
  - Process scores may be more likely to capture weaknesses in these areas

# Neurotransmitter deficits associated with cognitive decline in PD and DLB <sup>5</sup>



# Cognitive Domains Affected Early <sup>6</sup>

- Frontal-striatal networks
  - Executive functioning
  - Phonemic fluency
  - Memory retrieval (but not storage)
- Posterior Cortex
  - Visuospatial abilities



# Risks for Cognitive Decline

- Presence of hallucinations
- Older age of onset
- Severity of motor symptoms
  - Axial, rigidity vs. tremor
- REM Sleep Behavior Disorder (RBD)<sup>7</sup>
  - Frontostriatal deficits
  - Posterior cortical
  - Conversion to PDD
- Male gender
- Presence of depression
- Posterior cortical pattern of cognitive impairment
  - Visuospatial

# Parkinson's Disease Cognitive Impairment

## • Mild Neurocognitive Disorder

- “Mild Cognitive Impairment”
- Present in 20-30% PwP at diagnosis
- Evidence of **modest** cognitive decline from previous level of performance on one or more cognitive domains
  - Concern from others
  - Performance on objective measures
- Cognitive deficits **do not interfere** with instrumental activities of daily living
- Not better explained by other etiologies

## • Conversion to Dementia <sup>8</sup>:

- ParkWest Cohort estimated 60% conversion to dementia 5 years after diagnosis
- However, has been estimated that some of individuals revert to normal cognition and then experience cognitive decline later in the course of the disease

# MDS PD-MCI Criteria

## Box 1 | Movement Disorder Society PD-MCI diagnostic criteria<sup>7,120</sup>

### Level I – Abbreviated assessment

- Impairment on Parkinson disease (PD)-appropriate global cognitive ability scale (such as Montreal Cognitive Assessment (MoCA), Parkinson's Disease – Cognitive Rating Scale (PD-CRS), Mattis Dementia Rating Scale Second Edition (MDRS-2))
- Impairment on at least two neuropsychological tests when a limited set of tests is used (less than two tests per domain or less than five cognitive domains assessed)

### Level II – Comprehensive assessment

- Neuropsychological testing includes two tests per domain:
  - Attention and working memory
  - Executive functions
  - Language
  - Memory
  - Visuospatial skills
- Impairment on two tests in one domain or impairment on one test in two different domains
- Impairment shown by:
  - Score 1–2 SD below norm
  - Significant decline on serial testing
  - Significant decline from estimated premorbid functioning

### PD with mild cognitive impairment (PD-MCI) subtype classification (comprehensive level II assessment required)

- Single domain: impairment on two or more tests in one domain
- Multiple domain: impairment on at least one test in each of two or more domains



# PD-MCI Subtypes

- The dual syndrome hypothesis proposes that PD-related cognitive decline is likely caused by
  - (1) degeneration of dopamine neurons in the substantia nigra and subsequent dopamine depletion resulting in disruption to the frontal–subcortical system
    - Executive functioning
    - Processing speed
  - (2) widespread cortical atrophy caused by the buildup of alpha-synuclein and tau
    - Visuospatial skills
    - Language
    - Memory

# Parkinson's Disease Cognitive Impairment

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- **Major Neurocognitive Disorder**

- “Dementia”
- Evidence of **significant cognitive decline** from previous level of performance on one or more cognitive domains
  - Concern from others
  - Performance on objective measures
- Cognitive deficits **interfere** with independent activities of daily living
- Not better explained by other etiologies
  - Delirium
  - Major Depressive Disorder
- Affects 80% of PD patients 5-20 years after diagnosis
- Affects 50% of PD patients 10 years post diagnosis

# MDS PDD Criteria

## Box 2 | Diagnostic procedure Movement Disorder Society PDD criteria<sup>8,144</sup>

### Level I — Parkinson disease dementia (PDD)

- A diagnosis of Parkinson disease (PD) based on the UK Brain Bank criteria for PD
- PD developed prior to the onset of dementia
- Mini-Mental State Examination (MMSE) below 26
- Cognitive deficits severe enough to impact daily living (caregiver interview or Pill Questionnaire) independent of motor symptoms
- Impairment in more than one cognitive domain, that is, at least two of the following aspects:
  - Months Reversed or Seven Backward
  - Lexical Fluency or Clock Drawing
  - MMSE Pentagons
  - Three-Word Recall
- Absence of major depression
- Absence of delirium
- Absence of other abnormalities that obscure diagnosis

