



NUMBER 10

# CYTOPLAN

THE HEALTH INFORMATION SERIES



# Parkinson's Disease

FOR PROFESSIONAL USE ONLY

This publication is for information only and is not intended as a replacement for medical advice that is based on individual circumstances.



# CYTOPLAN

NATURE MEETS SCIENCE

Cytoplan celebrates 26 years in the field of food-based supplementation and from the moment of conception to the present day we have promoted the philosophy that nutrients are best delivered to the body "in the same form as food".

The philosophy and message of Cytoplan was founded on the simple logic that our bodies are designed to eat food and utilise its components for the maintenance of life. The ultimate goal of Cytoplan is to "improve the health of the nation" by supplying supplements in a bio-effective form for optimal absorption and utilisation.

SINCE



1990

## KEY TO SYMBOLS



= Raw Food



= Food State™



= Tablets



= Powder



= Suitable for  
Vegans



= Wholefood



= Capsules



= Liquids



= Suitable for  
Vegetarians



= Amount in  
container



= Organic Registered

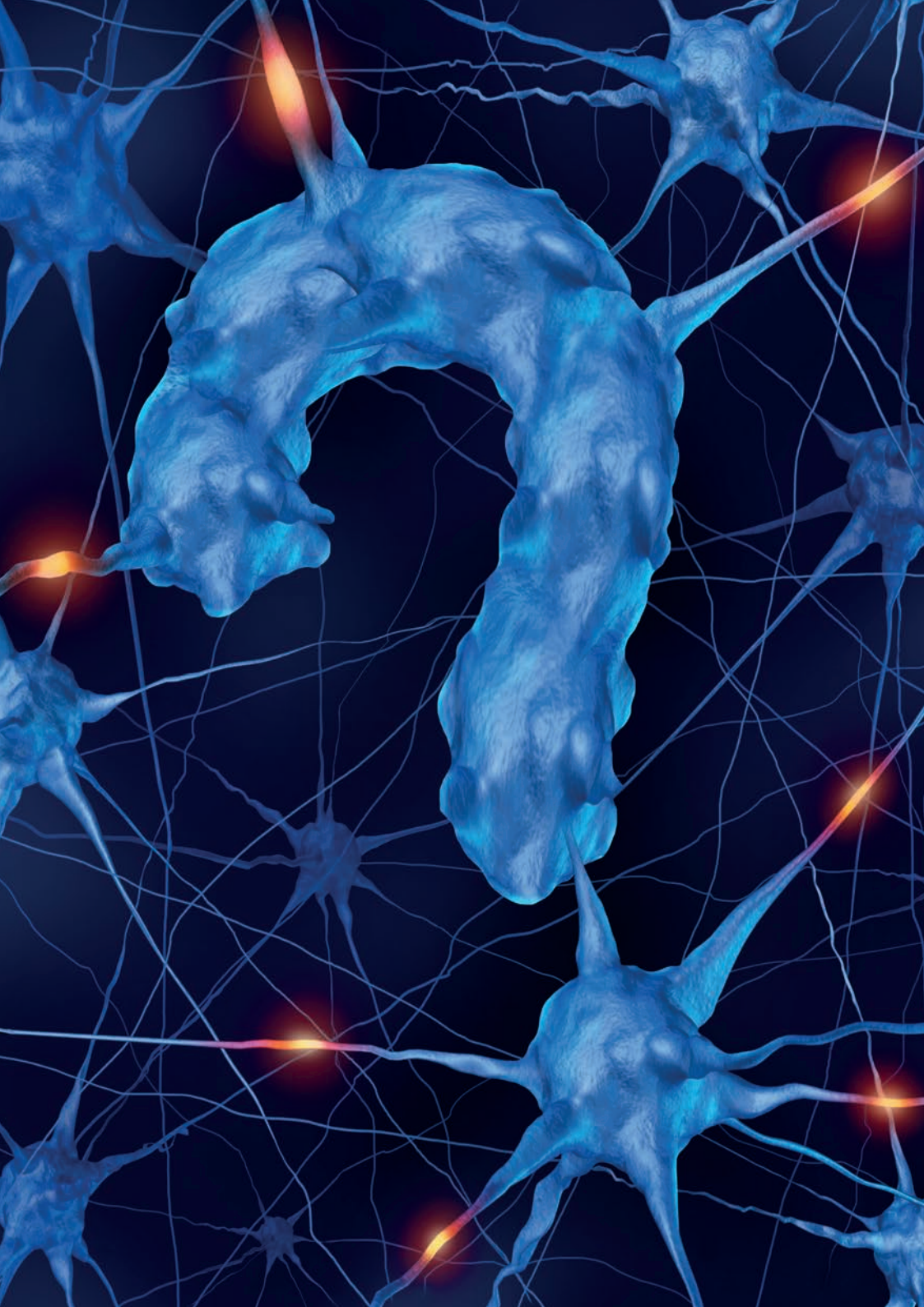
## **Also available in the Cytoplan Health Information Series:**

