Best practice guideline for dietitians on the management of Parkinson’s
Introduction

People living with Parkinson’s are particularly susceptible to weight loss and malnutrition. Involuntary movements associated with Parkinson’s result in increased energy expenditure, while disease symptoms and medication side effects can limit food intake. They may also choose to follow unconventional nutritional therapies that consequently exacerbate malnutrition. Dietitians play a key role in helping patients with Parkinson’s to optimise their nutritional status and manage various nutrition-related symptoms and medication side effects. To assume this role, dietitians need to have up-to-date knowledge about the nutritional consequences of Parkinson’s, as well as strategies for managing a variety of nutrition-related symptoms.

Nutritional management at different stages of Parkinson’s

Diagnosis and early disease

Parkinson’s can be a challenging diagnosis to make in its early stages. It is a diagnosis based on clinical observation and exclusion of other conditions.¹

Dietetic assessment

The initial assessment could be facilitated by another healthcare professional as well as a dietitian e.g. Parkinson’s Nurse, GP or practice nurse. Examples of assessments that could be undertaken are: weight; height; body mass index (BMI); and/or using a validated nutrition screening tool e.g. the Malnutrition Universal Screening Tool (MUST).²

Dietary intervention

Healthy eating

There is no specific diet required; however, people newly diagnosed with Parkinson’s should be advised to eat a healthy and well-balanced diet. Good nutrition is essential to the wellbeing of this client group. It is imperative to establish and maintain good eating habits throughout the course of the condition.³

Written and verbal information should be provided on how to adopt the Healthy Eating Guidelines set out by the Food Standards Agency (FSA) and incorporated by the Parkinson’s UK booklet Diet and Parkinson’s.

Weight maintenance

Being underweight, something commonly experienced by those living with Parkinson’s, as well as being overweight can lead to adverse health effects. In overweight individuals, it is advised that an agreed goal for ideal body weight should be established. This is especially significant in those who have co-morbidities e.g. hypertriglyceridaemia, diabetes, CVD etc.⁴

In the case of weight loss, when other medical causes of weight loss can be safely excluded, NICE has advised that people living with Parkinson’s experiencing weight loss should be referred to a dietitian.⁵

Most likely in the early stages of Parkinson’s, oral nutrition support where deemed appropriate, will be provided initially before the need for artificial nutrition support (i.e. NG/PEG). However this client group is unique and must be treated as individuals; it is well known that this condition does not affect one person in the same way as the next.
**High fibre diet**

Constipation is one of the most common problems experienced by Parkinson’s patients. About 60–80% of patients with Parkinson’s complain of constipation, which usually appears about 10 to 20 years before motor symptoms become evident. It is thought to be caused by damage to the peripheral or central nervous system. Other causes of constipation in Parkinson’s patients are lack of fibre intake, lack of fluid intake, decreased mobility and as a result of some Parkinson’s medications.

Dietary advice on how to prevent constipation by following a high fibre/fluid intake is well outlined in the Parkinson’s UK booklet *Looking after your bladder and bowel*.

**Antioxidants**

Once Parkinson’s has been diagnosed, there is no evidence to support that taking supplemental doses of antioxidants slows the progression of the condition or enhances the effects of Parkinson’s drugs. For those who are concerned about their intake of antioxidants, they should be reassured that a well-balanced diet will meet their antioxidant needs and that food sources of these nutrients (particularly fruit and vegetables) may be better, safer and less expensive than consuming large quantities of supplements. Providing them with the Parkinson’s UK information sheet *Antioxidants and Parkinson’s* (FS67) will help reinforce your advice and guidance.

**Co-enzyme Q10**

It was previously thought that taking supplementary co-enzyme Q10 would delay the progression of Parkinson’s. Owing to the lack of scientific evidence, the NICE Guideline for Parkinson’s disease has recommended that co-enzyme Q10 should not be used as a neuroprotective therapy, except in the context of clinical trials. Co-enzyme Q10 can be found in offal, beef, soya oil, oily fish and small amounts in peanuts. More information is available in the Parkinson’s UK information sheet *Co-enzyme Q10* (FS74).

**Monitoring**

Dietitians at established Parkinson’s centres agree that people newly diagnosed with, or in the early stages of the condition, should be reviewed on an annual basis or at the patients’ and/or multidisciplinary team’s request.

