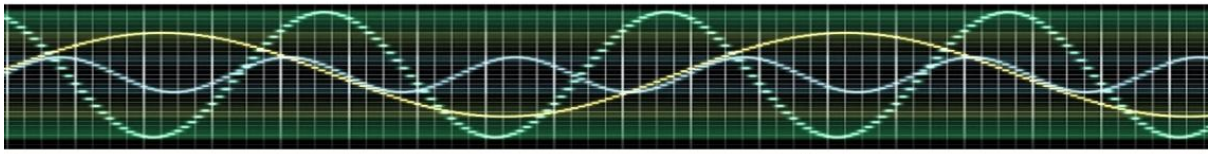
Redland Reflexology

BODY OPTIMISER - SOUND WAVE TECHNOLOGY



Diminishing Function Risk			Optimal and Balanced			Increasing Inflammation Risk		
1	2	3	4	5	6	7	8	9
Critical	Severe	Moderate	Mild	Normal	Mild	Moderate	Severe	Critical

PARKINSONS DISEASE

GROK QUERY: 17th Jan 2026

Which of the following Amino Acids and brain nerves are associated with Parkinsons disease? List from Body Chemistry Results.

For your reference see:
Body Chemistry Results/ Nutritional Analysis/ Nutritional Analysis 1/Amino Acids

Body Chemistry Results/ Physical Functionality/ Physical Functionality 2/Brain nerves

GROK RESPONSE:

Parkinson's disease (PD) is primarily characterized by the progressive loss of dopaminergic neurons in the substantia nigra, leading to reduced dopamine levels. This directly links to certain amino acids and aspects of brain function/nerves.
Associated Amino Acid From the list provided, the key amino acid strongly associated with Parkinson's disease is Tyrosine.

- Tyrosine is the direct precursor to L-DOPA (levodopa), which is converted to dopamine in the brain via the enzyme tyrosine hydroxylase (the rate-limiting step in dopamine synthesis).
- The degeneration in PD disrupts this pathway, leading to dopamine deficiency.
- Tyrosine supplementation has been studied in PD (e.g., for related issues like orthostatic hypotension), and disruptions in tyrosine metabolism are implicated in PD pathogenesis.
- Phenylalanine (which converts to tyrosine) is sometimes mentioned in related contexts, but tyrosine is the most directly relevant.

Other amino acids from your list (e.g., Glutamic Acid, Aspartic Acid, Glycine, or branched-chain ones like Leucine, Isoleucine, Valine) show some changes in serum levels or metabolic studies in PD patients, but they are not as centrally or causally linked as tyrosine/dopamine pathway.Here are some visual examples of the dopamine synthesis pathway involving tyrosine:(These illustrate the biochemical pathway from tyrosine → L-DOPA → dopamine, highlighting its relevance to PD.)Associated Brain Nerves / FunctionsFrom the list, the most consistently associated with Parkinson's disease are:

- Cranial Nerve 1, Olfactory — Olfactory dysfunction (loss of smell) is one of the earliest and most common non-motor symptoms in PD, often appearing years before motor symptoms. Alpha-synuclein pathology frequently starts in the olfactory system.
- Cranial Nerve 10, Vagus — The vagus nerve (especially its dorsal motor nucleus) is implicated in early PD pathology (Braak staging suggests pathology may begin in the gut and ascend via the vagus to the brainstem). It contributes to autonomic symptoms like gastrointestinal issues, which are common in PD.

Other mentions in research include:

- Cranial Nerve 9, Glossopharyngeal — Involved in pharyngeal sensory issues and dysphagia (swallowing problems) in PD.
- Cranial Nerve 7, Facial — Related to facial masking/expressionless face (rigidity of facial muscles).
- Cranial Nerve 5, Trigeminal — Some studies link it to sensory changes or olfactory interactions.

However, the strongest and earliest associations are with Olfactory (CN 1) and Vagus (CN 10).Items like Cerebral Arteriosclerosis (vascular issues, not primary in idiopathic PD), most other cranial nerves (e.g., 2, 3, 4, 6, 8, 11, 12), or Sympathetic/Parasympathetic NS Function (affected secondarily via autonomic involvement, but not as directly as vagus), and Memory Index (ZS) (likely refers to memory/cognitive testing; memory issues occur in advanced PD but are not a core defining feature like motor or olfactory loss) are not primary associations.PD's core pathology affects dopaminergic systems, but early involvement often includes olfactory and vagal pathways. If you're experiencing symptoms or considering this for personal reasons, consult a neurologist for proper evaluation. Thank you!

GROK QUERY: 17th Jan 2026

Are imbalances in any of the following nerves an indication of Parkinsons disease.? List from Body Organ Results.

