Research – Therapy Best Practice

Brief	In order to develop business rules for the funding of CB supports as part of the Participant Budget Model, we need the following information: • For the following disability groups: Parkinson's Disease, multiple sclerosis, muscular dystrophy, dementia, Huntington's Disease, arthritis, chronic fatigue, chronic pain, amputation. • What is considered best practice in terms of: a) The allied health team members of a multidisciplinary team, i.e. who should be involved in managing the disability? b) The frequency of intervention i.e. approximate dosage – how many hours per year is required for each professional? c) Evidence based practice for widely accepted therapy approaches. Not too much detail required, mainly eg "For MS, X therapy approach is often recommended, which involves intensive blocks of 20 sessions every X months". Looking for information again regarding number of hours that would be considered best practice.
Date	28/06/21
Requester(s)	Jane s47F - personal p - Assistant Director (TAB) Jean s47F - personal priv - Senior Technical Advisor (TAB)
Researcher	Jane s47F - personal priv - Research Team Leader (TAB)
Cleared	N/A

Please note:

The research and literature reviews collated by our TAB Research Team are not to be shared external to the Branch. These are for internal TAB use only and are intended to assist our advisors with their reasonable and necessary decision-making.

Delegates have access to a wide variety of comprehensive guidance material. If Delegates require further information on access or planning matters they are to call the TAPS line for advice.

The Research Team are unable to ensure that the information listed below provides an accurate & up-to-date snapshot of these matters.

The contents of this document are OFFICIAL

1 Contents

2 Summary

- Information provided has been obtain from a rapid review of the literature. This includes best practice guidelines, systematic reviews from the Cochrane Collaboration and other high quality meta-analyses and reviews.
- The personal circumstances, goals of each individual, and severity of the disease impacts the level of intervention required. Therefore, it is often not possible to provide an exact number of hours required for each intervention. This is reflected in the literature as studies investigating the same intervention often deliver it at a different frequency, leading to a lack of agreement around gold standard levels.
- If the agency requires precise numbers around how many hours of intervention are useful per clinician they will need to commission systematic reviews of each type of intervention delivered, across various disease severities. This is a substantial tasks. Current literature

focuses on the effectiveness rather than the intensity of intervention. The level of intervention is often decided by the allied health professional looking after the patient.

3 Parkinson's disease

3.1 Clinician involved in management

A systematic review and meta-analysis of integrated care in Parkinson's disease provides a list of core team members to be included in interventions [1].

- Movement disorders specialist
- General neurologist
- PD specialist nurse
- Physiotherapist
- Occupational therapist
- Speech therapist
- Clinical psychologist
- Neuropsychologist
- Community mental health team
- Social worker
- Dietician

Models of care varied significantly, ranging from 4-8 weeks, 1-4 sessions a day (30 minutes to 2 hr per session) ranging from 1-7 days a week. No indication of what hours were allocated to each profession.

3.2 Best practice treatment and frequency of intervention

Recommendations for treatment are taken from the NICE UK guidelines [2].

1) First-line treatment
    a. Offer levodopa to people in the early stages of Parkinson's disease whose motor symptoms impact on their quality of life.
    b. Consider a choice of dopamine agonists, levodopa or monoamine oxidase B (MAO-B) inhibitors for people in the early stages of Parkinson's disease whose motor symptoms do not impact on their quality of life.

2) Non-pharmacological management
    a. Nurse specialist interventions
        i. Clinical monitoring and medicines adjustment.
        ii. A continuing point of contact for support, including home visits when appropriate.

iii. A reliable source of information about clinical and social matters of concern to people with Parkinson's disease and their family members and their carers (as appropriate).

b. Physiotherapy and physical activity [3]
   i. General physiotherapy: 4 weeks to 12 months. Only 2 studies reported duration of sessions which included 12 hrs over 4 weeks and 18 hrs over 6 weeks.
   ii. Exercise: Treatment sessions lasted from 30 minutes to two hours, and took place over a period of three to 24 weeks.
   iii. Treadmill: Treatment sessions lasted from 30 to 60 minutes, and took place over a period of four to eight weeks.
   iv. Cueing: Treatment sessions lasted from four to 30 minutes and took place over a period of a single session to 13 weeks.
   v. Dance: Dance classes lasted one hour over 12 to 13 weeks, with a trained instructor teaching participants the tango, waltz, or foxtrot.
   vi. Martial arts: Treatment lasted one hour and took place over a period of 12 to 24 weeks

c. Speech and language therapy [4]
   i. Median duration of therapy for those treated was four weeks with 68% attending a single weekly session, a further 22%, who were predominantly receiving Lee Silverman Voice Therapy (LSVT), had four or more therapy sessions per week. Most sessions (80%) lasted between 30-60 minutes.

d. Occupational therapy [5]
   i. A Cochrane Review from 2007 only found 2 studies that met inclusion criteria. These studies delivered intervention of 12 hours across 4 weeks, and 20 hours over 5 weeks.

e. Nutrition [6]
   i. Monitoring every four to six weeks if there have been any changes to medications or treatment plan, with particular focus on the swallowing recommendations.
   ii. Every three months if the patient's condition is stable.
   iii. For oral nutrition support, regular review of ONS prescriptions every three months is advisable, to ensure the appropriateness of the intervention.
   iv. Some centres offer one-day holistic reviews to re-assess mobility, swallow, speech and nutritional status.

* Dysphagia management should be conducted by speech and language therapists in conjunction with nurses and dietitians. No information provided on level/duration of intervention [7].

3) Deep brain stimulation
   a. Surgery is performed to implant a device that sends electrical signals to brain areas responsible for body movement. Electrodes are placed deep in the brain and are connected to a stimulator device.

4 Multiple sclerosis

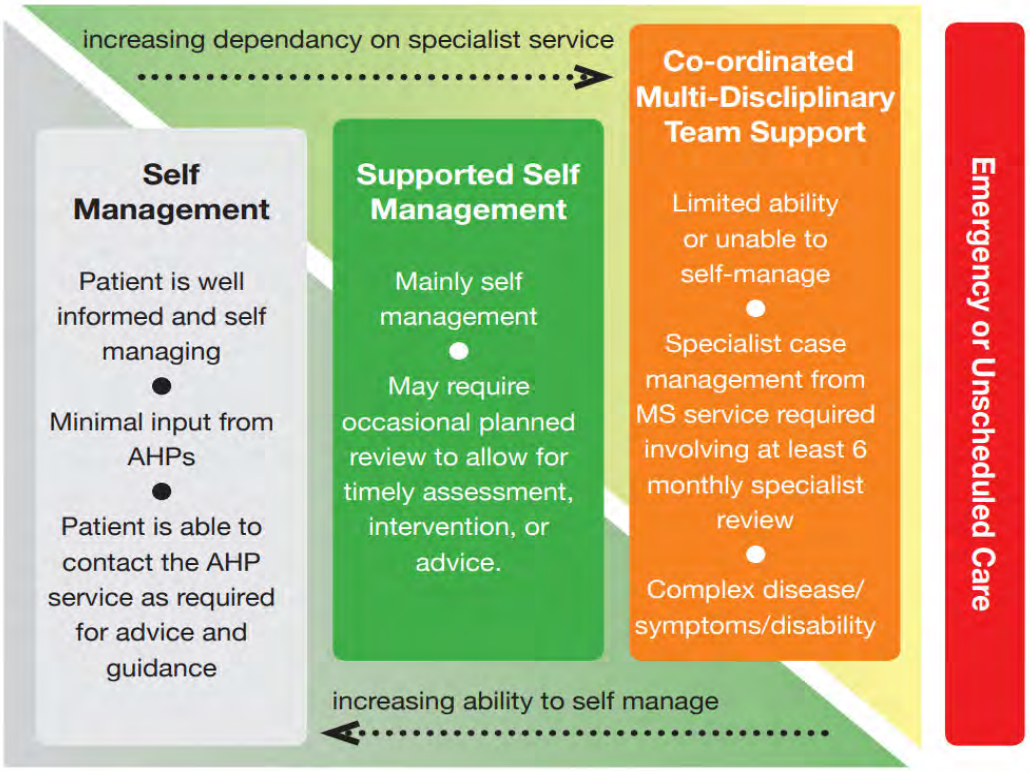
4.1 Clinician involved in management

There is variation in the make-up of MS multidisciplinary teams. The NICE MS Clinical Guideline states that: “As a minimum, the specialist neurological rehabilitation service should have as integral members of its team, specialist [8, 9]:

- Doctors (GPs, Neurologist)
- Nurses
- Physiotherapists
- Occupational therapists
- Speech and language therapists
- Dieticians
- Continence specialists
- Clinical psychologists
- Ophthalmologist/orthoptist
- Social workers.

General rehabilitation – patients must be seen for 6-8 sessions or for a 6-8 week period, however, appointments should be booked according to the needs of the patient [8]. The figure below describes the level of dependency on specialist services for varying levels of disease severity.

Figure 2[3]: Self Management/Specialist Service Dependency Model for People with MS



Patients are able to move fluidly in both directions between the different aspects of care illustrated, and such moves can be triggered either by the patient or their carer, or by the service professionals.

4.2 Best practice treatment and frequency of intervention

Determine how often the person with MS will need to be seen based on [9]:

- Their needs, and those of their family and carers
- The frequency of visits needed for different types of treatment (such as review of disease-modifying therapies, rehabilitation and symptom management).
    - *"Review information, support and social care needs regularly"*

The below interventions are listed in the NICE UK guidelines for the management of MS [9]

1) Exercise programs
2) Mindfulness-based training
3) Cognitive behavioural therapy
4) Fatigue management
5) Mobility rehabilitation
6) Spasticity management

7) Occupational therapy – memory or cognitive problems
8) Diet
9) Ocular rehab

A Cochrane Review of Multidisciplinary Rehabilitation (MD) for the treatment of MS has been conducted to determine its effectiveness [10]. The concept of MD comprises elements of physical therapy, occupational therapy, speech pathology, psychology and or neuropsychology, cognitive therapy and or behaviour management, social work, nutrition, orthotics, counselling input, recreation and vocational therapy.

Intensity of MD rehabilitation programme was subdivided into 'high' or 'low' intensity

- High intensity therapy involved input from at least two disciplines, a minimum of thirty minutes per session and total duration of at least 2-3 hours of interrupted therapy per day for at least 4 days per week. This is usually provided in inpatient settings and some outpatient programmes.
- Low intensity programmes varied, the intensity and duration of therapy was lesser than that provided in inpatient rehabilitation settings and was dependent upon the type of rehabilitation setting and available resources

From this review, it has not been possible to suggest best 'dose' of therapy, further studies are needed to suggest optimum number, duration and intensity of treatment sessions.

Neuropsychological rehabilitation

A Cochrane Review of neuropsychological rehabilitation (delivered by psychologists) for MS was conducted in 2014 [11]. It found that the number of intervention sessions varied from eight to 36, the duration of the rehabilitation intervention from four weeks to six months, and the frequency from two times per month to five times per week. When analysing the results with regard to the number of sessions, duration and frequency, no definite conclusions can be drawn about the effect of these factors on rehabilitation outcomes.

Exercise

Ranging from 6 to 24 weeks in duration, ranging from once to 5 times weekly frequency [12].

5 Muscular dystrophy

5.1 Clinician involved in management

Muscular dystrophy (MD) is a group of diseases that cause progressive weakness and loss of muscle mass. The most common form of MD is Duchenne's MD which most commonly occurs in young boys. The below will be presented for Duchenne's MD.

The care team should include a [13]:

- Neurologist with expertise in neuromuscular diseases
- Physical medicine and rehabilitation specialist

- Physiotherapist
- Occupational therapists.
- Speech-language pathologists
- Orthotist
- Psychologist
- Dietician.

Some people might also need a lung specialist (pulmonologist), a heart specialist (cardiologist, a sleep specialist, a specialist in the endocrine system (endocrinologist), an orthopedic surgeon and other specialists.

5.2 Best practice treatment and frequency of intervention

Several types of therapy and assistive devices can improve the quality and sometimes the length of life in people who have muscular dystrophy. Examples include [13]:

- **Range-of-motion and stretching exercises.** Muscular dystrophy can restrict the flexibility and mobility of joints. Limbs often draw inward and become fixed in that position. Range-of-motion exercises can help to keep joints as flexible as possible.
- **Exercise.** Low-impact aerobic exercise, such as walking and swimming, can help maintain strength, mobility and general health. Some types of strengthening exercises also might be helpful.
    - Optimal exercise modality and intensity of exercise for people with a muscle disease is still unclear. Large variation in frequency, duration and intensity exists within the literature [14-16].
- **Braces.** Braces can help keep muscles and tendons stretched and flexible, slowing the progression of contractures. Braces can also aid mobility and function by providing support for weakened muscles.
- **Mobility aids.** Canes, walkers and wheelchairs can help maintain mobility and independence.
- **Psychosocial intervention**
- **Gastrointestinal and nutritional management**

Guidelines published for the diagnosis and management of Duchenne's MD essentially states that patients should be assessed/reviewed every 6 months by allied health professionals involved in their multidisciplinary care [17].

There is no specific guidance on how many hours/visits are required for each rehabilitation intervention or clinician.

"Provide direct treatment by physical and occupational therapists, and speech-language pathologists, based on assessments and individualised to the patient."

The above also goes for psychological assessment and intervention. The number of visits will depend on the patient's current needs and ability to cope with their diagnosis.

6 Dementia

6.1 Clinician involved in management

The needs of people with dementia vary widely and tailoring care to each person's circumstances can be complex. A multidisciplinary approach in which different health professionals work together is important [18].

A medical specialist is required to make a dementia diagnosis. These include:

- General physicians
- General practitioners
- Geriatricians
- Neurologists
- Psychiatrists
- Rehabilitation physicians

A number of different allied health professionals may be required at different points in time, including but not limited to [19]:

- Audiologists
- Dentists
- Dietitians
- Occupational therapists
- Orthoptists
- Physiotherapists
- Podiatrists
- Psychologists
- Social workers
- Speech pathologists

Nurses and aged care workers are also involved in the care of patients with dementia.

6.2 Best practice treatment and frequency of intervention

Best practice care has been taken from the UK NICE guidelines on dementia [20]:

1) Person centred care
    a. Involving people in decision making
    b. Providing information
    c. Advance care planning
2) Care coordination
    a. Provide people living with dementia with a single named health or social care professional who is responsible for coordinating their care.
3) Interventions to promote cognition, independence and wellbeing

a. "Offer a range of activities to promote wellbeing that are tailored to the person's preferences" – i.e. previous hobbies/interests
b. Cognitive Stimulation for mild to moderate dementia
    i. Cochrane Review found that intervention ranged from 4 weeks to 24 months [21]. Median session length across the studies was 45 minutes, and the median frequency was three times a week, ranging from one to five times a week. The total possible exposure to the intervention varied dramatically, from 10 to 12 hours to 375 hours in the two-year study. Across the 15 studies, the median exposure time was 30 hours.
c. Group reminiscence therapy for mild to moderate dementia
    i. Cochrane Review concluded that duration and frequency of the sessions could differed. Sessions ranged from 2-8 times at either 1-2 hours (face to face or telephone) and were delivered by occupational therapists, trained recreation therapists [22].
d. Cognitive rehabilitation or occupational therapy for mild to moderate dementia
    i. A Cochrane Review found that intervention duration ranged from 2 to 104 weeks. Sessions ranged from 1-12 per week. More intense was classified as more than 3 formal sessions per week. Duration was 30 to 240 minutes. Those in day care facilities were often longer [23].

NOTE: The Cochrane Collaboration have undertaken various reviews of non-pharmacological interventions for dementia and found that many lack convincing evidence or well described treatment protocols. These include homeopathy, acupuncture, aromatherapy, snoezelen, validation therapy or dance movement therapy.

There is promising evidence that exercise programs may improve the ability to perform ADLs in people with dementia, although some caution is advised in interpreting these findings. Included studies were highly heterogeneous in terms of subtype and severity of participants' dementia, and type, duration, and frequency of exercise [24].

4) Pharmacological interventions
    a. acetylcholinesterase (AChE) inhibitors donepezil, galantamine and rivastigmine as monotherapies are recommended as options for managing mild to moderate disease
5) Caregiver education and skills training
    a. A meta-analysis of 23 randomized clinical trials provides strong confirmation of the benefits of caregiver education and skills training interventions for reducing behavioural symptoms [19]. Collectively, these trials involved 3,279 community-dwelling caregivers and patients. Effective interventions were wide-ranging and included caregiver education, skills training (problem solving, communication strategies), social support (linking caregivers to others), and/or environmental modifications (assistive device use, creating a quiet uncluttered space). Interventions varied in dose, intensity, and delivery mode (telephone, mail, face-to-face, groups, computer technologies.
    b. Successful interventions identified included approximately **nine to 12 sessions** tailored to the needs of the person with dementia and the caregiver and were

delivered individually in the home using multiple components **over 3–6 months** with periodic follow-up [19].

While pharmacological intervention can be conveniently packaged and standardised, with a measured dose, non-pharmacological interventions can be more difficult to evaluate [25]. The same intervention may be used in different studies, but it may comprise quite different components [25]. Non-pharmacological interventions have rarely used a standardised treatment manual; mainly due to the range of individual differences between people with dementia [25].