1. IBS
2. Candida
3. Arthritis
4. Endometriosis
5. Osteoporosis
6. Metabolic Syndrome
7. Eczema and Allergies
8. M.E.
9. How to protect your eyesight
10. Parkinson's Disease
11. Skin
12. Personality
13. Fertility and Conception
14. Cholesterol and Hypertension
15. Childrens Supplements/Nutrition
16. What constitutes a good diet

Cytoplan Limited, Unit 8, Hanley Workshops, Hanley Swan, Worcestershire WR8 0DX  
Telephone: 01684 310099 Fax: 01684 312000 Email: [info@cytoplan.co.uk](mailto:info@cytoplan.co.uk)

**[www.cytoplan.co.uk](http://www.cytoplan.co.uk)**

Copyright Cytoplan Limited 2016



## Parkinson's disease

Parkinson's was first identified by James Parkinson in 1817, he wrote a paper entitled "The Shaking Palsy". He was the first person to accurately record the symptoms of the condition, then described as Paralysis agitans.

Later, the French neurologist Dr Jean-Martin Charcot added further symptoms to the description and named the condition "Parkinson's Disease" in recognition of the work of Dr Parkinson.

## Pathophysiology

Parkinson's disease is a progressive neurological disorder in which initial pathology leads to the degeneration of neurons in the substantia nigra. These neurons are responsible for the production of the neurotransmitter dopamine, a critical signalling molecule involved in the control of movement and emotional response. Levels of dopamine continue to fall slowly during the disease progression; however, the body appears to continue to function with a reduced availability of dopamine. It is believed that symptoms of Parkinson's appear when approximately eighty percent of dopamine has been lost. Autopsy shows the presence of Lewy bodies within the substantia nigra. Lewy bodies are normal cellular proteins which have been marked for degradation but in Parkinson's they appear to escape normal degradation processes.

**A condition known as Parkinsonism produces symptoms similar to those of Parkinson's disease. This can be a result of other disorders, a response to certain prescription drugs or exposure to toxins.**

## Onset of symptoms

Symptoms of Parkinson's disease often first appear after the age of 50, although later onset or diagnosis is common (average age is approximately 60). Early onset of Parkinson's does arise and is referred to in those diagnosed before the age of 40. Juvenile Parkinson's, diagnosed before the age of 18, does occur but is rare. The condition is more common in men than in women.

## Symptoms

Often Parkinson's is diagnosed by observing a collection of symptoms associated with the disease. However, these symptoms can differ greatly from person to person; slow onset, means that symptoms may have been present for some time before a diagnosis is made. Not all symptoms will be present in an individual, although others often develop as the condition progresses.

The tremor of Parkinson's is perhaps one of the most well-known symptoms, although not always present. Early appearance of tremor may just involve the hand. As the condition progresses, tremors may involve the arms, legs, head or jaw. The tremor is more noticeable when relaxing and reduced when moving or sleeping.

Bradykinesia, which simply means 'slowness of movement', is also a recorded symptom: patients experience difficulty initiating movement (akinesia), often referred to as freezing, or movement taking longer to perform. Walking can become progressively difficult and slow, with patients needing to make a conscious effort to lift their feet to prevent shuffling or falling.

Mobility problems occur due to stiffness or rigidity of muscles, which can also result in falls; facial expression may be affected; speech may become quiet or rapid; and small or difficult handwriting is noted by some.



Additional symptoms include:

- Low mood and/or anxiety
- Poor memory and reasoning ability
- Psychotic symptoms such as hallucinations or delusions
- Gastro-intestinal symptoms particularly constipation
- 'Pill-rolling' – movement between thumb and forefinger
- Stooped posture

There is no specific test currently available that can confirm the presence of the condition: diagnosis is made on symptom observations and after tests have eliminated other potential causes or conditions.

NHS National Guidelines advise that Parkinson's should be suspected in people presenting with tremor, stiffness, slowness and balance or gait problems, doctors should refer patients prior to treatment to a specialist in differential diagnosis of Parkinson's disease. The condition should be diagnosed clinically and based on the UK Parkinson's Disease Society Brain Bank Criteria. Regular reviews of the diagnosis should be undertaken.

## Medical Treatment

Currently there is no cure for Parkinson's disease: treatment focuses on the use of medication to control the symptoms with a variety of drugs including:

- Drugs that replace dopamine
- Drugs that mimic the action of dopamine
- Drugs that prevent the breakdown of dopamine
- Drugs which inhibit the action of acetylcholine

As symptoms of the condition vary between individuals, patients' responses to drugs can be variable and it can take time to find the most suitable. Early response to treatment is usually good; as the condition progresses symptoms may be more difficult to resolve and increased dosage and/or additional or different medications are used.

A number of surgical procedures are used to address symptoms such as tremor. New treatments for Parkinson's, currently in early stages, include stem cell and gene therapy.