### Level II — Comprehensive assessment for characterizing PDD

The level II evaluation assesses four domains:

- Decreased global cognitive efficiency
- Subcorticofrontal features of PDD
- Instrumental (cortically mediated) functions:
  - Language
  - Visuoconstructive
  - Visuospatial
  - Visuoceptive
- Neuropsychiatric features:
  - Apathy
  - Depression
  - Visual hallucination
  - Psychosis

# Parkinson's Disease Dementia vs. Dementia with Lewy Bodies

- Traditionally, the 1-year rule has been used to distinguish DLB from PDD:
  - If dementia occurs more than 1 year after the diagnosis of PD, the diagnosis is PDD, whereas parkinsonism occurring after or simultaneously with dementia is classified as DLB.
- Distinct cognitive profiles:
  - PDD: Executive functioning, visuospatial, attention/processing speed
  - LBD: Visuospatial, memory, language decline
    - Very similar to the posterior subtype of Parkinson's disease

# Parkinson's Disease and Dementia

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- Dementia in PD compromises quality of life
  - Decreased independence, health, and social relationships
  - Care partners affected by increased need for assistance with **multiple** symptoms
- Financial strain related to caregiving:
  - Respite care
  - Additional support
  - Medical expenses

# Management of Cognitive Impairment

- Pharmacotherapy
  - Rivastigmine (cholinesterase inhibitor)
  - RCT showed modest clinical improvements in cognition<sup>9</sup>
  - Approved by the FDA for PDD, but MCI-PD
- If PDD psychosis is present, stepwise withdrawal of non-levodopa medications (anticholinergic medication) is recommended
- Cognitive Intervention
  - One study found that computer-based interventions for 2-3 times per week over 3-12 weeks resulted in improvements in aspects of executive functioning, memory, processing speed, and attention<sup>10</sup>
  - In particular, aerobic and resistance exercise (such as treadmill training), and combined physical and cognitive training, have shown short-term maintenance/improvement of global cognition, processing speed, sustained attention, mental flexibility and memory in patients with PD<sup>10</sup>

# Management of Cognitive Impairment

- Indirect strategies:
  - Treatment of comorbidities:
    - Depression
    - Anxiety
    - Apathy
    - Obstructive sleep apnea
    - Sleep disorders
    - Hearing impairment : Has been closely linked to decreased quality of life

# Depression

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- Present is 50-60% of PwP
- Depressive symptoms include sadness, lack of enjoyment, pessimism, guilt, and suicidality.
- Depression is likely to narrow the behavioral repertoire of the person with PD (PwP) and reduce warmth and reciprocity in the care partner relationship.
- Depression amplifies fatigue and reduces motivation, leading to a further reduction in independence and the capacity to perform activities of daily living.
- TREATMENT: SSRI, psychotherapy



# Apathy

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- Present in 40% of PwP
- Apathy includes both loss of motivation and loss of emotionality.
- This is manifested in diminished goal-directed activity and reduced spontaneous or evoked emotional display.
- Lack of desire to engage or find the meaning of activities in social engagements
- Lose interest in projects they used to enjoy
- Become more sedentary
- Persons who are more severely affected will require their care partners to structure their daily routine and may even need prompting to complete basic tasks such as washing or brushing of teeth.
- TREATMENT:
  - Mirapex and Exelon have been shown to reduce apathy
  - SSRI's make increase apathy ( decreased dopamine receptor binding in frontal-striatal circuit)

# Anxiety

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- Present in 50% of PwP
- Anxiety often precipitates avoidance of triggering situations, stigma, manifesting as agoraphobia, which is a profound fear to leave the home.
  - This may include a fear of the unexpected and unpredictability of their symptoms.
  - Fear of when their medications will lose effect and they will “freeze.”
  - Panic disorder
- However, anxiety may also lead to excessive reliance on the care partner for reassurance and supervision, a form of conditioned safety behavior that can be overwhelming for the care partner. In extreme situations, PwP are intolerant of being alone and become distressed when the care partner is out of sight.