Although some interventions can be offered for a discrete period of time, such as half an hour per day, many others involve intervention at the level of the care setting or in the general approach or interactive style of those providing care (i.e. depends on disease severity, level or care and care providers) [25].

Frequency of intervention is briefly mentioned in the Australian Clinical Practice Guidelines and Principles of Care for People with Dementia [18]. Statements include:

- *Health system planners should ensure that people with dementia have access to a care coordinator who can work with them and their carer's and families from the time of diagnosis. If more than one service is involved in the person's care, services should agree on one provider as the person's main contact, who is responsible for coordinating care across services at* ***whatever intensity is required****.*
- A care plan developed in partnership with the person and his or her carer(s) and family that ***takes into account the changing needs of the person.***
- ***Formal reviews of the care plan at a frequency agreed between professionals involved*** *and the person with dementia and/or their carer(s) and family.*

7 Huntington's disease

7.1 Clinician involved in management

The multidisciplinary team assesses the stage of the disease and formulates, coordinates and implements the individual care and treatment plan and consists of [26]:

- Physician
- Psychologist
- Speech and language therapist
- Social worker
- Occupational therapist
- Case manager
- Psychologist
- Dentist/oral health specialist

7.2 Best practice treatment and frequency of intervention

Only non-pharmacological recommendations will be presented [27].

Motor Disorders

- Chorea
  - Mouth guards splints.
  - Physiotherapy, OT, speech intervention to assess protective measures.
- Dystonia
  - Active and passive rehabilitation with a physiotherapist to maintain range of movement.
- Rigidity
  - Physiotherapy is recommended to improve or maintain mobility and prevent the development of contractures and joint deformity.
- Swallowing disorders
  - Motor skills training with speech therapist.
  - Psychology for mood, behaviour, emotional status and cognition
  - Provision of information and advice by a dietician, on food textures and consistency and food modifications, bolus size and placement, safe swallowing procedures, elimination of distractions and on focusing attention on just one task at a time can help to avoid aspirations and leads to improvement of swallowing disorders.
- Gait and balance disorders
  - Rehabilitative methods (e.g. physiotherapy and occupational therapy) may improve walking and balance disorders and prevent from their main complications (falls, fractures, loss of autonomy). Interventions for gait and balance should start as early as possible and be continued and adapted throughout the progression of the disease.
  - Supervised low impact exercise.
- Manual dexterity
  - Management with physiotherapy and occupational therapy may be useful to reduce the functional impact of fine motor skill deterioration.
  - OT may suggest adaptive aids to compensate for the deterioration of manual dexterity (adapted cutlery, computer keyboard, adapted telephone, etc.)
- Global motor capacities
  - Referral to a physiotherapist is recommended in order to facilitate the development of a therapeutic relationship, promote sustainable exercise behaviours and ensure long-term functional independence. Exercise programs should be personalized (considering abilities and exercise capacity), goal directed and task specific.
- Cognition
  - Multiple rehabilitation strategies (speech therapy, occupational therapy, cognitive and psychomotricity) might improve or stabilise transitorily cognitive functions (executive functions, memory, language...) at some point of time in the course of the disease.
  - Cognitive stimulation
- Language and communication disorders
  - Communication disorders in HD are variable, requires comprehensive assessment of language and of other factors such as mood, motivation and behaviour.

- Multi-disciplinary input such as Speech & Language Therapy and Physiotherapy help to retain communication and social interaction
- The changing communication needs of the person with HD will be monitored and reassessed throughout the course of the disease to plan effective management strategies at all stages.

- Psychiatric disorders
  - Based on data from other neurodegenerative conditions, mindfulness-based cognitive therapy and Acceptance and Commitment Therapy may be useful.
  - Underlying triggers causing changes in mood or behaviour should be addressed.
  - The duration of treatment is generally for over 6 months and can be for several years

*Unable to find precise data on frequency or duration of interventions for each professional.

8 Arthritis

The main treatment for arthritis is Methotrexate.

The NICE UK guidelines provides the below recommendations [28].

Non-pharmacological management

- Physiotherapy
  - Adults with RA should have access to specialist physiotherapy, with periodic review
  - Improve general fitness and encourage regular exercise
    - 3 to 6 face to face sessions over 3-6 month period [29].
  - Learn exercises for enhancing joint flexibility, muscle strength and managing other functional impairments
  - Learn about the short-term pain relief provided by methods such as transcutaneous electrical nerve stimulators (TENS) and wax baths.
- Occupational therapy
  - Adults with RA should have access to specialist occupational therapy, with periodic review if they have:
    - Difficulties with any of their everyday activities, or
    - Problems with hand function.
- Hand exercise programmes
  - Consider a tailored strengthening and stretching hand exercise programme for adults with RA with pain and dysfunction of the hands or wrists if:
    - They are not on a drug regimen for RA, or
    - They have been on a stable drug regimen for RA for at least 3 months.

The tailored hand exercise programme for adults with RA should be delivered by a practitioner with training and skills in this area.

- Podiatry
  - All adults with RA and foot problems should have access to a podiatrist for assessment and periodic review of their foot health needs.

- Functional insoles and therapeutic footwear should be available for all adults with RA if indicated.

- Psychological interventions
  - Offer psychological interventions (for example, relaxation, stress management and cognitive coping skills [such as managing negative thinking]) to help adults with RA adjust to living with their condition.
  - Meta-analysis of psychological interventions for arthritis pain found that interventions tested were most commonly delivered in a total of nine sessions of 85 min duration, offered on a weekly or biweekly basis [30].
- Diet and complementary therapies
  - Inform adults with RA who wish to experiment with their diet that there is no strong evidence that their arthritis will benefit. However, they could be encouraged to follow the principles of a Mediterranean diet (more bread, fruit, vegetables and fish; less meat; and replace butter and cheese with products based on vegetable and plant oils).
  - Inform adults with RA who wish to try complementary therapies that although some may provide short-term symptomatic benefit, there is little or no evidence for their long-term efficacy.
  - If an adult with RA decides to try complementary therapies, advise them: these approaches should not replace conventional treatment.

Monitoring

Ensure that all adults with RA have:

- Rapid access to specialist care for flares
- Information about when and how to access specialist care, and
- Ongoing drug monitoring.

Consider a review appointment to take place **6 months** after achieving treatment target (remission or low disease activity) to ensure that the target has been maintained.

Offer all adults with RA, including those who have achieved the treatment target, an annual review to:

- Assess disease activity and damage, and
- Measure functional ability (using, for example, the Health Assessment Questionnaire [HAQ]).
- Check for the development of comorbidities, such as hypertension, ischaemic heart disease, osteoporosis and depression.
- Assess symptoms that suggest complications, such as vasculitis and disease of the cervical spine, lung or eyes.
- Organise appropriate cross referral within the multidisciplinary team.

9 Chronic fatigue syndrome

9.1 Clinician involved in management

In most cases, a GP should be able to diagnose chronic fatigue syndrome (CFS). However, if, after a careful history, examination and screening investigations, the diagnosis remains uncertain, the opinion of a specialist physician, adolescent physician or paediatrician should be sought [31].

Other non-medical professionals include:

- Physiotherapists
- Occupational therapists
- Psychologists
- Social workers
- Dieticians

9.2 Best practice treatment and frequency of intervention

Care should be provided to people with CFS using a coordinated multidisciplinary approach. Based on the person's needs, include health and social care professionals with expertise in the following [31, 32]:

- self-management strategies, including energy management
- symptom management
- managing flares and relapse
- activities of daily living
- emotional wellbeing, including family and sexual relationships
- diet and nutrition
- mobility, avoiding falls and problems from loss of dexterity, including access to aids and rehabilitation services
- social care and support
- support to engage in work, education, social activities and hobbies

No detailed information could be sourced around how many hours are required per clinician for each of these approaches. It is clearly stated that service providers should be "adapting the timing, length and frequency of all appointments to the person's needs" [32].

There is still little evidence to support any particular management or intervention for CFS in primary care that can provide an effective early intervention [33]. The only two evidence based therapies recommended by NICE are:

- Cognitive Behavioural Therapy
    - Five to 16 sessions. Sessions ranged from 30 minutes to 150 minutes [34]
    - People with CFS should not undertake a physical activity or exercise programme unless it is delivered or overseen by a physiotherapist or occupational therapist who has training and expertise in CFS [32].
    -

- Exercise Therapy
    - Duration of the exercise therapy regimen varied from 12 weeks to 26 weeks
    - three and five times per week, with a target duration of 5 to 15 minutes per session using different means of incrementation, often exercise at home [35]

10 Chronic pain

This is a very broad area. Treatments depend on location of pain. Musculoskeletal pain, particularly related to joints and the back, is the most common single type of chronic pain.

Information provided in the section on arthritis directly relates to the management of chronic pain.

A substantial systematic review by Skelly, Chou [36] investigated non-pharmacological interventions for chronic pain. Interventions that improved function and/or pain for ≥1 month included:

- Low back pain:
    - Exercise
    - Psychological therapy
    - Spinal manipulation
    - Low-level laser therapy
    - Massage
    - Mindfulness-based stress reduction
    - Yoga
    - Acupuncture
    - Multidisciplinary rehabilitation
- Neck pain
    - Exercise
    - Low-level laser
    - Mind-body practices
    - Massage
    - Acupuncture
- Knee osteoarthritis
    - Exercise
    - CBT
- Hip osteoarthritis
    - Exercise
    - Manual therapies
- Fibromyalgia
    - Exercise
    - CBT
    - Myofascial release massage
    - Mindfulness practices
    - Acupuncture

Substantial variability in the numbers of sessions, length of sessions, duration of treatment, methods of delivering the interventions and the experience and training of those providing the interventions present a challenge to assessing applicability [36].

The range and duration of sessions of interventions are provided below.

- Psychological therapy sessions ranged from six to eight, and the duration of therapy ranged from 6 to 8 weeks
- Exercise therapy ranged from 6 weeks to 12 months, and the number of supervised exercise sessions ranged from 3 to 52.
- Ultrasound therapy was 4 and 8 weeks and the number of sessions was 6 and 10.
- Laser therapy ranged from 2 to 6 weeks and the number of sessions ranged from 10 to 12.
- Manipulation therapy sessions ranged from 4 to 24 and the duration of therapy ranged from 4 to 12 weeks.
- Massage therapy ranged from 2 to 10 weeks and the number of massage sessions ranged from 4 to 24
- Mindfulness based stress reduction 1.5 to 2 hour weekly group sessions for 8 weeks.
- Yoga therapy ranged from 4 to 24 weeks and the number of sessions ranged from 4 to 48.
- Acupuncture therapy ranged from 6 to 12 weeks and the number of acupuncture sessions ranged from 6 to 15.
- Relaxation training and muscle performance exercise therapy were done in 30-minute sessions three times per week for 12 weeks,

11 Amputation

11.1 Clinician involved in management

The Limbs 4 Life is the peak body for amputees in Australia. They provide a list of professionals who assist with rehabilitation of amputees [37].

- Rehabilitation Consultant (doctor)
  - Oversees and coordinates medical care.
- Occupational Therapist
  - Helps adjust to day to day activities like: personal care, domestic tasks such as: meal preparation, accessing your place of residence, driving, education or work readiness. If you are an upper limb amputee the occupational therapist will assist you to set goals, teach you how to perform tasks, explore modifications required to achieve goals (e.g. changes within the home or workplace), explore equipment to assist with completing tasks and assist you with the functional training of your prosthesis.
- Physiotherapist
  - Design a tailored exercise program tailored. They will assist with balance, flexibility, strength and stamina. They will help with mobility aids such as: wheelchairs, walking frames, crutches and other assistive devices.
- Prosthetist

- Will look after the design, manufacture, supply and fit of the prosthesis. Together, you will discuss and decide on the prosthetic components to suit your needs and lifestyle.

- Psychologist
  - Supports individuals and fosters positive mental health outcomes and personal growth.
- Nursing team
  - Assists with your medications, personal hygiene, bathing and dressing and any wound care and diabetic management that is required.
- Dietitian
- Podiatrist

11.2 Best practice treatment and frequency of intervention

Physiotherapy
The physiotherapist progresses the patient through a programme based on continuous assessment and evaluation [38]. Through regular assessment, the physiotherapist should identify when the individual has achieved optimum function with a prosthesis, facilitating discharge to a maintenance programme.

The consensus opinion is that the physiotherapist should contribute to the management of wounds, scars, residual limb pain and phantom pain and sensation together with other members of the multidisciplinary team [38].

During prosthetic rehabilitation *patients should receive physiotherapy as often as their needs and circumstances dictate* [38].

Occupational therapy

The occupational therapy practitioner provides critical interventions, such as [39]"

- identifying the client's functional goals, which can include self-care, home management, work tasks, driving, child care, and leisure activities, and offering modifications to complete these goals if required
- analysing tasks and providing modifications to achieve functional goals
- providing education on compensatory techniques and equipment to accomplish tasks and activities
- providing prosthetic training
- identifying and addressing psychosocial issues

Occupational therapy intervention will vary according to individual needs, and phases of intervention may overlap, depending on the person's progress [39].

The administration of interventions for phantom limb have been shown to range between one day and 12 weeks, with one to five sessions per week [40] .

Psychology

Counselling and psychological support is available to the person and their valued others preoperatively and continues as part of lifelong management [41].

Experienced clinical counselling and psychological support should be available to assist with issues such as adjustment and pain management from the acute phase, and throughout lifelong management [41].

Psychosocial issues are evaluated and addressed as part of the overall treatment plan and reviewed regularly throughout the care journey [41].

No information could be sourced about how many sessions are required.

12 References

1. Rajan R, Brennan L, Bloem BR, Dahodwala N, Gardner J, Goldman JG, et al. Integrated Care in Parkinson's Disease: A Systematic Review and Meta-Analysis. Movement Disorders [Internet]. 2020 2020/09/01; 35(9):[1509-31 pp.]. Available from: https://doi.org/10.1002/mds.28097.
2. National Institute for Health and Care Excellence (NICE). Parkinson's disease in adults. 2017. Available from: https://www.nice.org.uk/guidance/ng71/resources/parkinsons-disease-in-adults-pdf-1837629189061.

3. Tomlinson CL, Patel S, Meek C, Herd CP, Clarke CE, Stowe R, et al. Physiotherapy versus placebo or no intervention in Parkinson's disease. Cochrane Database of Systematic Reviews [Internet]. 2013; (9). Available from: https://doi.org//10.1002/14651858.CD002817.pub4.
4. Herd CP, Tomlinson CL, Deane KHO, Brady MC, Smith CH, Sackley CM, et al. Speech and language therapy versus placebo or no intervention for speech problems in Parkinson's disease. Cochrane Database of Systematic Reviews [Internet]. 2012; (8). Available from: https://doi.org//10.1002/14651858.CD002812.pub2.
5. Dixon L, Duncan DC, Johnson P, Kirkby L, O'Connell H, Taylor HJ, et al. Occupational therapy for patients with Parkinson's disease. Cochrane Database of Systematic Reviews [Internet]. 2007; (3). Available from: https://doi.org//10.1002/14651858.CD002813.pub2.
6. The Association of UK Dieticians. Best practice guidance for dietitians on the nutritional management of Parkinson's. 2021. Available from: https://www.parkinsons.org.uk/sites/default/files/2021-02/Best%20practice%20guidance%20for%20dietitians%20on%20the%20nutritional%20management%20of%20Parkinson%27s%20FINAL.pdf.

7. Deane K, Whurr R, Clarke CE, Playford ED, Ben-Shlomo Y. Non-pharmacological therapies for dysphagia in Parkinson's disease. Cochrane Database of Systematic Reviews [Internet]. 2001; (1). Available from: https://doi.org//10.1002/14651858.CD002816.
8. Dix K, Green H. Defining the value of Allied Health Professionals with expertise in Multiple Sclerosis. 2013. Available from: https://support.mstrust.org.uk/file/defining-the-value-AHPs.pdf.

9. (NICE) NIfHaCE. Multiple sclerosis in adults: management. 2019. Available from: https://www.nice.org.uk/guidance/cg186/resources/multiple-sclerosis-in-adults-management-pdf-35109816059077.

10. Khan F, Turner-Stokes L, Ng L, Kilpatrick T, Amatya B. Multidisciplinary rehabilitation for adults with multiple sclerosis. Cochrane Database of Systematic Reviews [Internet]. 2007; (2). Available from: https://doi.org//10.1002/14651858.CD006036.pub2.
11. Rosti-Otajärvi EM, Hämäläinen PI. Neuropsychological rehabilitation for multiple sclerosis. Cochrane Database of Systematic Reviews [Internet]. 2014; (2). Available from: https://doi.org//10.1002/14651858.CD009131.pub3.