## Aetiology

It is generally agreed that, as with most chronic conditions, Parkinson's is a multi-factorial condition with a number of factors exacerbating, contributing or leading to onset with toxic exposure, genetic predisposition and diet playing a part. The cell death within the substantia nigra has been attributed to oxidative stress, inflammation and mitochondrial dysfunction, again consistent with chronic disease onset and which can be modified by environment, diet and lifestyle. Other risk factors include:

- Damage to basal ganglia by neurotoxins, such as glutamate
- Use of MPTP (designer heroin drug)
- Ageing
- Heavy metal toxicity



- Poor phase 2 liver detoxification
- Infection of central nervous system by mycobacterium
- High homocysteine and reduced s-adenosylmethionine (SAmE)
- Nutrient deficiencies in brain of zinc, vitamin E, selenium
- Glutathione deficiency
- Increased levels of iron within the substantia nigra

## **Genetics**

Some incidences of Parkinson's suggest a genetic predisposition but the majority of cases have no indication of a hereditary link. However recent studies have demonstrated a potential epigenetic link to Parkinson's whereby environmental factors have an effect on gene expression.

## **Oxidative Stress**

One of the first biochemical changes noted in Parkinson's is the depletion of glutathione (a major intracellular antioxidant and redox regulator) within the substantia nigra, suggesting that oxidative stress is a significant factor in the onset and progression of Parkinson's. Oxidative stress can cause damage to cells and lead to inflammation and apoptosis; this can also lead to mitochondrial dysfunction which will affect the cell's ability to produce energy and therefore the function of the cell will be significantly reduced. Glutathione depletion has been linked to the aggregation of defective proteins leading to death of dopaminergic neurons and increased susceptibility to endogenous and exogenous toxins.

Antioxidants have the ability to protect cells from damage and many studies have been carried out in this area with papers dating back to 1998.

## **Environmental toxins**

Studies have identified an association between Parkinson's and exposure to environmental toxins including:

- Pesticides (organochlorines, carbamates, paraquat, maneb, rotenone, pyrethroid and diethyldithiocarbamate)
- Certain trace metals including copper, iron and manganese as well as heavy metals such as lead
- Solvents

## **Phase 2 liver detoxification**

In addition to identifying and reducing exposure to environmental toxins, it is important to consider the individual's capacity to biotransform toxins via the detoxification pathways. A reduction in phase 2 detoxification is particularly relevant in Parkinson's. Studies have shown that patients have reduced capacity to detoxify xenobiotics in particular. It is thought that reduced detoxification function can increase susceptibility to neurotoxins. Reduced liver detoxification is also associated with a higher level of free radicals and inflammatory cytokines.

## **Iron**

Research into the aetiology of Parkinson's has found disrupted iron metabolism together with the presence of increased iron levels within the substantia nigra of some Parkinson's disease patients. Oxidative damage to cells, resulting from the Fenton reaction of iron and the accumulation of iron in the substantia nigra, is associated with neuronal loss and Lewy body pathology. (The Fenton reaction = the oxidation of organic substrates by iron(II) and hydrogen peroxide).

Intake of dietary iron and supplementation should be considered, as research has identified an association in men between high dietary iron intake and increased risk of Parkinson's disease via oxidative stress.



## Diet

Studies have shown that patients with Parkinson's tend to consume fewer vegetables, more animal fat and protein and have a higher carbohydrate consumption than controls.

A dietary study carried out with both males and females with a follow up after 16 years found that "Dietary patterns with a high intake of fruit, vegetables, legumes, wholegrains, nuts, fish and poultry, a low intake of saturated fat and moderate alcohol may protect against PD".

When comparing a "prudent" diet (high intakes of fruit, vegetables and fish) with a "Western diet" (high intake of red meat, processed foods, refined grains, desserts and high fat dairy), the prudent diet was inversely associated with the risk of Parkinson's disease. A similar result was shown when the Mediterranean diet was compared with a Western diet. The Mediterranean diet also had a positive effect on cardiovascular disease, cancer and the risk of Alzheimer's disease. The additional beneficial impact of the Mediterranean style diet was improved nutritional balance of diet and gastrointestinal function.

Caloric restriction has been indicated for many conditions associated with ageing and could also be useful in prevention and potential therapy for Parkinson's disease. Research suggests that caloric restriction can extend lifespan and increases resistance of the brain to insults that involve metabolic compromise and excitotoxicity. One potential mechanism for caloric restriction is also enhanced production of antioxidants via intrinsic pathways.

Much of the interest in neurological conditions such as Parkinson's and nutrition has looked at the impact of exposure to free radicals or pro-oxidants and the corresponding lack of antioxidants in the diet. Research has shown certain plant derived antioxidants can inhibit dopamine oxidation, especially ascorbic acid (vitamin C).

Patients on L-Dopa medication should be aware that protein or amino acids can inhibit its absorption in the digestive tract. Therefore, protein should be consumed at least 2 hours away from taking L-Dopa medication. B6 should not be taken in conjunction with L-Dopa medication as it can convert L-Dopa into dopamine before it has crossed the blood brain barrier; dopamine is unable to cross this barrier. It is therefore often recommended to take medication on an empty stomach - wait for it to 'kick in' before consuming a meal and then waiting 2 hours before taking medication again (this should be discussed with the responsible Medical Practitioner).