For your reference see:
Body Organ Results/Nerves Head
Body Organ Results/Nerves Upper Body

GROK RESPONSE

Yes, imbalances or dysfunction in certain nerves from your list can be associated with or indicative of Parkinson's disease (PD), though PD primarily affects the brain's dopaminergic system rather than causing direct, isolated cranial nerve palsies like those seen in strokes or tumors. Instead, PD involves alpha-synuclein pathology (Lewy bodies/neurites) that can affect specific cranial nerves and autonomic pathways early in the disease process, often before classic motor symptoms appear. This leads to non-motor symptoms like loss of smell, swallowing issues, or autonomic problems.
Most nerves in your list (e.g., oculomotor, trochlear, abducens, optic, vestibulocochlear, accessory, hypoglossal, phrenic, recurrent laryngeal, intercostal, or cervical spinal nerves C1–C8) show no strong or primary association with PD. These are typically spared or only secondarily affected in advanced stages due to general rigidity/muscle changes, not direct nerve imbalance. PD does not usually cause classic cranial nerve palsies (e.g., eye movement paralysis from CN III/IV/VI or hearing loss from CN VIII).Nerves Strongly Associated with

Parkinson's DiseaseThese are the most relevant from your list, often showing early involvement via pathology in their nuclei or pathways (Braak staging starts in the olfactory bulb and dorsal motor nucleus of the vagus):

- R Olfactory Nerve Branches and L Olfactory Nerve Branches (Cranial Nerve I)
  Olfactory dysfunction (reduced or lost sense of smell) is one of the earliest and most common non-motor symptoms in PD, often appearing years before tremors or stiffness. Pathology frequently begins in the olfactory bulb and anterior olfactory nucleus.
- R Vagus Nerve and L Vagus Nerve (Cranial Nerve X)
  The vagus nerve (especially its dorsal motor nucleus) is a key early site of alpha-synuclein pathology. It contributes to autonomic symptoms like gastrointestinal issues (constipation), swallowing difficulties (dysphagia), and cardiovascular problems (e.g., orthostatic hypotension). The vagus is implicated in the "gut-to-brain" spread hypothesis of PD.
- R Glossopharyngeal Nerve and L Glossopharyngeal Nerve (Cranial Nerve IX)
  Involved alongside the vagus in pharyngeal sensory issues, taste loss, and swallowing problems (dysphagia). Pathology affects sensory branches in the pharynx, contributing to aspiration risk.

Other Nerves with Some (Secondary or Less Direct) Links

- R Facial Nerve and L Facial Nerve (Cranial Nerve VII)
  Facial masking (reduced expression, "masked face") is common due to rigidity and bradykinesia affecting facial muscles, not a true nerve palsy. Emotion recognition deficits and subtle facial changes can occur.
- R Trigeminal Nerve V, R Ophthalmic Nerve V1, R Maxillary Nerve V2, R Mandibular Nerve V3, and left-sided equivalents
  The trigeminal system is generally not impaired in PD, even with olfactory loss. Some studies note minor sensory changes or orofacial pain, but trigeminal dysfunction is not a hallmark indicator.

Nerves Not Typically Indicative of PD

- Eye movement nerves (Oculomotor, Trochlear, Abducens) — PD can cause subtle oculomotor changes (e.g., saccadic abnormalities), but not frank palsies.
- Optic Nerve — Visual issues occur (e.g., contrast sensitivity), but not optic nerve imbalance.
- Vestibulocochlear — Hearing/tinnitus possible in advanced PD, but not primary.
- Accessory, Hypoglossal — Tongue/jaw weakness from rigidity, not direct nerve damage.
- Sympathetic Nerves, Cervical Spinal Nerves C1–C8, Intercostal Nerves, Phrenic, Recurrent Laryngeal — Autonomic dysfunction (sympathetic involvement) is common (e.g., orthostatic issues), but not specific to these as "imbalances." Cervical spinal changes are more related to posture/rigidity than direct nerve pathology.

In summary, the strongest indicators from your list are olfactory (CN I), vagus (CN X), and to a lesser extent glossopharyngeal (CN IX) branches. These often signal early/prodromal PD. If you're experiencing symptoms like loss of smell, swallowing difficulties, or autonomic issues (e.g., constipation, dizziness on standing), it's worth discussing with a neurologist specializing in movement disorders for evaluation (e.g., via smell tests, autonomic testing, or imaging). PD diagnosis relies on clinical criteria, not just nerve findings. Thank you!

GROK QUERY: 17th Jan 2026

Are imbalances in any of the following brain nerves an indication of Parkinsons disease.? List from Body Organ Results.