12. Hayes S, Galvin R, Kennedy C, Finlayson M, McGuigan C, Walsh CD, et al. Interventions for preventing falls in people with multiple sclerosis. Cochrane Database of Systematic Reviews [Internet]. 2019; (11). Available from: https://doi.org//10.1002/14651858.CD012475.pub2.
13. Mayo Foundation for Medical Education and Research. Muscular Dystrophy 2021 [Available from: https://www.mayoclinic.org/diseases-conditions/muscular-dystrophy/symptoms-causes/syc-20375388.
14. Voet NBM, van der Kooi EL, van Engelen BGM, Geurts ACH. Strength training and aerobic exercise training for muscle disease. Cochrane Database of Systematic Reviews [Internet]. 2019; (12). Available from: https://doi.org//10.1002/14651858.CD003907.pub5.
15. Birnkrant DJ, Bushby K, Bann CM, Alman BA, Apkon SD, Blackwell A, et al. Diagnosis and management of Duchenne muscular dystrophy, part 2: respiratory, cardiac, bone health, and orthopaedic management. The Lancet Neurology [Internet]. 2018 2018/04/01/; 17(4):[347-61 pp.]. Available from: https://www.sciencedirect.com/science/article/pii/S1474442218300255.
16. Birnkrant DJ, Bushby K, Bann CM, Apkon SD, Blackwell A, Colvin MK, et al. Diagnosis and management of Duchenne muscular dystrophy, part 3: primary care, emergency management, psychosocial care, and transitions of care across the lifespan. The Lancet Neurology [Internet]. 2018 2018/05/01/; 17(5):[445-55 pp.]. Available from: https://www.sciencedirect.com/science/article/pii/S1474442218300267.
17. Birnkrant DJ, Bushby K, Bann CM, Apkon SD, Blackwell A, Brumbaugh D, et al. Diagnosis and management of Duchenne muscular dystrophy, part 1: diagnosis, and neuromuscular, rehabilitation, endocrine, and gastrointestinal and nutritional management. The Lancet Neurology [Internet]. 2018 2018/03/01/; 17(3):[251-67 pp.]. Available from: https://www.sciencedirect.com/science/article/pii/S1474442218300243.
18. Guideline Adaptation Committee. Clinical Practice Guidelines and Principles of Care for People with Dementia. Sydney: Guideline Adaptation Committee; 2016. Available from: https://cdpc.sydney.edu.au/wp-content/uploads/2019/06/CDPC-Dementia-Guidelines_WEB.pdf.

19. Brodaty H, Arasaratnam C. Meta-Analysis of Nonpharmacological Interventions for Neuropsychiatric Symptoms of Dementia. American Journal of Psychiatry [Internet]. 2012 2012/09/01; 169(9):[946-53 pp.]. Available from: https://doi.org/10.1176/appi.ajp.2012.11101529.
20. National Institute for Health Care Excellence. National Institute for Health and Care Excellence: Clinical Guidelines. Dementia: Assessment, management and support for people living with dementia and their carers. London: National Institute for Health and Care Excellence (UK)

21. Woods B, Aguirre E, Spector AE, Orrell M. Cognitive stimulation to improve cognitive functioning in people with dementia. Cochrane Database of Systematic Reviews [Internet]. 2012; (2). Available from: https://doi.org//10.1002/14651858.CD005562.pub2.
22. Möhler R, Renom A, Renom H, Meyer G. Personally tailored activities for improving psychosocial outcomes for people with dementia in community settings. Cochrane Database of Systematic Reviews [Internet]. 2020; (8). Available from: https://doi.org//10.1002/14651858.CD010515.pub2.
23. Bahar-Fuchs A, Martyr A, Goh AMY, Sabates J, Clare L. Cognitive training for people with mild to moderate dementia. Cochrane Database of Systematic Reviews. 2019(3).
24. Forbes D, Forbes SC, Blake CM, Thiessen EJ, Forbes S. Exercise programs for people with dementia. Cochrane Database of Systematic Reviews [Internet]. 2015; (4). Available from: https://doi.org//10.1002/14651858.CD006489.pub4.

25. National Collaborating Centre for Mental Health. Dementia: A NICE-SCIE guideline on supporting people with dementia and their carers in health and social care: British Psychological Society; 2007. Dementia. 2014.
26. Veenhuizen RB, Kootstra B, Vink W, Posthumus J, van Bekkum P, Zijlstra M, et al. Coordinated multidisciplinary care for ambulatory Huntington's disease patients. Evaluation of 18 months of implementation. Orphanet J Rare Dis [Internet]. 2011; 6:[77- pp.]. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3253686/.
27. Bachoud-Lévi A-C, Ferreira J, Massart R, Youssov K, Rosser A, Busse M, et al. International Guidelines for the Treatment of Huntington's Disease. Frontiers in Neurology [Internet]. 2019 2019-July-03; 10(710). Available from: https://www.frontiersin.org/article/10.3389/fneur.2019.00710.
28. National Institute for Health and Care Excellence (NICE). Rheumatoid arthritis in adults: management. 2018.
29. Peter WF, Swart NM, Meerhoff GA, Vliet Vlieland TPM. Clinical Practice Guideline for Physical Therapist Management of People With Rheumatoid Arthritis. Physical Therapy [Internet]. 2021. Available from: https://doi.org/10.1093/ptj/pzab127.
30. Dixon KE, Keefe FJ, Scipio CD, Perri LM, Abernethy AP. Psychological interventions for arthritis pain management in adults: A meta-analysis. Health Psychology [Internet]. 2007; 26(3):[241-50 pp.].
31. Working Group of the Royal Australasian College of Physicians. Chronic fatigue syndrome. Clinical practice guidelines--2002. Med J Aust [Internet]. 2002 May 6; 176(S9):[S17-s55 pp.].
32. National Institute for Health and Care Excellence (NICE). Myalgic encephalomyelitis (or encephalopathy)/chronic fatigue syndrome: diagnosis and management. 2020. Available from: https://www.nice.org.uk/guidance/gid-ng10091/documents/draft-guideline.

33. Hughes JL. Chronic Fatigue Syndrome and Occupational Disruption in Primary Care: Is There a Role for Occupational Therapy? British Journal of Occupational Therapy [Internet]. 2009 2009/01/01; 72(1):[2-10 pp.]. Available from: https://doi.org/10.1177/030802260907200102.
34. Price JR, Mitchell E, Tidy E, Hunot V. Cognitive behaviour therapy for chronic fatigue syndrome in adults. Cochrane Database of Systematic Reviews [Internet]. 2008; (3). Available from: https://doi.org//10.1002/14651858.CD001027.pub2.
35. Larun L, Brurberg KG, Odgaard-Jensen J, Price JR. Exercise therapy for chronic fatigue syndrome. Cochrane Database of Systematic Reviews [Internet]. 2019; (10). Available from: https://doi.org//10.1002/14651858.CD003200.pub8.
36. Skelly AC, Chou R, Dettori JR, Turner JA, Friedly JL, Rundell SD, et al. Agency for Healthcare Research and Quality: Comparative Effectiveness Reviews. 2020. In: Noninvasive Nonpharmacological Treatment for Chronic Pain: A Systematic Review Update [Internet]. Rockville (MD): Agency for Healthcare Research and Quality (US). Available from: https://www.ncbi.nlm.nih.gov/books/NBK556229/.
37. Limbs 4 Life. Your recovery 2021 [Available from: https://www.limbs4life.org.au/steps-to-recovery/your-rehabilitation-team.
38. Broomhead P, Dawes D, Hale C, Lambert A, Quinlivan D, Shepherd R. Evidence based clinical guidelines for the physiotherapy management of adults with lower limb prostheses. British Association of Chartered Physiotherapists in Amputation Rehabilitation; 2003. Available from: https://docuri.com/download/csp-guideline-bacpar_59c1cb4df581710b2860eb11_pdf.

39. Gulick K. The occupational therapy role in rehabilitation for the person with an upper-limb amputation. American Occupational Therapy Association [Internet]. 2007. Available from:

https://www.aota.org/About-Occupational-Therapy/Professionals/RDP/upper-limb-amputation.aspx.
40. Othman R, Mani R, Krishnamurthy I, Jayakaran P. Non-pharmacological management of phantom limb pain in lower limb amputation: a systematic review. Physical Therapy Reviews [Internet]. 2018 2018/03/04; 23(2):[88-98 pp.]. Available from: https://doi.org/10.1080/10833196.2017.1412789.
41. Innovation AfC. ACI Care of the Person following Amputation: Minimum Standards of Care. Australia; 2017. Available from: https://aci.health.nsw.gov.au/__data/assets/pdf_file/0019/360532/The-care-of-the-person-following-amputation-minimum-standards-of-care.pdf.

yResearch – Ehlers Danlos Syndrome

Brief	Please provide information on the condition: Ehlers Danlos. I understand that there are two variants to this condition. What are the diagnostic markers, how does it present functionally? What are the evidence based treatments for this condition? What is the incidence of EDS as a primary condition? What kinds of conditions may comorbidly exist? Also please provide a list of possible experts in EDS in Australia.
Date	08/06/2021
Requester(s)	Shannon s47F - personal – Assistant Director (TAB/AAT)
Researcher	Jane s47F - personal priv - Research Team Leader (TAB)
Cleared	

Please note:

The research and literature reviews collated by our TAB Research Team are not to be shared external to the Branch. These are for internal TAB use only and are intended to assist our advisors with their reasonable and necessary decision-making.

Delegates have access to a wide variety of comprehensive guidance material. If Delegates require further information on access or planning matters they are to call the TAPS line for advice.

The Research Team are unable to ensure that the information listed below provides an accurate & up-to-date snapshot of these matters.

The contents of this document are OFFICIAL

1 Contents

2 Types and Clinical Presentation

Ehlers-Danlos syndrome (EDS) is a heterogeneous group of inheritable connective tissue disorders attributed to mutations in connective tissue genes [1]. These mutations cause defects in collagen. Collagen, a connective tissue protein that acts like glue, gives strength to the body and provides support and elasticity for movement. Thus, the altered gene affects the mechanical properties of skin, joints, ligaments, and blood vessels [1].

The International EDS Consortium provided a revised classification of EDS in 2017 which includes 13 subtypes [2, 3]. Vast genetic heterogeneity and phenotypic variability exists, and subtypes often overlap [3]. The condition is often diagnosed based upon the family history and clinical criteria, including the degree and nature of involvement of skin, joints, skeleton, and vasculature [3, 4]. Each subtype and their characteristic manifestations are provided below.

2.1 Classical

Classical EDS (formerly EDSI and EDSII) is associated with the primary problems such as:

- Skin hyper-extensibility
- Joint laxity
- Fragile blood vessels.

Scars are very thin, discoloured, and stretch with time. Such paper-like (papyraceous) scarring occurs especially over prominent bony pressure points such as the knees, elbows, shins and forehead. Joint hypermobility accidents (subluxations and dislocations) are generally easily managed. Additional findings may include the formation of small, fleshy, skin growths called ‘molluscoid pseudo-tumours’ or hard, round, movable lumps under the skin called ‘calcified spheroids’.

Some individuals with this subtype may have heart complications including

- Deformity of heart valves (especially the mitral/bicuspid valve between the atrium and the ventricle of the left side).
- Valve dysfunction can result in remodelling of the heart’s architecture and with time congestive heart failure (pump insufficiency).
- Dilatation of the aorta and increased risk also for aortic dissection. Aortic dissection is an immediate emergency that can result in acute heart failure.

2.2 Classical-like

Classical-like EDS is similar in clinical course to classical EDS (described above). However, genetic causes of classical EDS and classical-like EDS differ (described below).

2.2.1 Cardiac-valvular type

Cardiac-valvular type EDS is a rare subtype of EDS wherein patients may have minor signs of EDS with severe defects to their aorta, requiring surgical interventions.

2.2.2 Vascular type

Vascular type EDS (formerly EDSIV), can be identified at birth with:

In childhood, and pneumothorax (collection of air between the lung and chest wall, impairing proper lung inflation) are commonly experienced and are indicative of this syndrome. Individuals with vascular EDS may also have:

- Noticeable clubfoot deformities
- Dislocation of the hips
- Inguinal hernia (partial slip of intestine beyond the abdominal wall)
- Pneumothorax (collection of air between the lung and chest wall, impairing proper lung inflation)
- Abnormally decreased levels of fatty tissue under skin layers (subcutaneous adipose tissue) of the hands, arms, legs, feet, and face. Thus, some affected individuals may have a characteristic facial appearance. Cheeks are often taught and hollow. Lips and nose are often thin. Eyes are relatively prominent
- Skin of the hands and feet may appear prematurely aged (acrogeria).
- Arterial dissection and rupture, intestinal perforation, and uterine rupture.
- Carotid-cavernous sinus fistula
- Severe headaches
- Seizures
- Increased risk of stroke
- Early onset of varicose veins
- Acute pain in the abdominal or flank area may indicate arterial or intestinal rupture
- Pregnancies should be considered higher risk and watch closely for arterial and uterine ruptures

The median life expectancy for vascular EDS is 50, but with careful surveillance and management of complications this age can be well extended.

2.2.3 Hypermobility type

Hypermobility type EDS (formerly EDSIII) comes with a defined set of complications to be managed but is generally a less severe form of the syndrome. For example, aortic root dilation is usually minimal and does not significantly increase the risk for dissections. The major complications to patients with hypermobility EDS are musculoskeletal in nature.

- Frequent joint dislocation.

- Degenerative joint disease are common and associated with a baseline chronic pain, which affects both physical and psychological wellbeing.
- Problems with the autonomic nervous system, responsible for regulating body functions and the fight-or-flight response, are common.
    - Orthostatic intolerance, significant light-headedness on standing, due to a slowed response by their circulatory system to compensation against blood pressure and flow changes with shifts in body position.
- Bowel disorders are also more common with this condition, especially functional dyspepsia (indigestion), and irritable bowel syndrome.
- Psychological impairment and mood problems.

2.2.4 Arthrochalasia type

Arthrochalasia EDS (formerly EDSVII, A and B) is associated with the lifelong risk for the dislocation of multiple major joints concurrently. This condition makes achieving mobility significantly challenging. It is important to identify as early in life as possible as it carries consequences of physical disability with older age. Newborns may demonstrate severe muscular hypotonia and a bilateral dislocation of the hips at birth and might be difficult to distinguish from Kyphoscoliotic EDS (described below).

2.2.5 Dermatosparaxis type

Patient with dermatosparaxis EDS (formerly EDSVIIC) tend to show a set of common body features. These include:

- Short stature and finger length
- Loose skin of the face, with comparatively full eyelids
- Blue-tinged whites of the eye (sclera)
- Skin folds in the upper eyelids (epicanthal folds)
- Downward slanting outer corners of the eyes (palpebral fissures)
- Small jaw (micrognathia).

A major complication of dermatosparaxis EDS is herniation, the improper displacement of an organ through the tissues holding it in proper position. Hernias are especially common after certain surgeries, for example wherein there is an incision into the muscles of the abdomen. Due to the lengthy wound-healing process, intestinal contents may bulge through incisions. Patients with dermatosparaxis EDS are also prone to ruptures in the diaphragm and bladder.

2.2.6 Kyphoscoliotic type

Kyphoscoliotic EDS is accompanied by:

- Scleral fragility, increasing the risk for rupture of the white globe of the eye
- Microcornea,
- Near-sightedness (myopia)
- Glaucoma

- Retinal detachment, flashes and floaters

Kyphoscoliotic EDS can be evident at birth. Newborns may demonstrate severe muscular weakness (hypotonia) or abnormal spinal rotations and curvatures (scoliosis). Despite progressive scoliosis, the survival of patients with Kyphoscoliotic EDS is unaffected. The most severely affected adults may lose the ability to walk by their 20s-30s and it becomes important to watch that their scoliosis does not begin to impede normal breathing patterns.

2.2.7 Brittle cornea syndrome

Brittle cornea syndrome (BCS) is a variant of EDS that also involves the eyes. People with variant risk ruptures to the cornea following minor injuries with scarring, degeneration of the cornea (keratoconus), and protrusion of the cornea (keratoglobus). Patients may have blue sclera.

2.2.8 Spondylodysplastic type

Spondylodysplastic EDS, previously spondylocheirodysplastic type, describes an EDS variant with skeletal dysmorphology. It primarily involves the spine and the hands. Clinical presentation can include:

- Stunted growth
- Short stature
- Protuberant eyes with bluish sclera
- Wrinkled skin of the palms
- Atrophy of muscles at the base of the thumb (thenar muscles)
- Tapering fingers.

2.2.9 Musculocontractural type

Musculocontractural EDS is characterized by progressive multisystem complications. This subtype is especially associated with:

- Developmental delay
- Muscular weakness plus hypotonia
- Facial and cranial structural defects
- Congenital contractures of the fingers
- Severe kyphoscoliosis
- Muscular hypotonic
- Club foot deformity
- Ocular problems

2.2.10 Myopathic type

Myopathic EDS is characterized by:

- Muscle hypotonia evident at birth with muscles that do not function properly (myopathy)

- Scoliosis
- Sensorineural hearing impairment may accompany this condition. It shares many features with the kyphoscoliotic form of EDS.

2.2.11 Periodontal type

Periodontal EDS type (formerly EDS VII) has findings that include disease of the tissues surrounding and supporting the teeth (periodontal disease), potentially resulting in premature tooth loss.