## Nutrients

### Flavonoids

The benefits of flavonoids as potential antioxidants and anti-inflammatory agents with free radical scavenging abilities have been documented considerably over the years. It is therefore no surprise to discover the potential of flavonoids for conditions such as Parkinson's disease. Research has identified flavonoids as modulators of brain function and the central nervous system, providing protection for neurons and suppressing neurological inflammation.

Dr Jeremy Spencer of the University of Reading has carried out considerable research on the correlation between the consumption of diets rich in fruits and vegetables and a decreased risk of neurodegenerative disorders. His 2007 review suggested that dietary phyto-chemicals, in particular flavonoids, may exert beneficial effects in the central nervous system by protecting neurons against stress-induced injury, suppressing neuro-inflammation and by promoting neuro-cognitive performance.

We should consider the combined effects of phyto-nutrients and not follow many of the scientific trends in looking for single substance success; however here we list specific flavonoids identified in research as beneficial to Parkinson's disease.

- Green tea; catechins found in green tea are considered to offer benefits from metal chelating, antioxidant and anti-inflammatory activity in respect of neuronal death in the substantia nigra.
- Resveratrol and quercetin have been shown to protect dopaminergic neurons, diminishing apoptotic neuronal cell death by acting on the expression of pro- and anti-apoptotic genes.
- The mulberry fruit, which provides anthocyanins, has demonstrated neuroprotective effects *in vitro* and *in vivo* for Parkinson's disease models. The mulberry was found to significantly protect the cells from neurotoxicity in a dose dependent manner.
- Curcumin has demonstrated the ability to reduce reactive oxygen species and protect cells against apoptosis; curcumin's ability to cross the blood brain barrier makes it particularly interesting for neurological conditions, with potential therapeutic value for treating Parkinson's and other neurodegenerative disease. Studies are currently looking at the potential for curcumin to reduce  $\alpha$ -synuclein (a protein which is the major constituent in Lewy bodies) aggregation which is thought to be associated with dopaminergic neuron loss.
- In an animal study, researchers identified extracts of tangerine peel (described as 'rich in polymethoxylated flavones'), cocoa (rich in procyanidins) and red clover (providing isoflavones) as protective of the dopaminergic neurons.
- Flavonoids, particularly polyphenols have demonstrated membrane stabilising properties. It has been shown that restoring the lipid content, and therefore stabilising, the inner mitochondrial membrane is important for protecting both mitochondrial and neural function.

## **Glutathione**

An important antioxidant produced in the body, the levels are known to fall with ageing and have been shown to be reduced within the substantia nigra in patients with Parkinson's. Nutrients such as N-acetylcysteine and alpha-lipoic acid are precursors for glutathione and may be of some benefit. Taking other antioxidants alongside is also of benefit in order to conserve glutathione, particularly high dose vitamin C.

## **Coenzyme Q10**

In a double blind trial CoQ10 slowed functional decline in Parkinson's disease. Patients selected were those in the early stages of disease, not requiring treatment for their condition. At follow-up less disability was found to have developed in those receiving CoQ10, with the greatest benefits linked to the higher doses of 1200mg/day.

## **Vitamin B6, folate and B12**

A review on dietary intake of vitamins and the incidence of Parkinson's disease indicated a low intake of B6 being independently associated with an increased risk of the disease. This was via the potential for accelerated dopaminergic cell death resulting from increased homocysteine. Increased homocysteine levels are also associated with insufficient levels of folate and B12.

## **Vitamin E**

Research identified that the risk of Parkinson's disease was significantly reduced among men and women with high dietary intake of vitamin E from foods. However, results did not concur when vitamin E supplementation was taken; the researchers considered that other constituents in vitamin E rich food may be protective. We should, of course, consider the form of delivery for the vitamin E used; vitamin E occurs in nature as tocopherols and tocotrienols of which there are 4 of each. Most supplements only include alpha-tocopherol i.e. one of the 8 constituents. In addition, in food vitamin E is naturally complexed

with additional support nutrients such as bioflavonoids, carotenoids, lipids and more. The form of nutrient and/or additional co-factors would appear of relevance to these study results.

## **Vitamin D**

Studies of vitamin D levels have shown them to be insufficient in 55% of patients with Parkinson's disease in comparison to 'healthy people'. The substantia nigra contains high levels of vitamin D receptors; further research is required to establish at what stage the deficiency occurs and if supplementation or increased exposure to sunlight may be beneficial.

## **Ginkgo biloba**

Research, both *in vitro* and *in vivo*, has shown Ginkgo biloba to have a protective effect on Parkinson's disease models. It has been shown to inhibit lipid peroxidation of cell membranes, enhance energy utilisation and have membrane stabilising and antioxidant effects.





## An Individual and Holistic Approach

Many individual nutrients have been found to have beneficial effects in Parkinson's disease; here we have discussed just a few examples. However, as the disease is multi-factorial and can differ among individuals, prevention and support for Parkinson's disease may not be obtained by any individual agent, but by administering a rationally designed combination of therapies.