**Advanced disease**

**Dietetic assessment**

Assessment is similar to the early stages of the condition paying particular attention to:

- unintentional weight-loss
- decreased oral intake
- swallowing difficulties
- increased frequency of dyskinesias (abnormal involuntary movements)
- constipation
- side effects of medication (see table 1)
- physical difficulties impairing eating and drinking and preparing meals
- bone health
- weight-gain after deep brain stimulator (DBS) insertion
- frequency of ‘on’ (increased involuntary movements) and ‘off’ (increased muscle tone) periods
- orthostatic hypotension (OH) – characterised by a sudden fall in blood pressure that occurs when a person assumes a standing position
<table>
<thead>
<tr>
<th>Name of drug</th>
<th>Mode of action</th>
<th>Side effects</th>
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<tbody>
<tr>
<td><strong>Levodopa</strong> (Madopar, Sinemet)</td>
<td>The most effective drug in the treatment of Parkinson’s. It is a larger neutral amino acid – tyrosine derivative – and is absorbed in the small intestine. Once absorbed it crosses the blood-brain barrier where it is converted to dopamine.</td>
<td>Prolonged use can result in nausea, vomiting and hypotension. A decarboxylase inhibitor Benserazide, is combined with levodopa to minimise these side effects.</td>
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<tr>
<td><strong>Dopamine agonists</strong> (Pergolide, Apomorphine)</td>
<td>These are a class of drugs that provide antiparkinsonian effects while avoiding some of the problems associated with levodopa.</td>
<td>Acute side effects of these drugs included nausea, vomiting, postural hypotension and psychiatric problems. They tend to occur with the initiation of treatment and as intolerance develops over several days to weeks.</td>
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<tr>
<td><strong>MAO-B inhibitors</strong> (Selegiline)</td>
<td>This is used in the early stages of Parkinson’s to delay the use of levodopa. It provides reduced motor fluctuations and increased ‘on’ time.</td>
<td>It has amphetamine and metamphetamine metabolites which can cause insomnia, therefore could induce daytime sleeping.</td>
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<tr>
<td><strong>Anticholinergics</strong> (Benzhexol)</td>
<td>This is typically used in younger Parkinson’s patients (&lt;60 years) in whom resting tremor is the dominant clinical feature and cognitive function is preserved.</td>
<td>Side effects may include dry mouth, constipation and nausea.</td>
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<tr>
<td><strong>Amantidine</strong></td>
<td>This is an antiviral agent that is believed to increase dopamine release by blocking dopamine reuptake by stimulating receptors and possibly by anticholinergic effects.</td>
<td>Side effects include confusion, hallucinations, insomnia, nightmares and dry mouth.</td>
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Domperidone, an anti-emetic, is usually used in conjunction with these medications to ease the symptoms of nausea and vomiting.
**Dietary intervention**

**Oral nutrition support (ONS)**

ONS should be provided when the patient can no longer maintain their body weight through oral diet/intake alone.\(^8\) Suggestions such as food fortification, high energy/protein advice, and regular small energy-dense meals should be explored first. The use of oral sip feeds should be considered for those who are unable to adhere to food fortification due to anxiety at meal times, early satiety or lack sufficient help to prepare the fortified meals and snacks. Imaginative use of these products will help reduce taste fatigue and increase adherence to recommendations.

Particular attention should be paid to those on a texture modified diet (TMD), especially puree diets. Puree diets are known to provide varying nutritional adequacy and can be unpalatable, if not prepared imaginatively. All patients on texture-modified diets should be assessed by the dietitian for nutritional support.\(^9\) It is recommended that the dietitian and speech and language therapist work closely to ensure the patient receives appropriate and optimal nutrition and hydration.

**Artificial Nutrition Support (ANS)**

In Parkinson’s it often helps to anticipate artificial feeding at an early stage through discussions with the patient and/or carer supported by the multidisciplinary team.\(^10\)

Swallowing difficulties need to be addressed promptly to prevent weight loss and malnutrition. About 95% of Parkinson’s patients experience swallowing difficulties at some stage of the condition.\(^11\) Dysphagia occurs in the later stages of Parkinson’s compared to atypical parkinsonian syndromes e.g. progressive supranuclear palsy (PSP) or multiple system atrophy (MSA).\(^12\) Significant problems with swallowing require expert assessment from a speech and language therapist and guidance regarding appropriate food texture modification. Since texture modified diets e.g. puree, may be nutritionally dilute and not energy dense enough to prevent weight loss, additional use of supplementary enteral nutrition support may be indicated.