3 Evidence Based Treatments

The treatment or management of EDS will depend on the type diagnosed. It can be debilitating to patients and challenging to manage. Multidisciplinary treatment is required, including mental health support for declining psychological wellbeing. There is very little published evidence on in this area and treatments are often based on consensus, expert opinion and case studies [5]. Some broad options relating to common manifestations will be provided below [2, 4, 5].

3.1 Treatment of manifestations

- Physical therapy tailored to the individual
- Assistive devices
    - Braces to improve joint stability
    - Wheelchair or scooter to offload stress on lower-extremity joints
    - Suitable mattress to improve sleep quality
- Pain medication tailored to symptoms
- Appropriate therapy for gastritis/reflux/delayed gastric emptying/irritable bowel syndrome
- Psychological and/or pain-oriented counselling.

3.2 Prevention of primary manifestations

Low-resistance exercise to increase both core and extremity muscle tone for improved joint stability; appropriate writing utensils to reduce finger and hand strain.

3.3 Prevention of secondary complications

Calcium, vitamin D, low-impact weight-bearing exercise to maximize bone density.

3.4 Surveillance

Dual-energy X-ray absorptiometry (DEXA) every other year if bone loss is confirmed.

Agents/circumstances to avoid

High-impact activity increases the risk of acute subluxation/dislocation, chronic pain, and osteoarthritis.

3.5 Genetic counselling

Hypermobile EDS is inherited in an autosomal dominant manner. Most individuals diagnosed with the syndrome have an affected parent. The proportion of cases caused by a *de novo* pathogenic variant is unknown. Each child of an individual with hypermobile EDS has a 50% chance of inheriting the disorder. Because the gene(s) and pathogenic variant(s) responsible for hypermobile EDS have not been identified, prenatal testing is not possible.

3.6 Surgery

Non-operative treatment should be maximized before surgery is indicated. This is because excessive bleeding after can occur after minor trauma or surgery.

4 Prevalence

The estimated overall prevalence of EDS is 1 in 5000, depending on type (hypermobility type is the most common) [1]. It affects males and females of all racial and ethnic backgrounds equally. Classical-type EDS occurs in 1 in 10,000 to 1 in 20,000 infants [1].

In individuals affected by vascular-type EDS, the average age for arterial rupture is 23 years and the median age of death is 48 years [1].

5 Co-existing Conditions

Multiple EDS website list various coexisting conditions but provide no supporting evidence. These include:

- Malfunctioning of the autonomic nervous system
- Gut dysmotility
- Chronic fatigue
- Small fibre neuropathy
- Cervico-cranial instability
- Sleep disorders

There is evidence to suggest that individuals with EDS are at increased risk of being diagnosed with psychiatric disorders [6]. These risk increases may have a genetic and/or early environmental background [6, 7].

Individuals with EDS have an overall 4.3x relative risk of being affected by any psychiatric disorder. Some of these include [6]:

- Autism spectrum disorder
- Bipolar disorder
- Attention deficit hyperactivity disorder
- Obsessive compulsive disorder
- Depression
- Attempted suicide
- Suicide
- Schizophrenia

A population based cohort study from Denmark is the first and only study to investigate comorbidities in patient with EDS [8]. This study found that the most common in Danish patients were:

- Gastrointestinal functional disorders
- Hernias
- Asthma
- Pneumonia
- Osteoporosis.

Other conditions such as epilepsy [9], migraine headache [9], cerebrovascular disease [10], postural orthostatic tachycardia syndrome [11], fibromyalgia [12] and diverticular diseases [12] have also been associated with EDS.

6 List of possible experts in Australia

A healthcare professional's directory is provided by the Ehlers-Danlos Society. This includes medical doctors, physiotherapists, occupational therapists, podiatrists and dentists. Below are a selection of medical professionals.

Raj Anand, MBBS, FRACP (General Medicine/ Rheumatology) FFPMANZCA
Rheumatology, Pain Medicine

Eastern Suburbs Pain Clinic
Specialist Medical Randwick
Barker Street
Suite 20, Level 7
Prince of Wales Private Hospital
NSW Randwick
0061 2 9650 4988
reception@pwphealth.com.au

Felicity Collins, MBBS
Clinical Genetics; Paediatrics

Clinical genetics Unit, Royal Prince Alfred Hospital
Missenden Rd, Camperdown
Sydney, NSW 2050
+612-95155080
felicity.collins@health.nsw.gov.au

Louise Tofts, MBBS, DCH, MSpMed, FRACP, FAFRM
Paediatrics, Physical Medicine and Rehabilitation
Narrabeen Sports Medicine Center
Wakehurst Parkway
Narabeen, NSW 2101
http://narrabeensportsmedicine.com.au/paediatrics/dr-louise-tofts/
+61 2 99711188
admin@narrabeensportsmedicine.com.au

Pavla Walsh, MBBS
FRACP, FFPMANZCA, Grad Dip Paed Rheum
Painless
5/136 Railway St
Cottesloe, WA 6011
1300429411
hello@painless.clinic

7 References

1. Lawrence EJ. The Clinical Presentation of Ehlers-Danlos Syndrome. Advances in Neonatal Care. 2005;5(6).
2. National Organisation for Rare Disorders. Ehlers Danlos Syndromes 2017 [Available from: https://rarediseases.org/rare-diseases/ehlers-danlos-syndrome/.
3. Malfait F, Francomano C, Byers P, Belmont J, Berglund B, Black J, et al. The 2017 international classification of the Ehlers–Danlos syndromes. American Journal of Medical Genetics Part C: Seminars in Medical Genetics [Internet]. 2017 2017/03/01; 175(1):[8-26 pp.]. Available from: https://doi.org/10.1002/ajmg.c.31552.
4. Levy HP. Hypermobile Ehlers-Danlos Syndrome: University of Washington, Seattle, Seattle (WA); 1993. Available from: https://www.ncbi.nlm.nih.gov/books/NBK1279.
5. Shirley ED, DeMaio M, Bodurtha J. Ehlers-Danlos Syndrome in Orthopaedics: Etiology, Diagnosis, and Treatment Implications. Sports Health [Internet]. 2012 2012/09/01; 4(5):[394-403 pp.]. Available from: https://doi.org/10.1177/1941738112452385.
6. Cederlöf M, Larsson H, Lichtenstein P, Almqvist C, Serlachius E, Ludvigsson JF. Nationwide population-based cohort study of psychiatric disorders in individuals with Ehlers–Danlos syndrome or hypermobility syndrome and their siblings. BMC Psychiatry [Internet]. 2016 2016/07/04; 16(1):[207 p.]. Available from: https://doi.org/10.1186/s12888-016-0922-6.

7. Sinibaldi L, Ursini G, Castori M. Psychopathological manifestations of joint hypermobility and joint hypermobility syndrome/ Ehlers–Danlos syndrome, hypermobility type: The link between connective tissue and psychological distress revised. American Journal of Medical Genetics Part C: Seminars in Medical Genetics. 2015;169(1):97-106.
8. Leganger J, Fonnes S, Kulas Søborg ML, Rosenberg J, Burcharth J. The most common comorbidities in patients with Ehlers-Danlos syndrome: a 15-year nationwide population-based cohort study. Disability and Rehabilitation [Internet]. 2020:[1-5 pp.]. Available from: https://doi.org/10.1080/09638288.2020.1761890.
9. Castori M, Voermans NC. Neurological manifestations of Ehlers-Danlos syndrome (s): A review. Iranian journal of neurology [Internet]. 2014; 13(4):[190 p.]. Available from: https://www-ncbi-nlm-nih-gov.ez.library.latrobe.edu.au/pmc/articles/PMC4300794/.
10. Kim ST, Cloft H, Flemming KD, Kallmes DF, Lanzino G, Brinjikji W. Increased Prevalence of Cerebrovascular Disease in Hospitalized Patients with Ehlers–Danlos Syndrome. Journal of Stroke and Cerebrovascular Diseases [Internet]. 2017 2017/08/01/; 26(8):[1678-82 pp.]. Available from: https://www.sciencedirect.com/science/article/pii/S1052305717301301.
11. Sobey G. Ehlers–Danlos syndrome: how to diagnose and when to perform genetic tests. Archives of Disease in Childhood [Internet]. 2015; 100(1):[57 p.]. Available from: http://adc.bmj.com/content/100/1/57.abstract.
12. Nelson AD, Mouchli MA, Valentin N, Deyle D, Pichurin P, Acosta A, et al. Ehlers Danlos syndrome and gastrointestinal manifestations: a 20-year experience at Mayo Clinic. Neurogastroenterology & Motility [Internet]. 2015 2015/11/01; 27(11):[1657-66 pp.]. Available from: https://doi.org/10.1111/nmo.12665.

Research Request – Postural Orthostatic Tachycardia Syndrome

Brief	Known available and appropriate evidence based clinical, medical or other treatments for Postural Orthostatic Tachycardia Syndrome
Date	05/10/2020
Requester	Wendy s47F - persb (Assistant Director TAB) Naomi s47F - personal pr (Senior Technical Advisor TAB)
Researcher	Jane s47F - personal priv (Research Team Leader TAB)

Contents

Please note:

The research and literature reviews collated by our TAB Research Team are not to be shared external to the Branch. These are for internal TAB use only and are intended to assist our advisors with their reasonable and necessary decision making.

Delegates have access to a wide variety of comprehensive guidance material. If Delegates require further information on access or planning matters they are to call the TAPS line for advice.

The Research Team are unable to ensure that the information listed below provides an accurate & up-to-date snapshot of these matters

Summary

- Postural Orthostatic Tachycardia Syndrome (POTS) is a variant of cardiovascular autonomic disorder characterised by orthostatic intolerance and abnormal heart rate response on standing associated with symptoms of deconditioning, cognitive impairment and a myriad of unspecific symptoms
- The aetiology of POTS is unknown and predominantly effects young females
- Patient education is key to managing the symptoms of POTS
- Exercise training and acute saline infusion are the only treatments with Class IIA recommendations ('should be considered')
- Other treatments that 'may be considered' include:
    - Increased fluid and salt intake
    - Midodrine
    - Beta-blockers
    - Fludrocortisone
    - Pyridostigmine, clonidine
    - Alpha-methyldopa

What is Postural Orthostatic Tachycardia Syndrome?

Postural orthostatic tachycardia syndrome (POTS) is a common, although not so well-known variant of cardiovascular autonomic disorder. It is characterised by an excessive heart rate increase on standing, symptoms of orthostatic intolerance and occasional syncope. [1, 2] The syndrome affects younger individuals, with a distinct predominance of women (>80%). [3, 4] The prevalence estimates are imprecise and range between 0.2% and 1.0% based on statistics from the United States. [3, 4]

The onset of POTS is typically precipitated by immunological stressors such as viral infection, vaccination, trauma, pregnancy, surgery or psychosocial stress. [5-13] The aetiology of POTS is largely unknown, however, there are three main hypotheses which include an autoimmune disorder, abnormally increased sympathetic activity and catecholamine excess, and sympathetic denervation leading to central hypovolaemia and reflex tachycardia. [14]

Diagnostic criteria

The current diagnostic criteria for POTS is summarised in Table 1 below and has been endorsed by the American Academy of Neurology, American Autonomic Society, American College of Cardiology, American Heart Association, European Federation of Autonomic Societies, European Heart Rhythm Association, European Society of Cardiology and Heart Rhythm Society. [1, 3, 15, 16]

Table 1: Diagnostic criteria of postural orthostatic tachycardia syndrome

Sustained heart rate increment of not less than 30 beats min or above 120 beats min within 10 min of active standing or head-up tilt

o For individuals who are younger than 19 years the required increment is at least 40 beats min
Absence of orthostatic hypotension (i.e. sustained systolic blood pressure drop of not less than 20 mmHg)
Reproduction of spontaneous symptoms such as light-headedness, palpitations, tremulousness, generalized weakness, blurred vision and fatigue. In some patients, tachycardia may evoke vasovagal syncope corresponding to spontaneous attacks from patient's history
History of chronic orthostatic intolerance and other typical POTS associated symptoms (for at least 6 months[a])
Absence of other conditions provoking sinus tachycardia such as anxiety disorders, hyperventilation, anaemia, fever, pain, infection, dehydration, hyperthyroidism, pheochromocytoma, use of cardio active drugs (sympathomimetic, anticholinergics)

[a] *This criterion may be controversial and is not unanimously accepted as patients may seek medical advice earlier due to increasing awareness of the syndrome. However, symptoms of shorter duration than 3 months should be re-evaluated to confirm the diagnosis.*

Clinical presentation

A characteristic patient with POTS is a young woman approximately 25 years of age, reporting first symptoms at around 15 years, although the range of disease onset may extend until the fifth decade of life and 20% are men. [15] Common clinical symptoms of POTS can be found in Table 2. [15]

Table 2 Clinical presentation of POTS

Cardiovascular system	**Main:** Orthostatic intolerance, orthostatic tachycardia, palpitations, dizziness, light-headedness, Pre-syncope, exercise intolerance **Other frequent symptoms:** dyspnoea, chest pain/discomfort, acrocyanosis, Raynaud's phenomenon, venous pooling, limb oedema
General symptoms	General deconditioning, chronic fatigue, exhaustion, heat intolerance, fever, debility, bedridden
Nervous system	Headache/migraine, mental clouding ('brain fog'), cognitive impairment, concentration problems, anxiety, tremulousness, light and sound sensitivity, blurred/tunnel vision, neuropathic pain (regional), sleeping disorders, involuntary movements
Musculoskeletal system	Muscle fatigue, weakness, muscle pain
Gastrointestinal system	Nausea, dysmotility, gastroparesis, constipation, diarrhoea, abdominal pain, weight loss
Respiratory system	Hyperventilation, bronchial asthma, shortness of breath
Urogenital system	Bladder dysfunction, nycturia, polyuria
Skin	Petechiae, rashes, erythema, telangiectasias, abnormal sudomotor regulation, diaphoresis, pallor, flushing

Management

The heterogeneity and wide spectrum of POTS related symptoms create a great challenge for clinicians and affected patients. Moreover, poorly explored pathophysiology of POTS makes it literally impossibly to treat the root cause of the disease and the management of POTS is usually focused on symptom alleviation. [17, 18] The long-term prognosis of POTS is not well explored. It is estimated that around 50% of all POTS patients **spontaneously recover within 1–3 years**. [15]

Once a diagnosis has been made, patients should be thoroughly educated about non-pharmacological measures to alleviate symptoms, long term prognosis and available therapeutic options (see Table 3). It should be noted that **large randomized trials investigating POTS treatments are not available** [19] and there are no Class I (is recommended) recommendations to date. [3] The only Class IIA (should be considered) recommendations are exercise training against chronic symptoms and acute saline infusion in decompensated POTS [3] both lead to positive effects on plasma volume. [20] Among Class IIB (may be considered) recommendations are increased fluid and salt intake, midodrine, beta-blockers, fludrocortisone, pyridostigmine, clonidine and alpha-methyldopa. [3, 19, 21]

Table 3: Widely used empirical therapeutic options in POTS

Therapy	Comments
Non-pharmacological treatment	
Patient Education • Understanding of orthostatic intolerance and POTS pathophysiology • Avoidance of immobilisation, prolonged lying down and physical deconditioning • Gradual rising from supine and sitting position, especially in the morning, after meals, and after urination/defecation • Small and frequent instead of large meals • Avoidance of prolonged standing, high ambient temperature and high humidity • Physical counter-manoeuvres (leg crossing, muscle tensing, squatting, etc.) during standing and prodromal symptoms [22, 23]	This point is crucial and should form the fundament of treatment. [3] It is rarely sufficient alone in pronounced symptoms. Patients and their families should understand the basics of orthostatic physiology and importance of non-pharmacological methods. Educational materials such as brochures, instruction films may be very helpful
Exercise Training	A regular, structured, graduated, and supervised exercise programme featuring aerobic reconditioning with some resistance training for the thighs is preferable. Initial training should avoid upright position. Mild-to moderate- intensity endurance training, progressing from semi recumbent to upright position plus strength training is recommended. Rowing machines, recumbent bicycles and swimming may be applied. [3, 24, 25]