TREATMENT: SSRIs, benzodiazepines, psychotherapy

# Impulse Control Disorders



- One of the most challenging behaviors that may be part of PD
- Can lead to embarrassment and difficulty talking to medical professionals and/or social supports about these issues
- Compulsive behaviors such as pathological gambling, hypersexuality, binge-eating, compulsive shopping, and dopamine dysregulation are relatively common complications of PD treatment.
- The behaviors themselves, even if transient, may have lasting financial, social, and legal consequences that can add to the burden on a household.

## TREATMENT:

- Re-evaluating dopaminergic medications and doses can help alleviate this
- Can also happen in the context of Deep Brain Stimulation (DBS) treatment

# Psychosis

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- These symptoms may be caused by dopaminergic medications or disease itself
  - In approximately 50% of PwP 5-10 years after diagnosis
- Hallucinations
  - Visual
    - Seeing people inside the home
    - Deceased family members
    - Animals running around the floor
  - Auditory
    - Voices in other rooms
    - Voices calling out for them
- Delusions
  - Intruders coming into home
  - Constantly watching outside their windows
  - Theft
  - Jealousy

TREATMENT: Pimavanserin (Nuplazid)

# Rural Communities and Parkinson's Disease

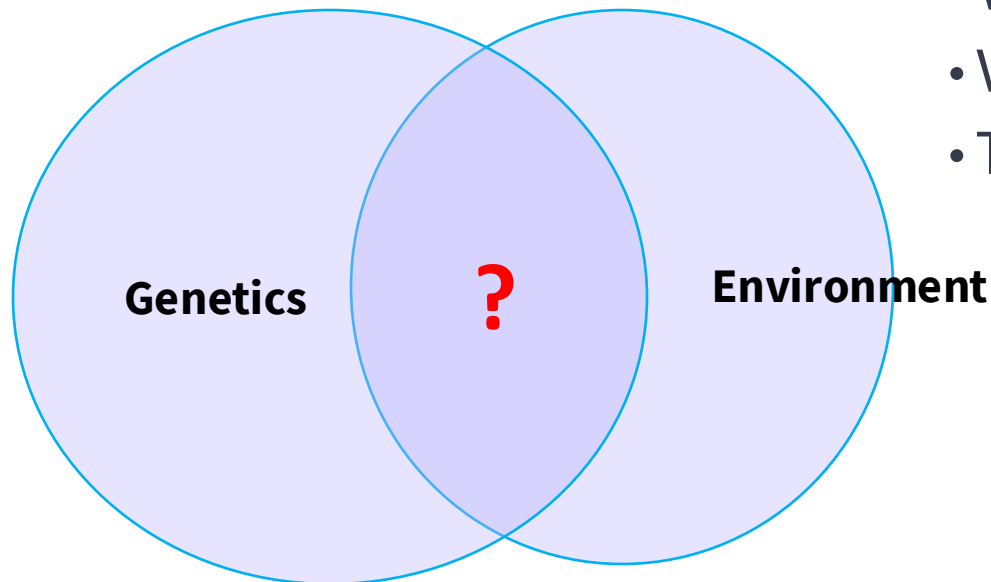
# Causes of Parkinson's Disease

- 5-15% of genetic aberrations

- More than 20 genes have been identified
- Most PwP do not have an identified gene mutation

- Environmental Risk Factors

- Pesticides/Herbicides
- Air pollution
- Agent Orange
- Welding (Manganese, copper, iron)
- Well water
- Traumatic brain injury



# Parkinson's Disease in Washington State

- As of 2018, Washington state has a rate of PD 14% higher than the national average, believed to be secondary to its agricultural economy in the central and eastern regions of the state and subsequent exposure to environmental factors that have been associated with PD <sup>11</sup>
- In 2022, the EPA banned the use of chlorpyrifos (apple orchards)
- Younger-onset (<65 years of age) are associated with environmental factors <sup>12-15</sup>.
  - Consumption of well water
  - Agricultural chemical exposure
  - Residing in rural regions