### **Diet, lifestyle and environmental factors to consider for prevention and support for Parkinson's disease.**

This is a diet:

- High in vegetables and fruit, with a variety of colours in order to obtain a range of antioxidants and phytonutrients.
- With good quality healthy fats – foods high in monounsaturated fatty acids (eg avocado, olive oil, nuts and seeds) and omega 3 fatty acids (oily fish, flax and chia seeds).
- Low in inflammatory omega 6 fatty acids (animal fats, vegetable oils and margarine)
- With good quality lean protein
- Low or avoiding refined and processed carbohydrates

In addition:

- Reducing exposure to environmental pollutants by opting for organic food, using a filter for water, avoiding food and drink in soft plastic containers, taking care with choice of personal and cosmetic products as well as household cleaning products.
- Supporting digestive function with fermented and prebiotic foods (eg vegetables in particular) and considering a live bacteria supplement. Constipation is common in patients with Parkinson's disease. Dietary assistance to alleviate constipation has been shown to improve pharmacokinetics of Parkinson's drugs.

- Supporting liver detoxification pathways with adequate protein and liver supporting foods such as brassica vegetables, onions, garlic, bitter leafy greens, green tea.
- Supporting methylation pathways to reduce homocysteine with adequate intake of folate, B12 and B6.
- Increasing foods rich in vitamin E eg nuts, seeds, avocados and eggs.
- Supporting mitochondrial function if needed (eg co-enzyme Q10, alpha lipid acid, essential fatty acids and phospholipids).

**Nutrients to consider in relation to Parkinson’s disease**

Nutrient	Examples of functions in relation to Parkinson’s disease
Vitamin A	Vitamin A is a lipid antioxidant, therefore it is important for reducing oxidative stress within brain tissue which is predominantly comprised of fat
Vitamin C	Major antioxidant and recycles glutathione, aids heavy metal detoxification and important for cell membrane integrity
Vitamin B6	Co-factor for production of dopamine (caution with levo-dopa) important for homocysteine methylation
Folate	Methyl donor for homocysteine methylation
B12	Methyl donor for homocysteine methylation
Vitamin D	Anti-inflammatory properties, has been shown to be insufficient in patients with Parkinson’s
Vitamin E	Free radical scavenger and protects against lipid peroxidation
Selenium	Protects cells from oxidative stress and can chelate heavy metals particularly mercury
Zinc	Stimulates the antioxidant superoxide dismutase (SOD), protects cells from oxidative stress and contributes to normal brain function

Nutrient	Examples of functions in relation to Parkinson's disease
<b>Alpha-lipoic acid</b>	Antioxidant, supports mitochondrial function as a coenzyme for the production of energy
<b>CoQ10</b>	Cofactor in the electron transport chain therefore supports mitochondrial function and energy production and has antioxidant properties
<b>N-acetylcysteine</b>	Antioxidant, stimulates glutathione synthesis and chelates heavy metals
<b>EPA/DHA</b>	Anti-inflammatory properties (EPA precursor for anti-inflammatory prostaglandins), DHA contributes to normal brain function and development
<b>Curcumin</b>	Anti-inflammatory as blocks COX-2 expression. Also has antioxidant, antiaging and neuroprotective effects and has potential to prevent Lewy body aggregation
<b>Other flavonoids and carotenes</b>	Antioxidant properties. Flavonoids also shown to have beneficial effects on neuro-inflammation and have metal chelating properties
<b>Ginkgo biloba</b>	Enhances utilisation of oxygen and glucose, antioxidant and membrane stabilising effects

## Points to consider in the nutritional support of those diagnosed with Parkinson's disease

- Patient, Medical Practitioner and Nutritional Therapist communication to ensure that at all times the Medical Practitioner is aware and in agreement with any proposed dietary changes or supplement support.
- Potential nutrient/drug interactions.
- In the elderly, consider issues such as food preparation and difficulties with chewing and swallowing.



## RELEVANT CYTOPLAN PRODUCTS

### Multivitamin and mineral formulae

For overall health – address the basics first with all-round nutritional support and gut health support if needed. Due to the 'nutrition gap' a multi is recommended to provide a foundation for health, including brain health. For example, B vitamins for methylation; vitamin C for cell membrane health; vitamin D for anti-inflammatory benefits, trace minerals that act as enzyme co-factors including zinc important for antioxidant properties.

All our multiformulae are vegan and contain good levels of B vitamins and antioxidants. The full range can be viewed on our website or ask for a copy of our catalogue.



### CoQ10 Multi

A comprehensive wholefood multi with higher levels of B vitamins, vitamins C and D and trace minerals. Contains coenzyme Q10, studies have shown benefits in Parkinson's patients; this antioxidant is produced in the body however levels decrease with age (and it is depleted by statin medication).

Alternative products: *Foundation Formula 1* (contains iron so more important for menstruating woman and children), *Foundation Formula 2*, *Wholefood Multi* and *Cyto-Gold*

## Other Formulations



**Liposomal Glutathione Complex** – antioxidant formula designed for cellular regeneration and energy production. Provides 250mg of liposomal glutathione, depleted glutathione levels are associated with Parkinson's (liposomal glutathione is absorbed intact and can cross the blood brain barrier). Also contains N-acetyl L-carnitine, alpha lipoic acid, Gingko biloba, rosemary leaf and resveratrol, supporting mitochondrial function and providing antioxidant properties.