Increased salt and fluid intake including oral water bolus if needed	A daily dietary intake of more than 10 g of sodium per day or salt tablets (e.g. 1g 3x day) and a fluid intake of at least 2.5 litre per day is recommended. [3, 26]
Compression stockings/garments	Reduction of peripheral pooling in the lower limbs and splanchnic region. Class 2 compression garments (>30 mmHg) are recommended. [3, 26, 27] They might be considered in the 'hypovolemic' subtype and low-BP phenotype. Especially, when venous pooling is observed or suspected
Pharmacological treatments	
Heart rate controlling agents	
Beta-blockers	Beta-blockers are especially recommended in 'hyper adrenergic' subtype associated with sinus tachycardia >120 bpm on standing. Beta-blockers may aggravate orthostatic intolerance in low-BP phenotype, asthma and paroxysmal chest pain. [3, 26, 28, 29]
Ivabradine	This drug is effective in low-BP phenotype or when beta-blockers are not well tolerated. It is usually seen as an alternative to beta-blockers. The evidence is based on small patient series. [30]
Verapamil	Can be tested in 'hyper adrenergic' type associated with higher BP, migraine, and chest pain. The evidence and clinical experience are very limited. [31]
Vasoactive and volume-expanding agents	
Clonidine	Generally recommended for 'hyper adrenergic' subtype and hypertensive tendency on standing. [3, 17, 32]
Midodrine	One of the few pharmacological agents positively tested in placebo-controlled studies for orthostatic hypotension. It may be effective in 'hypovolemic' subtype and low-BP phenotype with pronounced orthostatic intolerance. [3, 28, 29, 33, 34]
Droxidopa	Drug has been empirically used off label in severe POTS. Not included in the current guidelines. [28]
Pyridostigmine	May be considered in POTS phenotype associated with suspected autonomic neuropathy, gastrointestinal dysfunction and non-specific muscle weakness. Effect on BP is small. [3, 35, 36]
Fludrocortisone	Increases sodium reabsorption and enhances sensitivity of alpha adrenoreceptors. May

	worsen supine hypertension and hypokalaemia. It is recommended in 'hypovolemic' subtype and low-BP phenotype. [3, 37]
Ephedrine and pseudoephedrine	Efficacy controversial. [28]
Desmopressin	Increases water reabsorption and reduces nycturia. Sparse evidence exists. Efficacy uncertain. [38]
In-hospital acute physiological saline infusion (during consecutive 3–5 days)	In acute decompensated POTS, this method should be considered to alleviate the short-term symptoms. This is considered a Class IIA recommendation [3, 39, 40]

Reference List

1. Freeman R, Wieling W, Axelrod FB, Benditt DG, Benarroch E, Biaggioni I, Cheshire WP, Chelimsky T, Cortelli P, Gibbons CH, Goldstein DS. Consensus statement on the definition of orthostatic hypotension, neurally mediated syncope and the postural tachycardia syndrome. Clinical Autonomic Research. 2011 Apr 1;21(2):69-72.
2. Benarroch EE. Postural tachycardia syndrome: a heterogeneous and multifactorial disorder. InMayo Clinic Proceedings 2012 Dec 1 (Vol. 87, No. 12, pp. 1214-1225). Elsevier.
3. Sheldon RS, Grubb BP, Olshansky B, Shen WK, Calkins H, Brignole M, Raj SR, Krahn AD, Morillo CA, Stewart JM, Sutton R. 2015 Heart Rhythm Society expert consensus statement on the diagnosis and treatment of postural tachycardia syndrome, inappropriate sinus tachycardia, and vasovagal syncope. Heart rhythm. 2015 Jun 1;12(6):e41-63.
4. Arnold AC, Ng J, Raj SR. Postural tachycardia syndrome–diagnosis, physiology, and prognosis. Autonomic Neuroscience. 2018 Dec 1;215:3-11.
5. Low PA, Opfer-Gehrking TL, Textor SC et al. Postural tachycardia syndrome (POTS). Neurology 1995; 45: S19–25.
6. Thieben MJ, Sandroni P, Sletten DM et al. Postural orthostatic tachycardia syndrome: the Mayo clinic experience. Mayo Clin Proc 2007; 82: 308–13.
7. Sandroni P, Opfer-Gehrking TL, McPhee BR, Low PA. Postural tachycardia syndrome: clinical features and follow-up study. Mayo Clin Proc 1999; 74: 1106–10.
8. Brinth LS, Pors K, Theibel AC, Mehlsen J. Orthostatic intolerance and postural tachycardia syndrome as suspected adverse effects of vaccination against human papilloma virus. Vaccine 2015; 33: 2602–5.
9. Blitshteyn S, Brook J. Postural tachycardia syndrome (POTS) with anti-NMDA receptor antibodies after human papillomavirus vaccination. Immunol Res 2016; 65: 282–4.
10. Watari M, Nakane S, Mukaino A et al. Autoimmune postural orthostatic tachycardia syndrome. Ann Clin Transl Neurol 2018; 5: 486–92.
11. Mathias CJ, Low DA, Iodice V, Owens AP, Kirbis M, Grahame R. Postural tachycardia syndrome-current experience and concepts. Nat Rev Neurol 2012; 8: 22–34.
12. Schofield JR, Hendrickson JE. Autoimmunity, autonomic neuropathy, and the HPV vaccination: a vulnerable subpopulation. Clin Pediatr (Phila) 2018; 57: 603–6.
13. Blitshteyn S. Postural tachycardia syndrome following human papillomavirus vaccination. Eur J Neurol 2014; 21: 135–9.
14. Fedorowski A. Postural orthostatic tachycardia syndrome: clinical presentation, aetiology and management. Journal of internal medicine. 2019 Apr; 285(4):352-66.

15. Bhatia R, Kizilbash SJ, Ahrens SP, Killian JM, Kimmes SA, Knoebel EE, Muppa P, Weaver AL, Fischer PR. Outcomes of adolescent-onset postural orthostatic tachycardia syndrome. The Journal of pediatrics. 2016 Jun 1; 173:149-53.
16. Stewart JM, Boris JR, Chelimsky G, Fischer PR, Fortunato JE, Grubb BP, Heyer GL, Jarjour IT, Medow MS, Numan MT, Pianosi PT. Pediatric disorders of orthostatic intolerance. Pediatrics. 2018 Jan 1;141(1).
17. Wells R, Spurrier AJ, Linz D, Gallagher C, Mahajan R, Sanders P, Page A, Lau DH. Postural tachycardia syndrome: current perspectives. Vascular health and risk management. 2018;14:1.
18. Miller AJ, Raj SR. Pharmacotherapy for postural tachycardia syndrome. Autonomic Neuroscience. 2018 Dec 1;215:28-36.
19. Wells R, Elliott AD, Mahajan R, Page A, Iodice V, Sanders P, Lau DH. Efficacy of therapies for postural tachycardia syndrome: a systematic review and meta-analysis. In Mayo Clinic Proceedings 2018 Aug 1 (Vol. 93, No. 8, pp. 1043-1053). Elsevier.
20. Convertino VA. Blood volume response to physical activity and inactivity. The American journal of the medical sciences. 2007 Jul 1;334(1):72-9.
21. Hasan B, Almasri J, Marwa B, Klaas KM, Fischer PR. Treatment of Postural Orthostatic Tachycardia Syndrome With Medication: A Systematic Review. Journal of Child Neurology. 2020 Aug 24:0883073820948679.
22. Raj SR, Coffin ST. Medical therapy and physical maneuvers in the treatment of the vasovagal syncope and orthostatic hypotension. Progress in cardiovascular diseases. 2013 Jan 1;55(4):425-33.
23. Wieling W, Van Dijk N, Thijs RD, de Lange FJ, Krediet CP, Halliwill JR. Physical countermeasures to increase orthostatic tolerance. Journal of internal medicine. 2015 Jan;277(1):69-82.
24. George SA, Bivens TB, Howden EJ, Saleem Y, Galbreath MM, Hendrickson D, Fu Q, Levine BD. The international POTS registry: evaluating the efficacy of an exercise training intervention in a community setting. Heart Rhythm. 2016 Apr 1;13(4):943-50.
25. Winker R, Barth A, Bidmon D, Ponocny I, Weber M, Mayr O, Robertson D, Diedrich A, Maier R, Pilger A, Haber P. Endurance exercise training in orthostatic intolerance: a randomized, controlled trial. Hypertension. 2005 Mar 1;45(3):391-8.
26. Miller AJ, Raj SR. Pharmacotherapy for postural tachycardia syndrome. Autonomic Neuroscience. 2018 Dec 1;215:28-36.
27. Podoleanu C, Maggi R, Brignole M, Croci F, Incze A, Solano A, Puggioni E, Carasca E. Lower limb and abdominal compression bandages prevent progressive orthostatic hypotension in elderly persons: a randomized single-blind controlled study. Journal of the American College of Cardiology. 2006 Oct 3;48(7):1425-32.
28. Fedorowski A, Melander O. Syndromes of orthostatic intolerance: a hidden danger. Journal of internal medicine. 2013 Apr;273(4):322-35.
29. Lai CC, Fischer PR, Brands CK, Fisher JL, PORTER CB, Driscoll SW, Graner KK. Outcomes in adolescents with postural orthostatic tachycardia syndrome treated with midodrine and β-blockers. Pacing and Clinical Electrophysiology. 2009 Feb;32(2):234-8.
30. Tahir F, Arif TB, Majid Z, Ahmed J, Khalid M. Ivabradine in Postural Orthostatic Tachycardia Syndrome: A Review of the Literature. Cureus. 2020 Apr;12(4).
31. McDonald C, Koshi S, Busner L, Kavi L, Newton JL. Postural tachycardia syndrome is associated with significant symptoms and functional impairment predominantly affecting young women: a UK perspective. BMJ open. 2014 May 1;4(6).
32. Fagermoen E, Sulheim D, Winger A, Andersen AM, Gjerstad J, Godang K, Rowe PC, Saul JP, Skovlund E, Wyller VB. Effects of low-dose clonidine on cardiovascular and autonomic variables in adolescents with chronic fatigue: a randomized controlled trial. BMC pediatrics. 2015 Dec 1;15(1):117.

33. Ross AJ, Ocon AJ, Medow MS, Stewart JM. A double-blind placebo-controlled cross-over study of the vascular effects of midodrine in neuropathic compared with hyperadrenergic postural tachycardia syndrome. Clinical Science. 2014 Feb 1;126(4):289-96.
34. Izcovich A, Malla CG, Manzotti M, Catalano HN, Guyatt G. Midodrine for orthostatic hypotension and recurrent reflex syncope: a systematic review. Neurology. 2014 Sep 23;83(13):1170-7.
35. Gales BJ, Gales MA. Pyridostigmine in the treatment of orthostatic intolerance. Annals of Pharmacotherapy. 2007 Feb;41(2):314-8.
36. Kanjwal K, Karabin B, Sheikh M, Elmer L, Kanjwal Y, Saeed B, Grubb BP. Pyridostigmine in the treatment of postural orthostatic tachycardia: a single-center experience. Pacing and clinical electrophysiology. 2011 Jun;34(6):750-5.
37. Freitas J, Santos R, Azevedo E, Costa O, Carvalho M, de Freitas AF. Clinical improvement in patients with orthostatic intolerance after treatment with bisoprolol and fludrocortisone. Clinical Autonomic Research. 2000 Oct 1;10(5):293-9.
38. Coffin ST, Black BK, Biaggioni I, Paranjape SY, Orozco C, Black PW, Dupont WD, Robertson D, Raj SR. Desmopressin acutely decreases tachycardia and improves symptoms in the postural tachycardia syndrome. Heart rhythm. 2012 Sep 1;9(9):1484-90.
39. Moak JP, Leong D, Fabian R, Freedenberg V, Jarosz E, Toney C, Hanumanthaiah S, Darbari A. Intravenous hydration for management of medication-resistant orthostatic intolerance in the adolescent and young adult. Pediatric cardiology. 2016 Feb 1;37(2):278-82.
40. Jacob G, Shannon JR, Black B, Biaggioni I, Mosqueda-Garcia R, Robertson RM, Robertson D. Effects of volume loading and pressor agents in idiopathic orthostatic tachycardia. Circulation. 1997 Jul 15;96(2):575-80.

Research – Orthostatic Hypotension Causing Syncope and Hemiplegic Migraines

Brief	Diagnosis, evidenced based treatment of, and prognosis for: 1. Orthostatic Hypotension causing syncope 2. Hemiplegic Migraines
Date	October 09, 2020
Requester	Wendy s47F - pers (Assistant Director – TAB) Alicia s47F - personal privacy (Advisor – TAB)
Researcher	Craig s47F - persona (Tactical Research Advisor – TAB/AAT)

Please note:

The research and literature reviews collated by our TAB Research Team are not to be shared external to the Branch. These are for internal TAB use only and are intended to assist our advisors with their reasonable and necessary decision-making.

Delegates have access to a wide variety of comprehensive guidance material. If Delegates require further information on access or planning matters they are to call the TAPS line for advice.

The Research Team are unable to ensure that the information listed below provides an accurate & up-to-date snapshot of these matters.

Contents

Related TAB Research

RES HWB Postural Orthostatic Tachycardia Syndrome 2020/0129 (HPE: NED20/349401)

Summary

Orthostatic Hypotension Causing Syncope

- **Orthostatic hypotension** is also referred to as postural hypotension and is a sudden fall in blood pressure that occurs when the patient stands up quickly. It may be acute or chronic, as well as symptomatic or asymptomatic.
- **Syncopal** is the medical term for fainting or passing out. Syncopal episodes are typically triggered by a sudden, temporary drop in blood flow to the brain, which leads to loss of consciousness and muscle control.
- **Orthostatic Hypotension can cause Syncope** and is termed "Orthostatic Syncope".
- Diagnosis for suspected Orthostatic Hypotension begins by identifying reversible causes and underlying associated medical conditions.
- **Treatment** of Orthostatic Hypotension depends on the cause and includes medical and non-medical management.
- **Prognosis** for Orthostatic Syncope depends on the underlying cause of orthostatic hypotension. The prognosis is good in non-neurally mediated orthostatic syncope once the cause of hypotension is identified and treated.
- It appears that improving the hypotension will likely reduce occurrence of syncope.

Hemiplegic Migraines

- Hemiplegic migraine (HM) is a rare type of migraine headache affecting a small number of people who get migraine with aura. Aura includes visual symptoms, like flashes of light and zigzag patterns that happen before or during a migraine.

- Diagnosis is based on characteristic symptoms, patient history, clinical evaluation and other specialized tests. There are no pathognomonic clinical, laboratory or radiological findings to diagnose HM. Generally, affected individuals must have two episodes of migraine with aura that exhibit specific signs or symptoms.

- Treatment of HM is directed toward the specific symptoms that are apparent in each individual. A Multidisciplinary management approach is taken.

- Prognosis for HM is good, where most patients recover completely between episodes. However time may effect neurologic outcomes.

What is Orthostatic Hypotension and Syncope?

Orthostatic Hypotension (OH)

Orthostatic hypotension, also called postural hypotension, is a sudden fall in blood pressure that occurs when the patient stands up quickly. Hypotension is the term for low blood pressure.

When the patient stands up, gravity pulls blood into the legs and the blood pressure begins to fall. Certain reflexes in the body compensate for this change. The patient's heart beats faster to pump more blood and the blood vessels constrict to prevent blood from pooling in the legs.

Many drugs can affect these normal reflexes and lead to orthostatic hypotension. These reflexes may also begin to weaken as the patient ages. For this reason, orthostatic hypotension is more common in older adults.

People with orthostatic hypotension may feel dizzy when they stand up. The condition is often mild and lasts for just a few minutes after standing. Some people may faint or lose consciousness. [1]

According to a 2011 study, about 20 percent of people older than 65 experience orthostatic hypotension. [2]

Orthostatic hypotension may be acute or chronic, as well as symptomatic or asymptomatic. Common symptoms include:

- Dizziness
- Light-headedness
- blurred vision
- weakness
- fatigue
- nausea

- palpitations
- headache

Less common symptoms include:

- ***syncope***
- dyspnea
- chest pain
- neck and shoulder pain.

Causes include [2]:

- dehydration or blood loss
- disorders of the neurologic, cardiovascular, or endocrine systems
- several classes of medications

Syncope

Fainting, or passing out, is referred to medically as a syncopal episode, or syncope. Syncopal episodes are typically triggered by a sudden, temporary drop in blood flow to the brain, which leads to loss of consciousness and muscle control. The patient then falls down or over, which allows blood flow to return to the brain. Returning blood flow allows the patient to regain consciousness.

Syncope is common. It can happen at any age, including childhood, though fainting happens more frequently to people as they get older. Syncopal episodes usually last only seconds or minutes. They may be accompanied by temporary feelings of confusion when the patient regains consciousness.

Syncope can be caused by an underlying medical condition or from environmental triggers. Fainting can also result from an emotional response to a very difficult situation. Intense pain, low blood sugar, or a change in blood volume may also cause syncope. If the patient experiences a drop in blood pressure or heart rate, they may faint abruptly.