# Parkinson's Disease in Washington State

## Parkinson's Disease Mortality by State

Age-Adjusted Death Rates<sup>1</sup>

0 - 7.3

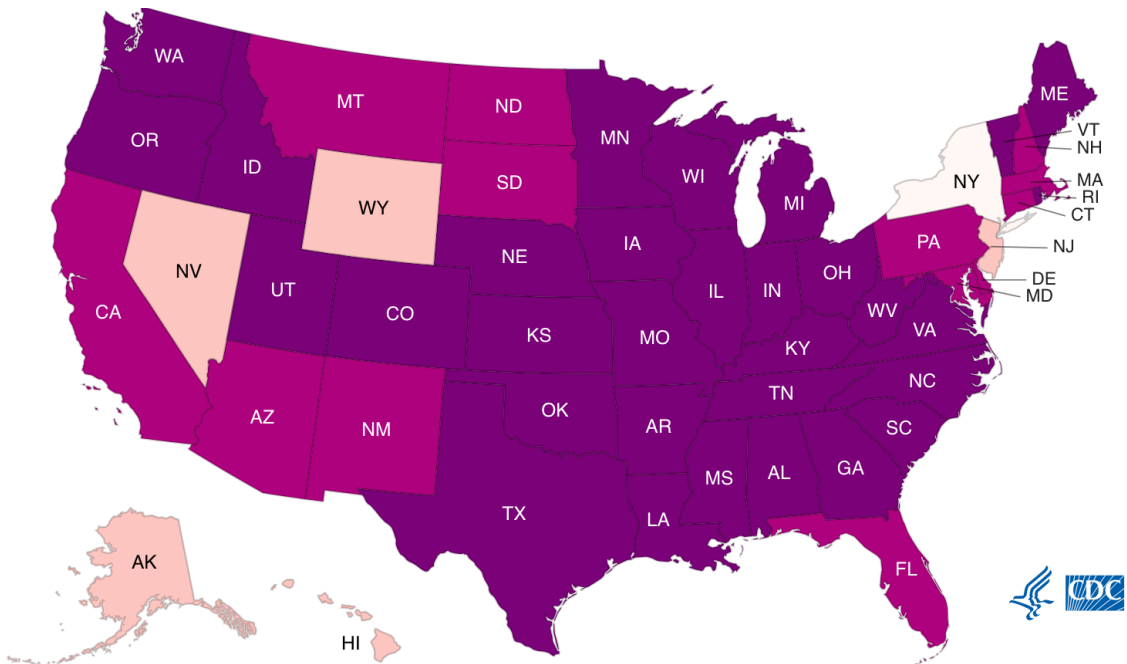
7.3 - 8.2

8.2 - 8.9

8.9 - 9.8

9.8 - 12.5

YEAR	STATE	RATE	DEATHS
2021	WA	10.7	887
2020	WA	9.5	820
2019	WA	9.4	801
2018	WA	8.7	713
2017	WA	8.6	671
2016	WA	9.1	703
2015	WA	8.7	654





# Herbicides Associated with Parkinsonism

## Paraquat <sup>16</sup>

- Herbicide continued to be widely used across the United States despite research indicating a close relationship to the incidence of PD
- In animal models has been demonstrated to replicated PD pathology including degradation of the dopaminergic system, accumulation of alpha-synuclein, and traverses the blood brain barrier.

**Table 2**  
Summary of various pesticide studies, dosage, effects, and study references involved in PD pathogenesis conducted in the different model organisms.