For your reference see:
Body Organ Results/Nerves Brain

GROK RESPONSE

Yes, imbalances (such as atrophy, volume loss, hyperechogenicity, iron accumulation, or dopaminergic dysfunction) in several structures from your list are strongly associated with and can serve as key indicators of Parkinson's disease (PD). PD's core pathology involves progressive degeneration in the basal ganglia and brainstem, particularly dopaminergic neuron loss, which is detectable via neuroimaging (e.g., MRI, transcranial sonography, PET/SPECT) and contributes to motor symptoms like bradykinesia, rigidity, and tremor.PD often shows asymmetric involvement early on (more pronounced on one side, usually contralateral to the more affected body side), reflecting uneven nigrostriatal degeneration. This asymmetry can be seen in the substantia nigra and striatum (putamen/caudate).Strongly Associated Structures (Primary Indicators of PD)These are central to PD pathology and frequently show changes like neuronal loss, Lewy body accumulation, iron deposition, or reduced dopamine function:

- R Substantia Nigra and L Substantia Nigra — The hallmark of PD is degeneration of dopaminergic neurons in the substantia nigra pars compacta (especially ventrolateral). This leads to dopamine deficiency in the striatum. Neuroimaging often shows hyperechogenicity (via transcranial sonography), loss of the "swallow tail" sign on MRI, or reduced dopamine uptake. Asymmetry is common early, with greater loss contralateral to dominant symptoms.
- R Putamen and (implied) left putamen (though not listed separately) — The putamen receives major projections from the substantia nigra (nigrostriatal pathway) and shows significant dopamine depletion, volume reduction, and microstructural changes. It's often more affected than the caudate in motor-dominant PD.
- R Caudate Nucleus and L Caudate Nucleus — Part of the striatum; shows volume loss and dopamine changes, though less severely than the putamen in early PD.
- R Medial Globus Pallidus and L Medial Globus Pallidus (internal segment, GPi) — Involved in basal ganglia output; shows secondary changes like iron accumulation or altered connectivity due to upstream nigrostriatal loss.
- R Lateral Globus Pallidus — External segment (GPe); affected in circuit dysfunction.

Structures with Some/Secondary AssociationThese may show changes in advanced PD or related non-motor symptoms, but are not primary indicators:

- R Amygdaloid Body and L Amygdaloid Body (amygdala) — Lewy pathology and neurodegeneration occur, linked to emotional/cognitive issues (e.g., anxiety, dementia in later stages).
- R Hippocampus and L Hippocampus — Atrophy and alpha-synuclein pathology in advanced PD, contributing to memory/cognitive decline (especially in PD dementia).
- R Hypothalamus and L Hypothalamus — Degeneration in multiple hypothalamic nuclei, associated with autonomic/sleep/endocrine symptoms.
- R Mammillary Body and L Mammillary Body — Part of limbic circuits; indirect involvement via hypothalamic or fornix changes.
- Brain Stem Center, Pons, R Medulla Oblongata, and L Medulla Oblongata — Early Braak staging starts in medulla (e.g., dorsal motor vagus nucleus), progressing to pons (locus coeruleus). Subtle atrophy or pathology possible.

Structures Not Typically Indicative of PDThese are generally spared or show minimal/no primary changes in idiopathic PD (changes, if any, are secondary or seen in mimics like MSA/PSP):

- Ventricular structures (R Lateral Ventricle, L Lateral Ventricle, Third Ventricle, Fourth Ventricle, Cerebral Aqueduct, R Interventricular Foramen)
- White matter tracts (R Fornix Crura, R Fornix Columns, L Fornix Body, L Fornix Crura)
- Colliculi (R Superior Colliculus, R Inferior Colliculus, L Superior Colliculus, L Inferior Colliculus)
- Other: Gracile Tubercle, R Cuneate, R Olive, L Olive, Pyramid, Pituitary Gland, Pineal Gland, Outer Dura, Cerebellum, Cerebrum, Corpus Callosum, R Thalamus, L Thalamus, L Subthalmic Nucleus (subthalamic nucleus is targeted in DBS but not primarily degenerated in PD)

In summary, the strongest indicators from your list are the substantia nigra (both sides, often asymmetric), putamen, caudate nucleus, and globus pallidus structures — these reflect the nigrostriatal dopaminergic deficit central to PD. Imbalances here (e.g., on MRI showing atrophy or sonography showing hyperechogenicity) support diagnosis, especially when combined with clinical symptoms. If this relates to personal imaging or symptoms, a movement disorder specialist can interpret findings in context (e.g., via DaTscan for dopamine function). Thank you!