Active nutritional support via the enteral route e.g. naso-gastric tube for short-term feeding or if indicated, a Percutaneous Endoscopic Gastrostomy (PEG) could be considered for long-term feeding. PEGs are being used increasingly in the treatment of patients with neurogenic dysphagia to prevent or reverse nutritional deficits,\(^13\) and this can improve fitness and quality of life for those patients unable to take sufficient supplements orally.

**Bone health**

Osteoporosis is often diagnosed in people living with Parkinson’s and correlation between disease severity and bone density has been found. This client group has been found to have a defect in the renal synthesis of 1, 25-dihydroxy-vitamin D (1, 25 – [OH]\(_2\)D).\(^14\) It has been suggested that they should be supplemented with 1-alpha-hydroxyvitamin D3 – the more active form of vitamin D, which can help increase bone density and dramatically lower the risk of fracture in these patients.\(^14\) This is essential for people who are bed/house-bound or immobile.

Calcium and vitamin D status should therefore be assessed early in the condition to prevent or decrease incidence of osteoporosis. Research offers compelling evidence for the benefits of vitamin D supplementation.\(^15, 16\) It has been advised that patients with Parkinson’s should be routinely supplemented with 1000–1500mg of calcium and 10–15µg vitamin D daily.\(^17\) Examples are Calcichew and Ad-Cal D3 Forte.
Orthostatic hypotension

The prevalence of symptomatic orthostatic hypotension (OH) may be as high as 50% in people living with Parkinson’s. This could be caused by: a) dopaminergic drugs which induce or worsen orthostatic hypotension and/or b) primary autonomic failure with an involvement of the peripheral autonomic system caused by Parkinson’s.

OH (also known as postural hypotension) is a sudden fall in blood pressure that occurs when a person assumes a standing position. OH sufferers may also experience post-prandial hypotension – PPH (blurred vision and dizziness after meals). These symptoms can occur any time from ingestion to 90 minutes post meal. Studies from long-term care facilities have shown that 24-36% were diagnosed with PPH. It has been suggested that the nutrient composition of meals affects the magnitude of the decrease in postprandial hypotension.

The intestines require a large amount of blood for digestion. When blood flows to the intestines after a meal, the heart rate increases and blood vessels in other parts of the body constrict to help maintain blood pressure. However with this patient group, such mechanisms may be inadequate. Blood flows normally to the intestines, but the heart rate does not increase adequately and blood vessels do not constrict enough to maintain blood pressure. As a result blood pressure falls.

Symptoms include:
- feeling dizzy and light-headed
- changes in vision such as blurring, greying or blacking vision
- feeling vague or muddled
- losing consciousness with or without warning – a ‘blackout’ or ‘faint’ as a result of hypotension
- pain across the back of shoulders or neck – ‘coat hanger’ pain
- pain in lower back or buttocks
- angina-type pain in the chest
- weakness
- fatigue

The symptoms are typically worse when standing and improve on lying down.

Dietary management of OH

The main aim of the dietary management is to assist in the alleviation of the symptoms of hypotension. This dietary management can often appear unusual or unconventional; however research is available to back up the advice. Often the objective of dietary manipulation is to improve the functional capacity and sense of wellbeing rather than to reach a fixed arterial blood pressure. The goal must be to alleviate the symptoms of postural and postprandial hypotension and assist with fatigue management. The skills of the dietitian are required when advising on ways to manipulate the diet, to ensure the nutritional adequacy of the diet is not compromised. It should also be ensured that restrictive diets are not followed, which would lead to further risk of malnutrition in an already nutritionally vulnerable group of patients.

Advice to be given to people with OH:
- Avoid large meals
- Reduce carbohydrate intake, especially simple sugars. It has been hypothesised that carbohydrates induce hypotension in autonomic failure through the vasodilating properties of insulin.
- Increase intake of salt
- Increase fluid intake
- Decrease or omit alcohol intake
**PPH accompanied by weight loss**

As the OH progresses, many patients experience weight loss, the reason being multifactorial. Patients may restrict intake as they associate meals or a certain food with post-prandial symptoms, or as a consequence of dysphagia.