Pesticide	Pesticide dosage	Mode of exposure	Duration of exposure	Modelorganism	Changesobserved	conclusion	References
Paraquat	10mg/kg	oral	Four months	mice	Decreased locomotor activity, decreased striatal DA levels ↓, SOD, GSH-Px ↑, MDA	Prolonged and repeated oral administration of PQ can selectively harm the nigrostriatal dopaminergic pathway in mice.	(Ren et al., 2009)
	10-15 mg/kg	i.p.	Three sequential weeks	mouse	Diminished Striatal Dopaminergic Nerve Fibers and Reduced Striatal Dopamine Content, Reduced TH-Positive Cells and Elevated α-Synuclein Levels in SNc	Exposure to paraquat has the potential to induce changes in the conformation and aggregation of alpha-synuclein, possibly contributing to the development of synucleinopathies.	(Manning-Bog et al., 2002)
	10 mg/kg	s.c.	5 days	rats	Decreased number of tyrosine hydroxylase-immunoreactive (TH-ir) neurons in the substantia nigra	Subchronic paraquat administration initiates early-stage dopaminergic neuron degeneration and activates compensatory mechanisms involving dopaminergic, noradrenergic, serotonergic and GABAergic transmissions	(Kuter et al., 2007)
Rotenone	30 mg/kg	oral	56 days	mice	Rotenone at 30 mg/kg caused significant loss of TH-positive neurons in the substantia nigra over 28 days.	Extended oral rotenone at 30 mg/kg for 56 days caused selective DA neuron loss, motor issues, and increased cytoplasmic alpha-synuclein in the surviving neurons.	(Inden et al., 2011)
	2-3 mg/kg	s.c.	7 days	Rat	Reduced dopaminergic neurons, elevated alpha-synuclein levels in the substantia nigra compacta (SNc), and peripheral toxicity.	The higher exposure level resulted in a much more severe Parkinson's-like condition than the lower dose.	(Cannon et al., 2009b)
	2.75-3.0 mg/kg	i.p.	3 days	Rat	Diminished dopaminergic neurons and heightened alpha-synuclein clumping observed in the SNc.	The high-dose exposure exhibited a much more severe Parkinson's-like condition than the low dose.	(Cannon et al., 2009b)
	50 mg/kg	oral	14 days	Mouse	Lower numbers of TH-positive cells were seen in the SNc, along with an elevation in mitochondrial apoptosis.	Rotenone seems to induce minimal neurotoxic effects.	(Chiu et al., 2015)
Dieldrin	0.3 mg/kg	oral	every 3 days for 30 days	mice	Dieldrin exposure during development revealed significant, sex-specific differences in methylation at CpGs (DMCs) and regions (DMRs).	Dieldrin exposure during development establishes poised epigenetic states with gender-specific characteristics in early life. These poised epigenomes could influence susceptibility to later toxic exposures, potentially contributing to the emergence of neurodegenerative diseases in late life, including PD.	(Kochmanski et al., 2019a)
chlorpyrifos	5 mg/kg	s.c.	From postnatal day 11 until day 14, daily.	newborn rats	Rats exposed to CPF show a notable decrease in dopaminergic neurons and increased expression of microglia and astrocytes in the substantia nigra at both 16- and 40-days post-exposure.	Exposure to CPF during neonatal stages might result in enduring damage to dopaminergic neurons in the substantia nigra.	(Zhang et al., 2015)
Cypermethrin	15 mg/kg	i.p.	A span of 12 weeks involving 24 doses	Wistar rats	Decreased mobility, lower dopamine levels, reduced metabolites, and a decline in tyrosine hydroxylase-positive cells. Serotonin levels and glutamic acid decarboxylase-positive cells in nigrostriatal tissues remained unchanged.	Cypermethrin exposure in adulthood leads to dopaminergic neurodegeneration in rats, and postnatal exposure increases susceptibility to dopaminergic neurodegeneration upon challenge in adulthood.	(Singh et al., 2012)
Deltamethrin	0.32 mg/kg	oral	90 days	Rat	↓ GSH ↓ CAT and GPx ↑ Cyt-c, Cas-3 ↑ MDA	DLM affects overall metabolism by inhibiting various energy and structural processes, including β-oxidation, glycolysis, and Krebs cycle reactions.	(Gauzi et al., 2017b)
	7.2 mg/kg	IP	2 weeks	Rat	↑ MDA ↓ SOD, CAT, GPx activities ↓ AChE activity	Deltamethrin's effect on brain tissues has the potential to induce dysfunction in the brain.	(Saoudi et al., 2017)