**Phyte-Inflam** – a combination of curcumin, ginger and piperine which collectively bestow a wide range of anti-inflammatory properties. Curcumin also has neuroprotective properties. Contraindications: If taking prescribed medications check for potential interactions.



**Phytoshield** – a very potent and powerful phyto-antioxidant nutrient formula containing high levels of flavonoids and carotenoids. Each capsule has an ORAC (Oxygen Radical Absorbance Capacity) score of 5,000 units. This shows exceptionally high antioxidant activity, when you consider that 5 portions of fruit and vegetables yield 1,500 ORAC units. Most people ingest on average 140mg flavonoids per day, against a recommended level of 650mg for optimum health; likewise, with carotenoids the average intake is 5mg daily, against recommendations of 20mg.



**Methyl Factors** – a formula designed to provide methyl donor nutrients to help those with elevated homocysteine and conditions that occur as a result of impaired methylation.

## Fatty Acids



**R-Omega** – omega-3 fatty acids from herring roe containing a high proportion of DHA (40%) (predominant omega-3 in the brain) as well as EPA (15%). Also contains phospholipids important for absorption of fatty acids, methylation and cell membranes. R-omega has been shown to be 3 times more bioeffective than other fish oils at the same levels.



**Krill Oil/CoQ10/K2** – contains the omega 3 fatty acids EPA and DHA as well as powerful antioxidant astaxanthin. CoQ10 an important antioxidant, diminished in patients with Parkinson's. Also provides K2 (lipid antioxidant), the most bioavailable form of vitamin K, which plays a key role in cell growth of neurons and glial cells.



**Omega-3 Vegan** provides DHA and EPA from a marine algal source. It is therefore suitable for vegetarians and vegans who may find it difficult to obtain sufficient omega-3 fatty acids from their diet.

*Alternative products: High potency fish oil capsules, Lem 0-3, Organic flaxseed oil, Golden Phospholec*

## Digestive Support

### Live bacteria

Research has demonstrated the benefits of certain species of live bacteria in reducing inflammation and permeability of the gut (i.e. leaky gut), as well as helping to reduce constipation. All our live bacteria products are grown on molasses and thus are suitable for vegans.

**Fos-A-Dophilus** – contains 6 strains of live bacteria. High in *Bifidobacterium spp* which tends to be less easily regenerated with ageing.

**Cyto-Biotic Active** – contains 9 strains of live bacteria, in a powdered form.

*Alternative products: Acidophilus Plus, Saccharomyces Boulardii*





Please also see our new supplement range to support The Bredesen Protocol and our full Product Information Catalogue.

The Bredesen Protocol™ Range		
Vitamin and Mineral Formulae		
9301	CoQ10 Multi: All-encompassing MVM with CoQ10 & Immune support	
3637	<b>Improved</b> Blood Glucose Support: GTF Chromium, Magnesium & Mineral complex with Cinnamon	
1203	Detox Support: Multimineral with Vitamin C, Garlic & Spirulina	
2212	<b>Improved</b> Methyl Factors: Providing methyl donor nutrients	
9312	<b>NEW:</b> Vitamin A as Retinol Palmitate	5000i.u.
4016	Pantothenic Acid (Vitamin B5)	50mg
1053	<b>NEW:</b> Vitamin B12 Sublingual, (as Methylcobalamin & Adenosylcobalamin)	500µg
1036	Vitamin B12 Sublingual (as Hydroxycobalamin)	1mg (1000ug)
1044	Vitamin C + Bioflavonoids (Ascorbic Acid)	1000mg
9302	<b>NEW:</b> Vitamin K2 & Vitamin D3	100µg/100ug
3306	Organic Kelp: 400mg providing 280ug of Iodine	400mg
4081	Food State Zinc & Copper	15mg/1mg Elemental
9303	<b>NEW:</b> Nicotinamide Riboside	
9304	<b>NEW:</b> Magnesium Threonate	
9307	<b>NEW:</b> Mixed Tocopherols & Tocotrienols	
Essential Fats		
1174	Omega 3 Vegan: From Marine Algae 333mg DHA & 167mg EPA per 2 capsule dose	
9310	<b>NEW:</b> R-Omega: DHA (40%) + EPA (15%) high in phospholipids	500mg
Gut Support		
4134	Fos-a-dophilus: 6 strains of live probiotics plus Inulin	
4130	Caprylic Acid Plus: with garlic, oregano, grapefruit seed extract & green tea	
3303	Slippery Elm	230mg
1269	Saccharomyces Boulardii: 5x10 <sup>9</sup> CFUs.	250mg
4133	Cyto-Zyme: High potency, digestive enzyme complex	
Anti-Inflammatory		
3326	Phyte-Inflam: Bio-activated Herbal Complex with Curcumin & Piperine	
3209	Phytoshield: Exceptionally powerful, high-potency phyto-antioxidant	
9309	<b>NEW:</b> Liposomal Glutathione Complex: Multi-nutrient antioxidant formula	
Herbals		
9306	<b>NEW:</b> Organic Ashwagandha	500mg
9308	<b>NEW:</b> Organic Bacopa Monnieri	500mg
9311	<b>NEW:</b> Organic Curcumin Plus (Herbal Complex Type 1)	
9313	<b>NEW:</b> Organic Gut Kola Plus (Herbal Complex Type 2)	