Common causes of syncope include:

- low blood pressure or dilated blood vessels
- irregular heart beat
- abrupt changes in posture, such as standing up too quickly, which can cause blood to pool in the feet or legs
- standing for long periods of time
- extreme pain or fear
- extreme stress
- pregnancy
- dehydration
- exhaustion

Syncopal episodes are often preceded by warning signs or symptoms. These may include [1]:

- nausea
- slurred speech

- weak pulse
- changes in body temperature that make you feel suddenly flushed or chilled
- sudden, clammy sweat
- pale skin
- disturbances to your vision, like seeing spots, tunnel vision, blurry vision, or dilated pupils
- feeling as if sounds are suddenly very far away
- light-headedness, wooziness, or feeling as if your head and body are weightless
- numbness
- dizziness
- vertigo, or a sensation that the room is moving
- rapid heartbeat
- body weakness
- shakiness
- headache

Orthostatic Hypotension causing Syncope (Orthostatic syncope)

Orthostatic syncope refers to syncope resulting from a postural decrease in blood pressure termed as OH. [3]

In the general population, syncope and OH are frequent events that often lead to hospitalisation or a visit to the emergency department. However, as a broad symptom, it is currently unclear whether they are independent risk markers for adverse prognosis or if syncope and OH are actually the initial symptom leading to diagnosis of severe underlying cardiac disease. [4]

OH is an important cause of syncope, and may contribute to morbidity, all-cause mortality and reduced quality of life. [5]

Prognosis for Orthostatic Syncope

Prognosis for orthostatic syncope depends on the underlying cause of orthostatic hypotension. The prognosis is good in non-neurally mediated orthostatic syncope once the cause of postural hypotension is identified and treated - fluid resuscitation in dehydration or volume depletion, transfusion for blood loss, discontinuation of offending antihypertensive medications. In neurally mediated syncope, prognosis depends on the course of the underlying medical condition. [3]

Diagnosis of Orthostatic Hypotension

Tests used to diagnose orthostatic hypotension include [6]:

- Medical history, including medical conditions and drugs taken on a regular basis
- Physical examination
- Measuring blood pressure when lying down versus standing up

- Blood tests to check, for example, blood sugars or adrenal hormones
- Echocardiography, an imaging scan of the heart, to check for certain heart conditions
- Other tests, depending on individual factors.

In a research paper focusing on the evaluation and management of OH the following diagnostic steps were indicated [2]:

- Identify reversible causes and underlying associated medical conditions
- Elicit symptoms of autonomic dysfunction involving the gastrointestinal and genitourinary systems
- Assess motor nervous system
- Measure blood pressure and pulse rate
- Test with head-up tilt-table

Evaluation of suspected OH begins by identifying reversible causes and underlying associated medical conditions.

The table below lists historical features that suggest a specific diagnosis in the patient with orthostatic hypotension. [7, 8]

Table 2. Historical Clues to Diagnosis of Orthostatic Hypotension

Historical features	*Possible etiology*
Abnormal uterine bleeding, fatigue, rectal bleeding	Anemia
Amaurosis fugax, aphasia, dysarthria, unilateral sensory and motor symptoms	Stroke
Bradykinesia, pill-rolling tremor, shuffling gait	Parkinson disease
Burns	Intravascular volume depletion
Chest pain, palpitations, shortness of breath	Congestive heart failure, myocardial infarction, myocarditis, pericarditis
Chills, fever, lethargy, nausea, vomiting	Gastroenteritis, sepsis
Extremity swelling	Congestive heart failure, venous insufficiency
High-risk sexual behavior	AIDS, neurosyphilis
Progressive motor weakness	Guillain-Barré syndrome, multiple system atrophy
Relapsing neurologic symptoms in various anatomic locations	Multiple sclerosis
Symptoms after a meal	Postprandial hypotension
Witnessed collapse	Cardiac arrhythmia, seizure

In addition to assessing for symptoms of orthostasis, the physician should elicit symptoms of autonomic dysfunction involving the gastrointestinal and genitourinary systems. [7]

Key physical examination findings in the evaluation of suspected orthostatic hypotension are listed in the table below. [9, 10]

Table 3. Physical Examination Clues to Diagnosis of Orthostatic Hypotension

Examination findings	*Possible diagnosis*	*Comments*
Aphasia, dysarthria, facial droop, hemiparesis	Stroke	—
Cardiac murmur or gallop	Congestive heart failure, myocardial infarction	—
Cogwheel rigidity, festinating gait, lack of truncal rotation while turning, masked facies	Parkinson disease	—
Confusion, dry mucous membranes, dry tongue, longitudinal tongue furrows, speech difficulty, sunken eyes, upper body weakness	Dehydration (in older patients)	Study of 55 patients 61 to 98 years of age in emergency care setting found these findings highly reliable[12]
Decreased libido, impotence in men; urinary retention and incontinence in women	Pure autonomic failure[12]	—
Dependent lower extremity edema, stasis dermatitis	Right-sided congestive heart failure, venous insufficiency	—

A detailed assessment of the motor nervous system should be performed to evaluate for signs of Parkinson disease, as well as cerebellar ataxia. [7]

Blood pressure and pulse rate should be measured in the supine position and repeated after the patient has been standing for three minutes. As many as two-thirds of patients with orthostatic hypotension may go undetected if blood pressure is not measured while supine. [11]

Head-up tilt-table testing should be ordered if there is a high index of suspicion for orthostatic hypotension despite normal orthostatic vital signs, and it may be considered in patients who are unable to stand for orthostatic vital sign measurements. [12, 13]

Treatment of Orthostatic Hypotension

Treatment of OH depends on the cause where options may include [6]:

- Fluids to treat dehydration
- Management of diabetes, such as regular insulin injections
- Changes in medication or altered doses if drugs are the cause (sometimes, however, stopping or altering the dose of a particular medication may cause more harm than good and must be carefully considered in consultation with your doctor)
- Medication, surgery or both to treat heart conditions
- Medication to increase blood volume or pressure, including corticosteroids
- Medications to treat orthostatic hypotension including pyridostigmine, a drug used in the treatment of myasthenia gravis
- A range of treatments, since orthostatic hypotension may have two or more causes
- In some cases, a lower body pressure suit is required.

The aims of treating OH should be ameliorating symptoms and improving quality of life rather than achieving target BPs, with the expectation of reducing adverse clinical outcomes, such as falls and syncope. However, there is no evidence to suggest that these outcomes are affected by the treatment of OH, particularly pharmacological treatment.

Initial management should include screening for acute precipitants, such as culprit medications and hypovolaemia. Pharmacological therapy should always be coupled with non-pharmacological measures and only after the latter has been proved to be inadequate. Patient and carer education is paramount, and it is important to pay attention to concurrent supine hypertension. [14]

Medication management

A 2017 research review [14] published by the Royal Australasian College of Physicians suggested the following medications are used in treatment:

- ***Midodrine***, an α1 agonist, stimulates arterial and venous adrenoceptors [15]
- ***Droxidopa*** is a noradrenaline prodrug with a short life of 2–3 h. It is not yet available in Australia but has been used in Japan for more than a decade and was recently approved in the United States for neurogenic OH. However, its efficacy is unclear in diabetes [15]
- ***Fludrocortisone***, a synthetic mineralocorticoid, acts by improving circulating blood volume and blood vessel sensitivity to pressor agents. Although it is suggested as the first-line treatment in some guidelines, given salt and water retention and its long half-life, it may not be suitable for patients with heart failure and nocturnal hypertension [16]
- ***Pyridostigmine***, a cholinesterase inhibitor, has the advantage of not aggravating supine hypertension. However, its pressor effect is modest, and it is less well-tolerated due to gastrointestinal side effects [17]
- ***Atomoxetine***, a noradrenaline reuptake inhibitor, has been shown to increase standing BP in a single-dose crossover trial, with improvement in BP and symptoms compared to midodrine and placebo. [18] However, its long-term effects are not known.
- ***Caffeine*** inhibits peripheral vasodilatation and thus increases standing BP [19]

Non-medical management

The Royal Australasian College of Physicians research review [14] suggested the following for non-medical management:

- Patients should be advised to stand slowly in stages to prevent sudden falls in BP.

- Bending forward when standing improves venous return by causing abdominal compression. It also improves cerebral perfusion by lowering the head to the level of the heart. [20]

- Manoeuvres that increase muscle contraction of the legs improve venous return, and examples include leg crossing, dorsiflexing the feet and squatting. [20,21]

- Compression stockings (waist-high with pressures of 40–60 mmHg at the ankle and 30–40 mmHg at the hip) have been shown to be effective in improving BP and symptoms; however, they are poorly tolerated. [22]

- Abdominal binders are a possible alternative, which recruit blood from the splanchnic reservoir. [23]

- Thromboembolic deterrent stockings are widely used, but there is only anecdotal evidence for their efficacy in mild OH. [23]

Hemiplegic Migraines

What are Hemiplegic Migraines? [24]

Hemiplegic migraine (HM) is a rare type of migraine headache. Like other migraines, hemiplegic migraine causes intense and throbbing pain, nausea, and sensitivity to light and sound. It also causes temporary weakness, numbness and tingling, and paralysis on one side of the body. These symptoms start before the headache.

HM affects a small number of people who get migraine with aura. Aura includes visual symptoms, like flashes of light and zigzag patterns that happen before or during a migraine. Aura also includes other sensory problems and trouble speaking. In people with hemiplegic migraine, the weakness or paralysis happens as part of the aura.

There are two types of HM which are based on family history of migraines:

1. ***Familial hemiplegic migraine (FHM)*** affects at least two close relatives in the same family. If a patient has FHM, each of their children will have a 50 percent chance of inheriting the condition.

2. ***Sporadic hemiplegic migraine (SHM)*** affects people who don't have any family history of the condition.

HM is caused by changes (mutations) to genes. A few genes have been linked to hemiplegic migraine, including:

- ATP1A2
- CACNA1A
- PRRT2
- SCN1A

Diagnosis

A diagnosis of hemiplegic migraine is based upon [25]:

- Identification of characteristic symptoms,
- A detailed patient history,
- A thorough clinical evaluation
- A variety of specialized tests.

There are no pathognomonic clinical, laboratory or radiological findings to diagnose HM. [26]

Two proposed diagnostic criteria have been published (The International Classification of Headache Disorders, 3rd edition [25] and a population-based study of familial hemiplegic migraine [26a] suggests revised diagnostic criteria, to help physicians diagnose hemiplegic migraine.

Generally, affected individuals must have two episodes of migraine with aura that exhibit specific signs or symptoms. By definition, fully reversible muscle weakness on one side of the body (hemiplegia) that occurs with at least one other type of aura symptom (vision, sensory, speech or brainstem) must be present for a diagnosis. For the familial form, at least one first- or second-degree relative must also have the disorder. [25]

Brain imaging is usually normal in individuals with hemiplegic migraine. A minority of individuals affected by hemiplegic migraine associated with permanent cerebellar symptoms have an atrophy of the cerebellum. [25]

Molecular genetic testing can confirm a diagnosis of familial hemiplegic migraine in some individuals. Molecular genetic testing can detect mutations in specific genes known to cause the disorder, but is available only as a diagnostic service at specialized laboratories. [25]

The table below gives the gives the diagnostic criteria according to the International Classification of Headache Disorders, third edition. [27]

Type	Diagnostic criteria ICHD-3
Hemiplegic migraine	**A.** At least two attacks fulfilling criteria **B.** One or more of the following fully reversible aura symptoms: 1. visual 2. sensory 3. speech and/or language 4. motor 5. brainstem 6. retinal **C.** At least three of the following six characteristics: 1. at least one aura symptom spreads gradually over 5minutes 2. two or more aura symptoms occur in succession 3. each individual aura symptom lasts 5–60 minutes 4. at least one aura symptom is unilateral 5. at least one aura symptom is positive 6. the aura is accompanied, or followed within 60min, by headache **D.** Aura consisting of both of the following:

Type	Diagnostic criteria ICHD-3
	1. fully reversible motor weakness 2. fully reversible visual, sensory and/or speech/ language symptoms.
Familial hemiplegic migraine (FHM)	A. Attacks fulfilling criteria for Hemiplegic migraine B. At least one first- or second-degree relative has had attacks fulfilling criteria for Hemiplegic migraine.
Familial hemiplegic migraine type 1 (FHM1)	A. Attacks fulfilling criteria for Familial hemiplegic migraine B. A mutation on the CACNA1A gene has been demonstrated
Familial hemiplegic migraine type 2 (FHM2)	A. Attacks fulfilling criteria for Familial hemiplegic migraine B. A mutation on the ATP1A2 gene has been demonstrated.
Familial hemiplegic migraine type 3 (FHM3)	A. Attacks fulfilling criteria for Familial hemiplegic migraine B. A mutation on the SCN1A gene has been demonstrated.
Familial hemiplegic migraine, other loci	A. Attacks fulfilling criteria for Familial hemiplegic migraine B. Genetic testing has demonstrated no mutation on the CACNA1A, ATP1A2 or SCN1A genes
Sporadic hemiplegic migraine (SHM)	A. Attacks fulfilling criteria for Hemiplegic migraine B. No first- or second-degree relative fulfils criteria for Hemiplegic migraine.

Treatment

The management of HM relies on the control of triggering factors and sometimes severe attacks can require hospitalisation to ensure fluid balance and food intake. Fever and seizures can be treated symptomatically. [26]

The treatment of HM is directed toward the specific symptoms that are apparent in each individual. Multidisciplinary management may require the coordinated efforts of a team of specialists [25:

- Paediatricians
- Physicians who specialize in diagnosing and treating disorders of the brain and central nervous system in children (paediatric neurologists)
- Neurologists
- Physicians who specialized in treating headaches or migraines
- Pain specialists
- Physicians who specialize in diagnosing and treating eye disorders (ophthalmologists)
- Social workers
- Psychosocial support for the entire family may be beneficial as well
- Genetic counselling may be of benefit for affected individuals and their families

Prognosis

Although most people with familial hemiplegic migraine (FHM) recover completely between episodes, neurological symptoms such as memory loss and problems with attention can last for weeks or months. The occurrence of FHM attacks tends to decrease with age. The eventual neurologic outcome is often benign; however, about 20 percent of individuals with FHM develop mild but permanent difficulty coordinating movements (ataxia), which may worsen with time, and nystagmus (rapid, involuntary eye movements). Unusually severe migraine episodes have been reported in some people with FHM. These episodes may include fever, seizures, prolonged weakness, coma, and, rarely, death. [28]

References

1 Healthline [Internet]. Healthline Media a Red Ventures; 2020. Dizziness on Standing Up (Orthostatic Hypotension). [Date cited 08 October 2020] . Available from: https://www.healthline.com/health/orthostatic-hypotension

2 Lanier JB, Mote MB, Clay EC. Evaluation and management of orthostatic hypotension. Am Fam Physician. 2011 Sep 1;84(5):527-36. PMID: 21888303.

3 StatPearls [Internet]. StatPearls; 2020. Orthostatic Syncope; 07/08/20 [Date cited 09 October 2020]. Available from https://www.statpearls.com/articlelibrary/viewarticle/26353

4 Ruwald MH. Syncope and orthostatic hypotension: early markers of cardiac disease in the general population. Heart. 2018 Mar;104(6):456-457. doi: 10.1136/heartjnl-2017-312112.

5 Mader SL. Identification and management of orthostatic hypotension in older and medically complex patients. Expert Rev Cardiovasc Ther 2012; 10: 387–95.

6 Better Health Channel [Internet]. Department of Health & Human Services, State Government of Victoria, Australia; 2020. Dizziness - orthostatic hypotension. [Date cited 08 October 2020]. Available from: https://www.betterhealth.vic.gov.au/health/ConditionsAndTreatments/dizziness-orthostatic-hypotension

7 Freeman R. Clinical practice. Neurogenic orthostatic hypotension. N Engl J Med. 2008;358(6):615-624.

8 Bradley JG, Davis KA. Orthostatic hypotension. Am Fam Physician. 2003;68(12):2393-2398

9 Gross CR, Lindquist RD, Woolley AC, Granieri R, Allard K, Webster B. Clinical indicators of dehydration severity in elderly patients. J Emerg Med. 1992;10(3):267-274.

10 Gorelick MH, Shaw KN, Murphy KO. Validity and reliability of clinical signs in the diagnosis of dehydration in children. Pediatrics. 1997;99(5):E6.

11 Carlson JE. Assessment of orthostatic blood pressure: measurement technique and clinical applications. South Med J. 1999;92(2):167-173.

12 Lahrmann H, Cortelli P, Hilz M, Mathias CJ, Struhal W, Tassinari M. EFNS guidelines on the diagnosis and management of orthostatic hypotension. Eur J Neurol. 2006;13(9):930-936.

13 Cooke J, Carew S, O'Connor M, Costelloe A, Sheehy T, Lyons D. Sitting and standing blood pressure measurements are not accurate for the diagnosis of orthostatic hypotension. QJM. 2009;102(5):335-339.

14 Chisholm P, Anpalahan M. Orthostatic hypotension: pathophysiology, assessment, treatment and the paradox of supine hypertension. Internal Medicine Journal. 2017;47(4):370-379.

15 Vijayan J, Sharma VK. Neurogenic orthostatic hypotension – management update and role of droxidopa. Ther Clin Risk Manag 2015; 8: 915–23.

16 Biaggioni I. New developments in the management of neurogenic orthostatic hypotension. Curr Cardiol Rep 2014; 16: 542.

17 Kanjwal K, Karabin B, Sheikh M, Elmer L, Kanjwal Y, Saeed B et al. Pyridostigmine in the treatment of postural orthostatic tachycardia: a single-center experience. Pacing Clin Electrophysiol 2011; 34: 750–5.

18 Ramirez CE, Okamoto LE, Arnold AC, Gamboa A, Diedrich A, Choi L et al. Efficacy of atomoxetine versus midodrine for the treatment of orthostatic hypotension in autonomic failure. Hypertension 2014; 64: 1235–40.

19 Jordan J, Shannon JR, Grogan E, Biaggioni I, Robertson D. A potent pressor response elicited by drinking water. Lancet 1999; 353: 723.