# Why are Hispanic-Latino's at Increased Risk for PD?

- PD was twice as likely to occur if living close to the ambient of Paraquat <sup>18</sup> (also known as pesticide drift)
- Risk was greater if exposure occurred in the 10-20 years prior to PD diagnosis

Table 1. Top five California communities with the most paraquat sprayed from 2017 to

2021					
City name	County	Population	Paraquat sprayed (lbs)	Latino population rate	Poverty rate
Shafter	Kern	21,282	71,691	82%	23%
Wasco	Kern	27,731	45,774	87%	21%
Delano	Kern	50,843	14,204	76%	20%
McFarland	Kern	14,019	11,171	90%	27%
Corcoran	Kings	22,837	10,068	74%	28%

Source: EWG, from [California Department of Pesticide Regulation](#)

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Original article



## Original article

### Agricultural paraquat dichloride use and Parkinson's disease in California's Central Valley

Kimberly C Paul <sup>1,\*</sup>, Myles Cockburn, <sup>2</sup> Yufan Gong, <sup>3</sup> Jeff Bronstein <sup>1</sup> and Beate Ritz <sup>1,3</sup>

<sup>1</sup>Department of Neurology, UCLA David Geffen School of Medicine, Los Angeles, CA, USA, <sup>2</sup>Department of Population and Public Health Sciences, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA and <sup>3</sup>Department of Epidemiology, UCLA Fielding School of Public Health, Los Angeles, CA, USA

\*Corresponding author. Department of Neurology, UCLA, 73-320B CHS, CAMPUS- 177220, Los Angeles, CA 90095, USA. E-mail: kimberlp@ucla.edu

#### Abstract

**Background:** Paraquat dichloride is currently among the most widely used commercial herbicides in the USA. In the present study, we provide epidemiological assessment of ambient paraquat exposure and Parkinson's disease (PD) risk in a population-based study of PD in agricultural regions of Central California.

**Methods:** Based on 829 PD patients and 824 community controls, we assessed associations between ambient paraquat dichloride exposure and PD. We estimated residential and workplace proximity to commercial agricultural applications in three California counties since 1974 using the CA pesticide use reporting (PURI) data and land use maps. We evaluated any, duration and average intensity (pounds (0.45 kilograms) per acre per year) of exposure for paraquat in four time windows.

**Results:** Ambient paraquat exposure assessed at both residence and workplace was associated with PD, based on several different exposure measures. The PD patients both lived and worked near agricultural facilities applying greater amounts of the herbicide than community controls. For workplace proximity to commercial applications since 1974, working near paraquat applications every year in the window (odds ratio (OR) = 2.15, 95% confidence interval (CI) = 1.46, 3.19) and a higher average intensity of exposure (per 10 pounds (4.54 kilograms), OR = 2.08, 95% CI = 1.31, 3.38) were both associated with an increased odds of PD. Similar associations were observed for residential proximity (duration: OR = 1.91, 95% CI = 1.30, 2.83; average intensity: OR = 1.72, 95% CI = 0.99, 3.04). Risk estimates were comparable for men and women, and the strongest odds were observed for those diagnosed at ≤60 years of age.

**Conclusion:** This study provides further indication that paraquat dichloride exposure increases the risk of Parkinson's disease.

**Keywords:** Parkinson's disease, paraquat, California, epidemiology, PUR, agriculture.

#### Key Messages

- This epidemiological study assessed ambient paraquat exposure based on historical pesticide application records and Parkinson's disease risk, using a population-based case-control approach in agricultural central California.
- We assessed associations between paraquat dichloride exposure, estimated based on residential and workplace proximity to commercial agricultural applications in three California counties since 1974, and Parkinson's disease.
- Higher levels of ambient paraquat exposure at either residence or workplace was associated with Parkinson's disease risk, based on several different exposure measures and exposure windows.
- This study provides further evidence that paraquat dichloride exposure increases the risk of Parkinson's disease.