## Bibliography

Alessandro Rimessia, Maurizio Previatib, Federica Nigroa, Mariusz R. Wieckowski, Paolo Pintona. Mitochondrial reactive oxygen species and inflammation: Molecular mechanisms, diseases and promising therapies. The International Journal of Biochemistry & Cell Biology. 2016 Jun 29. pii: S1357-2725(16)30155-8

Aquilano K, et al. 2008. Role of Nitric Oxide Synthases in Parkinson's Disease: A review on the antioxidant and anti-inflammatory activity of polyphenols. Neurochem Research. 33:2416-2426

Bastainetto S and Quirion R. 2004. Natural antioxidants and neurodegenerative disease. Frontiers in Bioscience. 9:3347-3452

Beate R, Fei Y. 2000. Parkinson's disease mortality and pesticide exposure in California. 1984-1994. International Journal of Epidemiology 29:323-329

Blázovics A, Lugasi A, Kemény T, Hagymási K, Kéry A. 2000. Membrane stabilising effects of natural polyphenols and flavonoids from *Sempervivum tectorum* on hepatic microsomal mixed-function oxidase system in hyperlipidemic rats. J Ethnopharmacol. 2000 Dec;73(3):479-85

Boumival J, et al. 2009. Protective effects of resveratrol and quercetin against MPP - induces oxidative stress act by modulating markers of apoptotic death in dopaminergic neurons. Cell Mol. Neurobiology. 29:1169-1180

Cereda E, et al. 2010. Controlled protein dietary regimens for Parkinson's disease. Nutritional Neuroscience. 13(1):29

Dajas F, et al. 2005. Flavonoids and the brain: Evidences and putative mechanisms for a protective capacity. Current Neuropharmacology. 3(3) 193-205

Datla KP, et al. 2007. Short-term supplementation with plant extracts rich in flavonoids protect nigrostriatal dopaminergic neurons in a rat model of Parkinson's disease. *J Am Coll Nutr.* 26(4):341-349

Diplock AT, et al. 1998. Functional food science and defence against reactive oxygen species. *British Journal of Nutrition.* 80(1) S77-S112 S77

Evatt ML, et al. 2008. Prevalence of Vitamin D Insufficiency in patients with Parkinson's disease and Alzheimer's disease. *Archives of Neurology.* 24:3174-3182

Freidlich AL, et al. 2009. Oxidative stress in Parkinson's disease. *The Open Pathology journal.* 3:38-42

Gao X, et al. 2007. Prospective study of dietary pattern and risk of Parkinson's Disease. *Am J Clin Nut.* Nov 85(5): 1486-94

Garcia JJ, et al. 2009. Effects of *Plantago ovata* husk on levo-dopa. Bioavailability in rabbits with autonomic gastrointestinal disorders. *Drug Metab Dispos.* 37:1434-1442

George JL, et al. 2009. Targeting the progression of Parkinson's disease. *Current Neuropharmacology.* 7:9-36

Gillette JS et al. 2003. Differential up-regulation of striatal dopamine transporter and  $\alpha$ -synuclein by the pyrethroid insecticide permethrin. *Toxicology and applied Pharmacology.* Vol 192:3 287-293

Gladson M, et al. 2008. Blockage of the proposed precipitating stage for Parkinson's disease by antioxidants: A potential measure for PD. *The FASEB Journal.* 22:715

Hancock DB et al. 2008. Pesticide exposure and risk of Parkinson's disease: A family based case-control study. BMC Neurology, 8:6 doi:10.1186/1471-2377-8-6

Kerry N and Rice-Evans C. 1999. Inhibition of peroxynitrate-mediated oxidation of dopamine by flavonoid and phenolic antioxidants and their structural relationship. J Neurochem. 73, 247-253

Kim HG, et al. 2010. Mulberry fruit protects dopaminergic neurons in toxic-induced Parkinson's disease models. Br J Nutr. 104:8-16

Leader L and Dr Leader G 2009. Parkinson's Disease – Dopamine Metabolism, Applied Biochemistry and Nutrition. Denor Press, UK

Lonneke ML, et al. 2006. Serum cholesterol levels and risk of Parkinson's disease. Am J. Epidemiology. 164(10):998-1002

Mandel S, et al. 2006. Green tea catechins as brain-permeable, natural iron chelators-antioxidants for the treatment of neurodegenerative disorders. Mol Nutr and Food Res. 50 (2):229-234

Middleton E, et al. 2000. The effects of plant flavonoids on mammalian cells: Implications for inflammation, heart disease and cancer. Pharmacology reviews. 52(4):673-751