20 Wieling W, van Dijk N, Thijs RD, de Lange FJ, Krediet CT, Halliwill JR. Physical countermeasures to increase orthostatic tolerance. J Intern Med 2015; 277: 69–82. 34 Ten Harkel AD,

21 Ten Harkel AD, van Lieshout JJ, Wieling W. Effects of leg muscle pumping and tensing on orthostatic arterial pressure: a study in normal subjects and patients with autonomic failure. Clin Sci (Lond) 1994; 87: 553–8.

22 Podoleanu C, Maggi R, Brignole M, Croci F, Incze A, Solano A et al. Lower limb and abdominal compression bandages prevent progressive orthostatic hypotension in elderly persons: a randomized single-blind controlled study. J Am Coll Cardiol 2006; 48: 1425–32.

23 Smeenk HE, Koster MJ, Faaij RA, de Geer DB, Hamaker ME. Compression therapy in patients with orthostatic hypotension: a systematic review. Neth J Med 2014; 72: 80–5.

24 Healthline [Internet]. Healthline Media a Red Ventures; 2020. What Is a Hemiplegic Migraine? [Date cited 09 October 2020] . Available from: https://www.healthline.com/health/migraine/hemiplegic-migraine

25 National Organisation for Rare Disorders [Internet]. NORD - National Organization for Rare Disorders, Inc; 2020. Hemiplegic Migraine [Date cited 09 October 2020] . Available from: https://www.healthline.com/health/migraine/hemiplegic-migraine

26 Di Stefano V, Rispoli MG, Pellegrino N, et al. Diagnostic and therapeutic aspects of hemiplegic migraine. Journal of Neurology, Neurosurgery & Psychiatry 2020;91:764-771.

26a Thomsen L, Eriksen M, Roemer S, Andersen I, Olesen J, Russell M. A population-based study of familial hemiplegic migraine suggests revised diagnostic criteria. Brain. 2002;125(6):1379-1391.

27 The International Classification of Headache Disorders, 3rd edition [Internet]. Ichd-3.org. 2020 [Cited 09 October 2020]. Available from: https://ichd-3.org/wp-content/uploads/2018/01/The-International-Classification-of-Headache-Disorders-3rd-Edition-2018.pdf

28 Genetic and Rare Diseases Information Centre [Internet]. Genetic and Rare Diseases Information Center (GARD); 2020. Familial hemiplegic migraine [Date cited 09 October 2020] . Available from: https://rarediseases.info.nih.gov/diseases/10975/familial-hemiplegic-migraine

Myalgic encephalomyelitis / Chronic fatigue syndrome

The content of this document is OFFICIAL.

Please note:

The research and literature reviews collated by our TAB Research Team are not to be shared external to the Branch. These are for internal TAB use only and are intended to assist our advisors with their reasonable and necessary decision-making.

Delegates have access to a wide variety of comprehensive guidance material. If Delegates require further information on access or planning matters, they are to call the TAPS line for advice.

The Research Team are unable to ensure that the information listed below provides an accurate & up-to-date snapshot of these matters

Research questions:

What are the diagnostic features of ME-CFS?

What is the prognosis for someone diagnosed with ME-CFS? What factors affect prognosis?

What evidence-based treatment or management strategies are most effective for people with ME-CFS?

What is the prevalence of communication difficulties for people diagnosed with ME-CFS? What evidence-based treatment or management strategies are most effective for addressing communication difficulties caused by ME-CFS?

Date: 18/3/2024

Requestor: Nicole s47F - personal privacy

Endorsed by: Jane s47F - personal priv

Researcher: Aaron s47F - personal privacy

Cleared by: Stephanie s47F - personal privacy

1. Contents

2. Summary

Myalgic encephalomyelitis-Chronic fatigue syndrome (ME-CFS) is a condition characterised by excessive fatigue, especially after activity, along with a wide variety of multi-system symptoms. ME-CFS can be debilitating and result in significant functional impairment.

There are important sites of disagreement in research related to ME-CFS preventing strong recommendations about diagnosis or management. The causes and mechanisms are still unclear. There are multiple definitions of ME-CFS with overlapping but distinct diagnostic criteria. Estimates of those that recover or improve after an ME-CFS diagnosis vary widely from 4% to 83%. There is no gold standard management strategy. There are some proposed pharmacological and non-pharmacological treatments, though their efficacy is still debated. Cognitive behavioural therapy, exercise therapy and energy conservation techniques are widely recommended though evidence in support of these strategies is often of low or very low quality.

Communication difficulties are a recognised symptom of ME-CFS. Word finding problems are the most reported speech difficulty. No studies or recommendations were found that directly address problems with language or communication in ME-CFS.

3. What is ME-CFS?

3.1 Symptoms

ME-CFS may include chronic, severe and unexplained fatigue, along with other symptoms affecting bodily functions including sleep, circulation, respiration, digestion, mood, cognition, thermoregulation, and sensory processing (Steiner et al, 2023; Grach et al, 2023; NICE, 2021a-b; Deumer et al, 2021).

According to the UK's National Institute for Health and Care Excellence (NICE), core symptoms of ME-CFS include:

- Debilitating fatigue that is worsened by activity, is not caused by excessive cognitive, physical, emotional or social exertion, and is not significantly relieved by rest.
- Post-exertional malaise after activity in which the worsening of symptoms:
  - is often delayed in onset by hours or days
  - is disproportionate to the activity
  - has a prolonged recovery time that may last hours, days, weeks or longer.
- Unrefreshing sleep or sleep disturbance (or both), which may include:
  - feeling exhausted, feeling flu-like and stiff on waking
  - broken or shallow sleep, altered sleep pattern or hypersomnia.
- Cognitive difficulties (sometimes described as 'brain fog'), which may include problems finding words or numbers, difficulty in speaking, slowed responsiveness, short-term memory problems, and difficulty concentrating or multitasking (2021a, p.12).

3.2 Diagnosis

There are different sets of diagnostic criteria used in research and medical practice (Steiner et al, 2023; Grach et al, 2023; NICE, 2021a-b; Deumer et al, 2021; Bateman et al, 2021; Noor et al, 2021).

The World Health Organisation (WHO) states, "Currently there is no consensus agreement amongst medical professionals as to how chronic fatigue syndrome may be definitively diagnosed" (WHO, n.d.). This is because "Without a biomarker it is not possible to definitively know if a person has or does not have ME/CFS. Without such a reference standard (or 'gold standard') it is not possible to assess the measurement validity of the different criteria" (NICE, 2021b, p.47).

Nevertheless, different sets of diagnostic criteria may be justified on pragmatic grounds, including ability to distinguish between cases and controls or the preference for over- or under-diagnosis (NICE, 2021b).

Emerge, the Australian peak body supporting people with ME-CFS, endorses the United States' National Academy of Medicine diagnostic criteria:

> Diagnosis requires that the patient have the following three symptoms:
>
> - A substantial reduction or impairment in the ability to engage in pre-illness levels of occupational, educational, social, or personal activities that persists for more than 6 months and is accompanied by fatigue, which is often profound, is of new or definite onset (not lifelong), is not the result of ongoing excessive exertion, and is not substantially alleviated by rest
> - Post-exertional malaise
> - Unrefreshing sleep.
>
> At least one of the two following manifestations is also required:
>
> - Cognitive impairment
> - Orthostatic intolerance (Institute of Medicine, 2015, p.6)

This contrasts with the 2021 NICE clinical guideline in three ways. According to the NICE criteria:

- Diagnosis can be made when symptoms are present for a minimum of 6 weeks for adults and 4 weeks for children.
- Cognitive symptoms are necessary for diagnosis.
- Symptoms cannot be explained by another condition (NICE, 2021a).

4. Management and recovery

Researchers disagree about whether there are effective treatments for ME-CFS (Vink & Vink-Niese, 2023; NICE, 2021a). There is still ongoing debate regarding the use of cognitive behavioural therapy and graded exercise programs. Seton et al (2024) identify a number of antivirals and other pharmacological treatments that may be effective but require further investigation.

Most clinical guidelines focus on symptom management and lifestyle changes. Lifestyle interventions include strategies such as scheduling activities and rest, ensuring good sleep hygiene and appropriate diet.

Reported recovery rates for people with ME-CFS are likely low, with one study reporting 8% recovery rate in a sample of 168 people (Ghali et al, 2022). Reported improvement rates vary

widely from 4% to 83% (Lim & Torpy, 2023; Ghali et al, 2022; Moore et al, 2021). The wide variance in improvement rates may be due to different definitions of improvement and different outcome measures used (Ghal et al, 2022; Moore et al, 2021).

Table 1 Symptom management strategies (Source: Grach et al, 2023, p.1549)

Symptom	Management
Post-exertional malaise	Pacing/rest, stimulus reduction, tracking devices or diaries for symptoms
Fatigue	Pacing, low-dose naltrexone, low-dose aripiprazole, anti-inflammatory diets, supplements, vitamin deficiency treatment
Sleep issues	Melatonin, trazodone, suvorexant, doxepin/tricyclic antidepressants, gabapentin/pregabalin
Cognitive dysfunction	Journaling, memory aids, occupational therapy, low-dose naltrexone, low-dose aripiprazole, careful use of stimulants
Orthostatic intolerance	Fluids/electrolytes/compression, fludrocortisone, midodrine, propranolol, pyridostigmine, guanfacine (best guided by postural orthostatic tachycardia syndrome subtype or tilt vital signs)
Dizziness (frequent)	Consider persistent postural-perceptual dizziness diagnosis, vestibular therapy, low-dose selective serotonin reuptake inhibitor or serotonin-norepinephrine reuptake inhibitor
Muscle or joint pain	Over-the-counter medications, duloxetine, milnacipran, pregabalin, gabapentin, tricyclic antidepressants, low-dose naltrexone
Neuropathy	Pregabalin, gabapentin, tricyclic antidepressants, compression or brace therapy

Symptom	Management
Sensory amplification	Noise-cancelling headphones, tinted glasses, crowd exposure reduction, low-dose aripiprazole
Gastrointestinal symptoms	Anti-inflammatory diets, small meals, pro/synbiotics, antidiarrheals or antihistamines for diarrhea, fibre or motility agents for constipation

4.1 Pacing

Pacing is a self-management technique for energy conservation that incorporates planned periods of activity and rest. It is recommended for all people with ME-CFS. Users plan activities around periods of rest and incorporate rest breaks where possible. Gradual increases in activity are a possible but not essential element of this strategy. The aim is to reduce the symptoms of post-exertional malaise and may improve quality of life and functional independence. This strategy makes possible regular activities that are part of a healthy lifestyle (good diet, exercise, social engagement etc.) by avoiding the over-exertion/exhaustion cycle of ME-CFS. It is not intended to be a cure or rehabilitation strategy (Barakou et al, 2023; Grach et al, 2023; NICE, 2021a). While this strategy is recommended in clinical guidelines, recent reviews suggest that further research is required to address heterogeneity of study designs, inconsistent findings and poor study quality (Sanal-Hayes et al, 2023; Barakou et al, 2023).

4.2 Cognitive behavioural therapy

NICE recommends discussing CBT with patients and carers (2021a). The evidence-review that informed their recommendation found all studies reviewed provided either low or very low quality evidence for the effectiveness of CBT in ME-CFS (2021c). The authors note:

> Based on criticisms in the qualitative evidence of cognitive behavioural therapy (CBT) being described as a 'treatment' (cure) for ME/CFS, the committee considered it was important to highlight that CBT is not a cure for ME/CFS and should not be offered as such. Instead, it aims to improve wellbeing and quality of life, and may be useful in supporting people who live with ME/CFS to manage their symptoms and reduce the distress associated with having a chronic illness. It should therefore only be offered in this context, and after people have been fully informed about its principles and aims (pp.84-85).

This recommendation caused some controversy as other guidelines recommend more strongly in favour of CBT as a way to manage core symptoms of ME-CFS (Vink & Vink-Neise, 2023). Two recent reviews (Kuut et al, 2024; Bermpohl et al, 2024) argue that CBT can lead to

significant reductions in fatigue, depression, anxiety and improvements in functional impairment and physical activity.

Kuut et al (2024) performed a meta-analysis incorporating data from 8 randomised controlled trials and including 1298 participants. They found statistically significant effects on fatigue, functional impairment and physical functioning. Effects were smaller for older people and people with more severe functional impairment. The authors found no significant effects on physical functioning for people with low levels of self-efficacy. Of note, none of the studies reviewed had low risk of bias and all 8 studies were conducted by the authors' own research group.

Bermpohl et al (2024) performed a meta-analysis incorporating data from 15 randomised controlled trials and including 2015 participants. They found small to moderate effects on fatigue, depression and anxiety. Of note, the studies reviewed were rated as either high risk of bias or as having some concerns.

4.3 Exercise and physical activity

Regarding exercise programs, the NICE guidelines do not recommend graded exercise programs or unstructured exercise programs. Instead, if the patient understands and requests a personalised exercise program, the program should begin with activities below their baseline level and ensure that they can tolerate that level for a period of time. The authors state:

> The committee concluded any programme using fixed incremental increases in physical activity or exercise (for example, graded exercise therapy), or physical activity or exercise programmes that are based on deconditioning and exercise avoidance theories, should not be offered to people with ME/CFS. The committee also wanted to reinforce that there is no therapy based on physical activity or exercise that is effective as a cure for ME/CFS (2021a, p.78).

These recommendations were controversial. Some researchers argued that the recommendation against graded exercise therapy does not reflect the definitions of that approach used in the studies that NICE reviewed, and ignores some studies that show benefit of low intensity exercise for some people with ME-CFS (White et al, 2023). Wormgoor and Rodenburg (2021) found some evidence that graded exercise therapy improves fatigue as measured by participant self-report measures. However, objective measures of fitness, level of physical activity and employment showed no benefit.

5. Communication difficulties in ME-CFS

Cognitive difficulties are either common in or essential to ME-CFS, depending on the set of diagnostic criteria. Five of the nine sets of diagnostic criteria reviewed by NICE (2021b) include word finding problems as an example of cognitive symptoms (Grach et al, 2023; Lim & Torpy, 2023; NICE, 2021a-b; Maksoud et al, 2020; Institute of Medicine, 2015). Grach et al (2023) suggest that word finding and language processing problems could be a feature of post-

exertional malaise, which is a core symptom of ME-CFS on several definitions. However, the extent or severity of linguistic problems in ME-CFS is not clear. One study found around 75% of subjects experienced difficulties with words, though the authors do not elaborate on the type, frequency or severity of the difficulty (Institute of Medicine, 2015).

Evidence presented in a 2022 meta-analysis of cognitive impairments in ME-CFS shows an uneven picture of linguistic ability (Sebaiti et al; 2022). The authors found a moderate to large effect of ME-CFS on language processing speed (as measured by Colour/Word tests) and long-term verbal memory (as measured by California verbal learning test recognition, Weschler logic and reading tests). They found no significant effect on instrumental linguistic skills (as measured by the Boston Naming Test and Weschler Adult Intelligence Test), short term verbal memory (as measured by Digit Span Forward and Backward) or linguistic efficiency (as measured by National Adult Test Reading and Weschler Adult Intelligence Test).

No studies were found that address management of language or communication impairment for people with ME-CFS.

6. References

Barakou, I., Hackett, K. L., Finch, T., & Hettinga, F. J. (2023). Self-regulation of effort for a better health-related quality of life: a multidimensional activity pacing model for chronic pain and fatigue management. *Annals of medicine*, *55*(2), 2270688. https://doi.org/10.1080/07853890.2023.2270688

Bateman, L., Bested, A. C., Bonilla, H. F., Chheda, B. V., Chu, L., Curtin, J. M., Dempsey, T. T., Dimmock, M. E., Dowell, T. G., Felsenstein, D., Kaufman, D. L., Klimas, N. G., Komaroff, A. L., Lapp, C. W., Levine, S. M., Montoya, J. G., Natelson, B. H., Peterson, D. L., Podell, R. N., Rey, I. R., … Yellman, B. P. (2021). Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Essentials of Diagnosis and Management. *Mayo Clinic proceedings*, *96*(11), 2861–2878. https://doi.org/10.1016/j.mayocp.2021.07.004

Bermpohl, F. M. G., Kucharczyk-Bodenburg, A. C., & Martin, A. (2024). Efficacy and Acceptance of Cognitive Behavioral Therapy in Adults with Chronic Fatigue Syndrome: A Meta-analysis. International journal of behavioral medicine, 10.1007/s12529-023-10254-2. Advance online publication. https://doi.org/10.1007/s12529-023-10254-2

Deumer, U. S., Varesi, A., Floris, V., Savioli, G., Mantovani, E., López-Carrasco, P., Rosati, G. M., Prasad, S., & Ricevuti, G. (2021). Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS): An Overview. *Journal of clinical medicine*, *10*(20), 4786. https://doi.org/10.3390/jcm10204786

Ghali, A., Lacout, C., Fortrat, J. O., Depres, K., Ghali, M., & Lavigne, C. (2022). Factors Influencing the Prognosis of Patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. *Diagnostics (Basel, Switzerland)*, *12*(10), 2540. https://doi.org/10.3390/diagnostics12102540

Grach, S. L., Seltzer, J., Chon, T. Y., & Ganesh, R. (2023). Diagnosis and Management of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. *Mayo Clinic proceedings*, *98*(10), 1544–1551. https://doi.org/10.1016/j.mayocp.2023.07.032

Institute of Medicine. (2015). *Beyond Myalgic Encephalomyelitis/Chronic Fatigue Syndrome: Redefining an Illness*. Washington, DC: The National Academies Press. https://doi.org/10.17226/19012.