Deciding upon which oral supplement to use can become a challenge if you are trying to get your patient to avoid/decrease glucose intake. Some patients can tolerate energy-dense oral sip feeds (e.g. Clinutren 1.5, Fortisip, Ensure Plus, Fresubin Energy) without experiencing symptoms, especially if sipped slowly. However, for those that do experience PPH symptoms, a lipid based solution such as Calogen/Procal shots, can be useful, although not all find them palatable. The provision of innovative recipes and suggestions on how to incorporate these supplements into meals will be helpful to the person living with PPH.

Artificial nutrition may be necessary for those who are experiencing difficulty maintaining weight through oral diet alone or develop dysphagia.

If your patient finds it difficult to follow these recommendations, it is important to let the Neurology Consultant know, so that alternative treatments can be offered. If the patient’s autonomic failure is so severe, dietary intervention may not offer any benefit.

**Weight gain post-chronic bilateral subthalamic or deep brain stimulation (CDBS)**

It has been shown that weight gain can occur in 87% of patient’s post-deep brain stimulation surgery. Around 38% of these patients were found to be overweight (BMI>26–29.9) six-months post-op. This is thought to be due to improvement in motor fluctuations, opposed to increase in oral intake.27

Dietary intervention (using ideal body weight to calculate energy requirements) at time of surgery has been advocated by researchers in Portugal28 to enhance the benefit of the surgery and prevent metabolic disorders. It is thought that excessive weight gain could exacerbate motor function impairment in Parkinson’s. It has been suggested that clinicians need to proactively monitor weight changes in these patients.

Healthy Eating Guidelines set out by the Food Standards Agency (FSA) and incorporated by the *Diet and Parkinson’s* booklet available from Parkinson’s UK can be a useful tool to prevent rapid weight gain in those who are undergoing or have undergone DBS surgery.

**Dietary protein manipulation**

Levodopa (L-dopa) is the standard and often initial therapy for patients with this condition; however, with continued treatment and as the condition progresses, up to 80% of patients experience ‘wearing-off’ symptoms, dyskinesias and other motor complications. It was believed in the late 1980s and early 1990s that dietary protein manipulation would help control motor fluctuations.29, 30, 31,32,33,34 L-dopa disappears from the blood very quickly, usually about 60–90 minutes after administering the drug. Protein can delay gastric emptying and competes with the absorption of L-dopa. Manipulations of dietary protein may reduce fluctuations in some patients; however there has been some controversy over the amount of protein needed to prevent the antiparkinsonian effect of L-dopa.

It is has been suggested that motor fluctuations may be delayed by the early use of sustained release L-dopa preparations or early combinations of L-dopa with a dopamine agonist.35 Medications, such as controlled-release levodopa, amantidine, clozapine, COMT inhibitors, MAO-B inhibitors and dopamine agonists; or Apomorphine injections/infusions; or DBS are now offered to those with extreme and debilitating motor fluctuations.
It is worth bearing in mind that any changes to the diet, e.g. decreasing or omitting protein, should not be done without the agreement of the Neurology Consultant or Parkinson’s nurse. It is more important to address the timing of L-dopa medications around mealtimes to ensure that dietary protein does not interfere with the absorption of the L-dopa across the blood-brain barrier. It has been suggested that there should be a 40-minute delay between taking L-dopa and eating a meal. This ensures that the L-dopa can be absorbed from the small intestine into the bloodstream, across the blood-brain barrier and then enzymatically converted to dopamine in the brain.

**Monitoring**

Every four to six weeks if there have been any changes to medications or every three months if the patient’s condition is stable. For oral nutrition support, regular review of ONS prescriptions every three months is advisable, to ensure the appropriateness of the intervention. Sample packs of oral sip feeds can be trialled before any changes made to the GP prescription. Some centres offer one-day medical reviews to re-assess mobility, swallow, speech and nutritional status. However it is important to liaise with other members of the multidisciplinary team caring for the patient to ensure you are keeping in line with the patient’s overall management plan.

**End stage**

The management of Parkinson’s remains largely palliative, despite huge advances in the medical field. NICE advise that “palliative care should be applied throughout the course of the disease and not limited to the terminal end-of-life period”. Achievement of best quality of life for patients and their carers/families is the goal of palliative care.

End-stage or palliative phase is defined by:

- inability to tolerate dopaminergic therapy
- unsuitable for surgery e.g. DBS
- the presence of advanced co-morbidity

It has been documented that a patient can spend on average 2.2 years in the palliative care stage. During this stage there may be a need to withdraw dopaminergic medications due to lack of drug efficiency. Decreased mobility and being bed-bound can result in the risk of pressure ulcer development. Decisions may need to be made about the nutritional management and treatment in the future to give the person with Parkinson’s an opportunity to state their preferences in case they lose capacity for making decisions in the future.