#### Introduction

Paraquat dichloride, commonly known as paraquat, is currently one of the most widely used commercial herbicides in the USA.<sup>1</sup> It is a quick-acting weed killer also used for desiccation purposes, which acts by killing green plant tissue on contact through inhibiting photosynthesis with redox-cycling activity inducing necrosis.<sup>2</sup> Its strong redox-cycling potential has been documented for nearly a century, an activity that is also highly toxic to animals and humans.<sup>3</sup> Paraquat can undergo cyclic oxidation/reduction reactions, with each cycle generating a highly reactive superoxide radical.<sup>4</sup>

Paraquat was initially scrutinized for its potential to cause Parkinson's disease (PD) due to its structural similarity to MPP<sup>+</sup> (1-methyl-4-phenylpyridinium), the toxic metabolite of MPTP (1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine), which was found to induce parkinsonism in humans in 1983.<sup>5</sup> This was followed by an ecological study in Quebec in 1987, which attributed differences in regional PD prevalence to soil and water contamination from agricultural pesticides, with paraquat being among those most prominently used.<sup>6</sup> Since then, at least 10 epidemiological studies have linked exposure to PD and a meta-analysis of 13 case-control studies with 3231 patients and

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# Parkinson's Disease in the Hispanic-Latino Community

Health disparities exist in Hispanic-Latino people with Parkinson's (PWP) <sup>19</sup>:

- Experience more severe motor symptoms, but take less medication
- Experience greater depressive/psychosis symptoms
- Worse health related quality of life
- Perceived discrimination worsens these symptoms

# Parkinson's Disease in the Hispanic-Latino Community

A study from rural California found <sup>20</sup>:

- N=138 Hispanic-Latino, 31% reported a history of working in farming communities
- Hispanic-Latino PWP present 5 years younger and with more severe motor symptoms than Caucasians
- Were suboptimally treated with medication despite severity of motor symptoms and severe disability
  - Limited access to healthcare, beliefs about medication
- Younger at the time of death
- Most held long term jobs in the farming industry

# Health-Related Quality of Life

Across PD centers of excellence (n=495; 5.8%) <sup>21</sup>

- 10% use speech and physical therapies (vs. 17% white)
- Hispanic-Latino and African Americans had lower cognitive scores
- Hispanic-Latino and African Americans less likely to be prescribed antidepressants despite higher rate of symptoms
- Hispanic-Latino worse mobility and activities of daily living

# Parkinson's Disease in the Hispanic-Latino Community

Hispanic-Latino PWP less likely to see an outpatient neurologist<sup>22</sup>:

- Limited access to medical specialists
- Language/Literacy barriers
- Financial barriers

**Hispanic-Latino PWP patients more likely to be undertreated for their symptoms**

# Risk of Parkinson's Disease Dementia in Hispanic-Latino Communities

- Older age of onset
- History of REM sleep behavior disorders
- Atypical (“rigid”) type of motor symptoms
- Lower MoCA scores in Hispanic-Latino populations have been linked to:
  - Lower levels of education
  - Higher levels of mood disorders
  - Increased hallucinations
  - Falls

# Diagnosing Parkinson's Disease Dementia



## •Challenges in Diagnosing Dementia

- Shame and discomfort to bring up issues to medical providers
- Dementia seen as a “normal” process in aging, especially in Hispanic-Latino communities
- Abilities to complete instrumental activities of daily living may be challenging due to language barriers and low literacy
  - Managing appointments, medications, finances, and driving
- Lower education/literacy levels and utilizing neurocognitive measures to test cognition



# Barriers for Care

Hispanic-Latino community face numerous challenges:

- Shame regarding symptoms
  - Anxiety
  - Depression
  - Autonomic: Constipation, erectile dysfunction
- Language barriers
- Low level of education, literacy
- Lack of specialist in their areas
- Lower utilization of specialty care that contributed to better health in those with PD:
  - Physical therapy
  - Occupational therapy
  - Speech and language therapy

Thank You!  
[ktorres2@uw.edu](mailto:ktorres2@uw.edu)

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