Miller RL et al. 2009. Oxidative and Inflammatory pathways in Parkinson's Disease. Neurochemical Research. 33:55-65

Murakami k, et al. 2010. Dietary intake of folate, vitamin B12 and riboflavin of Parkinson's disease: a case-control study in Japan. British Journal of Nutrition. Sep;104(5):757-64



Omale, James and Okafor, Polycarp Nnacheta. 2008. Comparative antioxidant capacity, membrane stabilization, polyphenol composition and cytotoxicity of the leaf and stem of *Cissus multistriata*. *African Journal of Biotechnology* Vol. 7 (17), pp. 3129-3133, 3 September, 2008

Oseiki H. 2006. *The Physician's Handbook of Clinical Nutrition*. Australia: Bioconcepts. Pp 777

Pizzomo JE, Murray MT. *Textbook of Natural Medicine* 4th Ed. 2013. Elsevier

Powers KM, et al. 2009. Dietary fats, cholesterol and iron as risk factors for Parkinson's disease. *Parkinsonism related Disorders*. Jan 15(1):47-52

Riveles K et al. 2008. Cigarette smoke, nicotine and cotinine protect against 6-hydroxydopamine-induced toxicity in SH-SY5Y cells. *Neuro Toxicology*. 29:3421-427

Savica R, et al. 2009. A case-control study. Medical records documentation of constipation preceding Parkinson's disease. *Neurology*. 2009; 73:1752-1758

Schultz JB, et al. 2000. Glutathione, oxidative stress and neurodegeneration. *Eur J. Biochem*. 267:4904-4911

Shults CW, et al. 2002. Parkinson's study group. Effects of coenzyme Q10 in early Parkinson's disease: evidence of slowing the functional decline. *Archives Neurology*. 59(10):1541-50

Sofi F, et al. 2008. Adherence to Mediterranean diet and health status: meta-analysis. *BMJ*;337 (10):1344

Spencer JPE. 2007. The interactions of flavonoids within neuronal signalling pathways. *Genes and Nutrition*. 2:257-273

Spencer JPE. 2008. Flavonoids: Modulators of brain function? *Br J Nut*. 99:ES60-ES77

Stacey E. Seidl, Jose A. Santiago, Hope Bilyk, and Judith A. Potashkin. The emerging role of nutrition in Parkinson's disease. *Front Aging Neurosci*. 2014; 6: 36. Published online 2014 Mar 7. doi: 10.3389/fnagi.2014.00036

Tolosa E, et al. 2009. Diagnosis and the premotor phase of Parkinson's disease. *Neurology*. 72:S12-S20

Tufekci KU, Meuwissen R, Genc S, Genc K. Inflammation in Parkinson's disease. *Adv Protein Chem Struct Biol*. 2012;88:69-132. doi: 10.1016/B978-0-12-398314-5.00004-0

Wang M, et al. 2010. Curcumin reduces a-synuclein induced cytotoxicity in Parkinson's disease cell model. *Bio Med Centra Neuroscience*. 11:57

Weinreb O, et al. 2004. Neurological mechanisms of green tea polyphenols in Alzheimer's and Parkinson's disease. *The J Nut Biochem*. 15(9):506-516

[www.parkinsons.org.uk/default.aspx?page=10057](http://www.parkinsons.org.uk/default.aspx?page=10057)

[www.parkinsons.org.uk/research/current\\_research/what\\_causes\\_parkinsons.aspx](http://www.parkinsons.org.uk/research/current_research/what_causes_parkinsons.aspx)

Zbarsky V, et al. 2005. Neuroprotective properties of the natural phenolic antioxidants curcumin and naringenin but not quercetin and fistin in a 6-OHDA model of Parkinson's disease. *Free Radicle Research*: 39(10):1119-1125



Zhang SM, et al. 2002. Intakes of vitamins E and C, carotenoids, vitamin supplements and PD risk. *Neurology*. 22;59(8) | 161-9

Zhiming Li, Zaozao Zheng, Jun Ruan, Zhi Li, and Chi-Meng Tzeng. Chronic Inflammation Links Cancer and Parkinson's Disease. Published online 2016 Jun 3



Cytoplan celebrates 26 years in the field of food-based supplementation and from the moment of conception to the present day we have promoted the philosophy that nutrients are best delivered to the body "in the same form as food".

The philosophy and message of Cytoplan was founded on the simple logic that our bodies are designed to eat food and utilise its components for the maintenance of life. The ultimate goal of Cytoplan is to 'improve the health of the nation' by supplying supplements in a bio-effective form for optimal absorption and utilisation.



CYTOPLAN

Cytoplan Limited, Unit 8, Hanley Workshops, Hanley Swan, Worcestershire WR8 0DX  
Telephone: 01684 310099 Email: [sales@cytoplan.co.uk](mailto:sales@cytoplan.co.uk)

[www.cytoplan.co.uk](http://www.cytoplan.co.uk)

THIS LEAFLET IS FOR HEALTH PROFESSIONALS

Science Based Supplements

Copyright Cytoplan Limited 2016