**Dietetic assessment**

Assessment will be similar to the early stage taking into special consideration:

- suitability of active +/- aggressive nutrition support
- prognosis
- palliative care team advice
- multidisciplinary team decision
- patients decision
- any advanced care planning

**Dietary intervention**

- Artificial nutrition support may be withdrawn if it is causing discomfort or distress, or not deemed to be in patient’s best interest
- You may be asked to reduce the volume of enteral feeds to prevent fluid overload
- Oral nutrition should always be offered but not forced upon those who decline it or when there is risk of aspiration or choking
Monitoring
Monthly reviews may be more appropriate or as agreed with/at the request of the multidisciplinary team/palliative care team.

Summary
The risk of malnutrition and body weight must be routinely monitored as the condition progresses. Worsening motor symptoms e.g. dyskinesias should also be monitored to prevent or reverse weight loss in people living with Parkinson’s. Dietitians have the skills to help people living with Parkinson’s to optimise their nutritional status and manage nutrition-related symptoms at all stages of the condition.

References:
2. ‘Malnutrition Universal Screening Tool (MUST)’, BAPEN (2006)
4. ‘Department of Health Care Pathway for the management of overweight and obesity’ Department of Health (2006)
6. ‘Protein Intake in Parkinsoniam Patients Using the EPIC Food Frequency Questionnaire’ (Movement Disorders: 21 (8), 1229-31), Marczewska A et al (2006)


27. ‘Comparison of weight gain and energy intake after subthalamic versus pallidal stimulation in Parkinson’s disease’ (Mov Disorders: 24(14) 2149-55), Sauleau P et al (2009)

28. ‘Modulation of nutritional stat in Parkinsonian patients with bilateral subthalamic nucleus stimulation’ (J Neurol), Guimaraes J et al (July 2009 online published ahead of print)


38. ‘Validation of pathways paradigm’ (Parkinsonism & Related Disorders: 5(S53) Ref ID: 19912), MacMahon DG et al (1999)
42. ‘Parkinson’s Disease: Implications for Nutritional Care’ (Canadian Journal of Dietetic Practice and Research, 63, (2) 81-7), Cushing, M.L et al (2002)

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BDA consensus on the nutritional management of PD

### Disease progression

- **Dietetic Assessment***
  - Establish the following:
    - Anthropometry – weight/height/BMI
    - Score from a validated nutritional screening tool e.g., MUST
    - Dietary intake

### Diagnosis and early disease

- **Dietary intervention**
  - Written and verbal information covering:
    - Healthy Eating Guidelines set out in the Eat Well Plate by the FSA or Diet and Parkinson’s booklet
    - Agree ideal goal weight especially if overweight
    - High fibre diet to prevent constipation
    - Antioxidants
    - Co-enzyme Q10
    - Dietary advice suitable for other co-morbidities e.g. Diabetes, CVD, CHD

- **Monitoring**

### Advanced disease

- Same as early disease paying particular attention to:
  - Unintentional weight loss
  - Decreased oral intake
  - Swallowing difficulties
  - Increased frequency of dyskinesias
  - GIT function i.e. constipation
  - Side effects of medication
  - Physical difficulties
  - Bone health
  - Weight gain post-DBS insertion
  - Frequency and length of ‘on’ and ‘off’ periods
  - Dietary intake

- **Nutrition support**
  - Oral:
    - Food fortification
    - High energy/protein
    - Small and regular energy-dense meals
    - Oral sip feeds
    - Texture modified diet (TMD) advice
  - Enteral:
    - PEG counselling
    - PEG insertion
    - Individualised feed regimen
    - PEG & TMD*** advice

### Weight gain post-DBS

- Low fat, high fibre diet
- Establish ideal healthy weight
- Regular exercise

- Consider dietary protein manipulation for uncontrolled motor fluctuations

### End stage

- Same as early stage taking into consideration:
  - Suitability of active +/- aggressive nutrition support
  - Prognosis
  - Palliative care team advice
  - MDT decision

- Monitoring

- Early disease: Annually or at Patient/multidisciplinary team request
- Advanced disease: Every three months
- End stage: Monthly or at the request of the multidisciplinary team/palliative care team