Kuut, T. A., Buffart, L. M., Braamse, A. M. J., Csorba, I., Bleijenberg, G., Nieuwkerk, P., Moss-Morris, R., Müller, F., & Knoop, H. (2024). Does the effect of cognitive behavior therapy for chronic fatigue syndrome (ME/CFS) vary by patient characteristics? A systematic review and individual patient data meta-analysis. *Psychological medicine*, *54*(3), 447–456. https://doi.org/10.1017/S0033291723003148

Lim, W. T., & Torpy, D. J. (2023). Chronic Fatigue Syndrome. In K. R. Feingold (Eds.) et. al., *Endotext*. MDText.com, Inc.

Maksoud, R., du Preez, S., Eaton-Fitch, N., Thapaliya, K., Barnden, L., Cabanas, H., Staines, D., & Marshall-Gradisnik, S. (2020). A systematic review of neurological impairments in myalgic encephalomyelitis/ chronic fatigue syndrome using neuroimaging techniques. *PloS one*, *15*(4), e0232475. https://doi.org/10.1371/journal.pone.0232475

Moore, Y., Serafimova, T., Anderson, N., King, H., Richards, A., Brigden, A., Sinai, P., Higgins, J., Ascough, C., Clery, P., & Crawley, E. M. (2021). Recovery from chronic fatigue syndrome: a systematic review-heterogeneity of definition limits study comparison. *Archives of disease in childhood*, *106*(11), 1087–1094. https://doi.org/10.1136/archdischild-2020-320196

National Institute for Health and Care Excellence. (2021a). Myalgic encephalomyelitis (or encephalopathy)/chronic fatigue syndrome: diagnosis and management [NG206]. https://www.nice.org.uk/guidance/ng206

National Institute for Health and Care Excellence. (2021b). Myalgic encephalomyelitis (or encephalopathy)/chronic fatigue syndrome: diagnosis and management: Identifying and diagnosing ME/CFS [Evidence review]. https://www.nice.org.uk/guidance/ng206/evidence/d-identifying-and-diagnosing-mecfs-pdf-9265183025

National Institute for Health and Care Excellence. (2021c). Myalgic encephalomyelitis (or encephalopathy)/chronic fatigue syndrome: diagnosis and management: Non-pharmacological management of CFS [Evidence review]. https://www.nice.org.uk/guidance/ng206/evidence/d-identifying-and-diagnosing-mecfs-pdf-9265183025

Sanal-Hayes, N. E. M., Mclaughlin, M., Hayes, L. D., Mair, J. L., Ormerod, J., Carless, D., Hilliard, N., Meach, R., Ingram, J., & Sculthorpe, N. F. (2023). A scoping review of 'Pacing' for management of Myalgic Encephalomyelitis/Chronic Fatigue Syndrome

(ME/CFS): lessons learned for the long COVID pandemic. *Journal of translational medicine*, *21*(1), 720. https://doi.org/10.1186/s12967-023-04587-5

Sebaiti, M. A., Hainselin, M., Gounden, Y., Sirbu, C. A., Sekulic, S., Lorusso, L., Nacul, L., & Authier, F. J. (2022). Systematic review and meta-analysis of cognitive impairment in myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS). *Scientific reports*, *12*(1), 2157. https://doi.org/10.1038/s41598-021-04764-w

Seton, K. A., Espejo-Oltra, J. A., Giménez-Orenga, K., Haagmans, R., Ramadan, D. J., Mehlsen, J., & European ME Research Group for Early Career Researchers (Young EMERG) (2024). Advancing Research and Treatment: An Overview of Clinical Trials in Myalgic Encephalomyelitis/Chronic Fatigue Syndrome (ME/CFS) and Future Perspectives. *Journal of clinical medicine*, *13*(2), 325. https://doi.org/10.3390/jcm13020325

Steiner, S., Fehrer, A., Hoheisel, F., Schoening, S., Aschenbrenner, A., Babel, N., Bellmann-Strobl, J., Finke, C., Fluge, Ø., Froehlich, L., Goebel, A., Grande, B., Haas, J. P., Hohberger, B., Jason, L. A., Komaroff, A. L., Lacerda, E., Liebl, M., Maier, A., Mella, O., … Scheibenbogen, C. (2023). Understanding, diagnosing, and treating Myalgic encephalomyelitis/chronic fatigue syndrome - State of the art: Report of the 2nd international meeting at the Charité Fatigue Center. *Autoimmunity reviews*, *22*(11), 103452. https://doi.org/10.1016/j.autrev.2023.103452

Vink, M., & Vink-Niese, A. (2023). The Draft Report by the Institute for Quality and Efficiency in Healthcare Does Not Provide Any Evidence That Graded Exercise Therapy and Cognitive Behavioral Therapy Are Safe and Effective Treatments for Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. *Diseases (Basel, Switzerland)*, *11*(1), 11. https://doi.org/10.3390/diseases11010011

White, P., Abbey, S., Angus, B., Ball, H. A., Buchwald, D. S., Burness, C., Carson, A. J., Chalder, T., Clauw, D. J., Coebergh, J., David, A. S., Dworetzky, B. A., Edwards, M. J., Espay, A. J., Etherington, J., Fink, P., Flottorp, S., Garcin, B., Garner, P., Glasziou, P., … Zeman, A. (2023). Anomalies in the review process and interpretation of the evidence in the NICE guideline for chronic fatigue syndrome and myalgic encephalomyelitis. *Journal of neurology, neurosurgery, and psychiatry*, *94*(12), 1056–1063. https://doi.org/10.1136/jnnp-2022-330463

World Health Organization. (n.d.). *Chronic fatigue syndrome*. https://www.who.int/standards/classifications/frequently-asked-questions/chronic-fatigue-syndrome

Wormgoor, M. E. A., & Rodenburg, S. C. (2021). The evidence base for physiotherapy in myalgic encephalomyelitis/chronic fatigue syndrome when considering post-exertional malaise: a systematic review and narrative synthesis. *Journal of translational medicine*, *19*(1), 1. https://doi.org/10.1186/s12967-020-02683-4

Thermoregulation and air conditioning

The content of this document is OFFICIAL.

Please note:

The research and literature reviews collated by our TAB Research Team are not to be shared external to the Branch. These are for internal TAB use only and are intended to assist our advisors with their reasonable and necessary decision-making.

Delegates have access to a wide variety of comprehensive guidance material. If Delegates require further information on access or planning matters, they are to call the TAPS line for advice.

The Research Team are unable to ensure that the information listed below provides an accurate & up-to-date snapshot of these matters

Research question:

What medical conditions or disabilities involve an impairment in thermoregulation?

What cooling systems are available for use in Australia?

Is air conditioning effective in managing symptoms of thermoregulation impairment compared to other cooling systems?

Date: 8/2/2024

Requestor: Helen s47F - personal privacy

Endorsed by: Melinda s47F - personal privacy

Researcher: Aaron s47F - personal privacy

Cleared by: Stephanie s47F - personal privacy

1. Contents

2. Summary

Note: This paper is a substantial revision of a research paper originally completed in October 2019 and reviewed in February 2024.

Thermoregulation impairment can result from a wide range of health conditions and disabilities. The human thermoregulatory system involves perceptual, physiological and behavioural components. A condition may result in a thermoregulatory impairment if it affects the peripheral or central nervous systems, or if the condition impacts strength, mobility, motor control, cognition or emotional regulation.

The main types of cooling systems found in Australian homes are fans, evaporative and refrigerative air conditioners. Refrigerative air conditions, including reverse cycle air conditioners, are the most common type of air conditioner used in Australia. The cost-effectiveness of cooling systems depends on several factors including climate, location, energy prices, architectural features of the home, device running time, temperature set-point and other lifestyle factors.

There is evidence for the benefits of air conditioner use in the general population to manage the effects of heat, especially in very hot and dry climates. However, there is very little evidence comparing air conditioning with other cooling devices or strategies and very little experimental evidence showing the circumstances in which air conditioning might contribute to managing the symptoms of thermoregulation impairment.

Despite this, public health messaging and recommendations from researchers and clinicians are consistent. They suggest that simple behavioural strategies and easily accessible cooling devices have a role in managing the symptoms of thermoregulation impairment. Behavioural strategies include:

- understanding personal heat tolerance and preferences
- staying inside during the hotter times of day
- planning outdoor or strenuous activities for cooler times of day
- wearing loose or light clothing
- wearing wet clothes or wraps
- taking regular breaks from activity
- consuming cold foods and drinks
- taking cold baths or showers.

Recommended equipment or devices include:

- space coolers (including evaporative coolers and air conditioning)
- electric fans
- cooling garments.

3. Human thermoregulation

Humans are homeothermic animals, which means that human body temperature is maintained at a nearly constant level largely, but not entirely, independent of the environment. Core human body temperature is maintained at around 37°C (+/- 0.5°C), while peripheral body temperature may vary more widely (Romanovsky, 2018; Cheshire, 2016).

When the core body temperature is too low, this is called hypothermia. When the core body temperature is too high, this is called hyperthermia. Some sources refer to hypo and hyperthermia as any variation outside the normal range of core body temperature. (Romanovsky, 2018). Other sources define states more specifically as below 35°C for hypothermia and above 40°C for hyperthermia (Cheshire, 2016).

Slight changes outside the accepted range can be controlled with physiological or behavioural responses. Extreme changes to core body temperature may lead to significant injury or death (Osila et al, 2023; Cheshire, 2016). Age can affect the ability to regulate body temperature due to both physiological changes (such as changes in metabolism or the cardiovascular system) and behavioural changes (spending more time at home, reduced activity), which is why older people are more susceptible to complications from environmental extremes (Osila et al, 2023; Bennetts et al, 2020).

Thermoregulation is the process of maintaining body temperature by balancing heat generation and heat loss. Temperature variations are picked up by thermoreceptors on the skin or inside the body. These receptors alert the thermoregulatory centre located in the hypothalamus to enact thermoeffectors, physiological or behavioural responses that regulate body temperature.

3.1 Thermoeffectors

Physiological thermoeffectors are involuntary body processes that help to control heat loss or heat generation. They include:

- skin vasodilation or vasoconstriction
- sweating
- shivering
- piloerection
- panting.

Behavioural thermoeffectors are voluntary or instinctual complex behaviours. They include behaviours such as changing posture, drinking water, adding or removing clothing, turning on a fan or air conditioning etc (Osila et al, 2023; Romanovsky, 2018).

Thermoeffectors aid in heat loss, conservation or generation by affecting one or more of the four processes of heat exchange: conduction, convection, radiation, and evaporation (Osila et al, 2023; Romanovsky, 2018; Cheshire, 2016).

Conduction

Conduction occurs when heat is transferred from one object to another object in direct contact. Materials with high conductivity are more able to draw heat away from the body. For example, water has a high conductivity and so submersion in water is a good way to draw heat from the body (Osila et al, 2023; Romanovsky, 2018).

Convection

Convection occurs when a body is submerged in a gas or liquid. Movement of the fluid replaces layers of fluid closer to the body with fluid further from the body. The layers of fluid closer to the body have a temperature closer to the temperature of the skin, while the more distant fluid has a temperature closer to the ambient temperature. Convection therefore intensifies conduction. If the environment is hotter, the body is exposed to hotter material and so heats up faster. If the environment is colder, the body is exposed to colder material and so cools down faster. For example, a ceiling fan cools by convection by increasing movement of air on the skin, removing warmer air closer to the body and replacing it with cooler air further from body (Osila et al, 2023; Romanovsky, 2018).

Radiation

All materials emit and absorb heat via radiation in the form of electromagnetic waves. The human body loses approximately 60% of its heat via radiation. Unlike conduction or convection, radiation does not require contact with a medium. For example, solar radiation can warm the earth despite passing through colder layers of earth's atmosphere (Osila et al, 2023; Connor, 2022; Romanovsky, 2018; Cheshire, 2016).

Evaporation

Liquid requires energy in the form of heat to evaporate. The heat required is drawn from the environment or from the liquid itself and transferred from the liquid to the gas. For example, animals make use of evaporative cooling in the form of sweating and panting (Osila et al, 2023; Romanovsky, 2018; Lohner, 2017). Evaporation accounts for about 22-30% of heat lost from the body (Osila et al, 2023; Cheshire, 2016). Evaporation is the most efficient form of heat loss in the human body, though it can be less effective in more humid environments and does consume large amounts of water. Evaporation is the only form of heat transfer that also works when the ambient temperature is higher than the temperature of the skin (Romanovsky, 2018).

4. Conditions resulting in thermoregulation impairment

Some conditions can impair our thermoregulatory processes and therefore increase the risk of temperature related health problems. The sections below describe some, though not all, conditions for which there is evidence of thermoregulatory impairment. For most conditions, whether thermoregulation impairment occurs, or whether the impairment is substantial and results in activity limitations or participation restrictions, will vary for individuals.

Conditions that affect the nervous system or skin (including brain and spinal cord injuries, severe burns, neuropathies, and neurodegenerative conditions) can impair physiological and behavioural thermoeffectors (Osila et al, 2023; Cheshire, 2016). Even when physiological thermoregulation processes are unaffected, some conditions can impair behavioural thermoeffectors, interrupting a person's capacity to voluntarily regulate their body temperature. For example, any condition that affects mobility may also reduce capacity for heat generation due to reduced or infrequent muscle contractions. Any condition that impairs judgement may also reduce a person's capacity to respond appropriately to changes in temperature (Cheshire, 2016). Refer to Table 1 for an incomplete list of conditions that may lead to or increase the risk of temperature related illness.

Conditions that result in thermoregulation impairment can significantly impact functional capacity and quality of life, though this is not always the case. These conditions may or may not result in activity limitations or participation restrictions in activities of daily living, social or economic participation. For example, there is evidence that most people with peripheral neuropathy experience anhidrosis or some level of impairment in their ability to sweat. However, only a quarter of those with this impairment will experience higher core body temperatures compared to the general population (Fealey, 2018). Therefore, the impairment to a thermoregulatory process (reduced ability to sweat) may not ultimately increase the risk of heat related illness or reduce the person's capacity to participate in any activity.

Table 1 Conditions that may contribute to thermoregulation impairment (Source: Cheshire, 2016)

Type	Condition
Conditions that may impair judgement	Dementia, head injury, schizophrenia, hepatic encephalopathy
Conditions that may impair mobility	Musculoskeletal injury, stroke, spinal cord injury, Parkinson's disease, multiple system atrophy, myopathy, severe peripheral neuropathy
Conditions that may impair thermal sensation	Peripheral neuropathy, severe burns
Conditions that may impair thermoregulatory responses	Wernicke encephalopathy, stroke, spinal cord injury, Guillain–Barré syndrome, amyotrophic lateral sclerosis, multiple sclerosis, myopathy
Conditions that may cause anhidrosis	Cholinergic neuropathy, autoimmune autonomic ganglionopathy, chronic idiopathic anhidrosis, botulism, generalized small fiber neuropathy,

	Sjögren syndrome, multiple system atrophy, Fabry's disease, bilateral cervical sympathectomy
Conditions that may increase thermogenesis	Status epilepticus, neuroleptic malignant syndrome, malignant hyperthermia
Other conditions that may lead to thermoregulatory impairment	Hypoglycemia, Diabetic ketoacidosis, Hypothyroidism, Adrenal failure, Hypopituitarism, Renal failure, Shock, Sepsis, Anorexia nervosa, Thyrotoxicosis, Pheochromocytoma

4.1 Spinal cord injury

There is evidence of impaired thermoregulation in people with spinal cord injury, mostly likely due to a combination of reduced activity of thermoreceptors to detect changes in temperature, reduced muscle mass and impairment in thermoeffectors such as sweating, vasoconstriction and vasodilation (Osila et al, 2023; Grossman et al, 2021; Zhang, 2019; Price & Trbovich, 2018; Cheshire, 2016; Girard, 2015). People with higher level of lesion show greater thermoregulatory impairment (Osila et al, 2023; Grossman et al, 2021). There is evidence that people with spinal cord injury below the level of T6 can regulate body temperature as effectively as people without spinal cord injury (Grossman et al, 2021; Price & Trbovich, 2018). There is some evidence that thermoregulation impairment in people with spinal cord injuries above T6 may also lead to activity limitations. For example, high or low temperatures may prevent people with tetraplegia from participating in activities outside the home (Price & Trbovich, 2018).

4.2 Acquired brain injury

Thermoregulatory impairment after brain injury (traumatic brain injury or stroke) may involve injury to the hypothalamus, changes in blood flow, vascular control and metabolism, and difficulties with mobility or judgement (Gowda et al, 2018; Cheshire, 2016; Thompson et al, 2003). There is evidence that around 70% of people experience hyperthermia during the acute phase after traumatic brain injury. This may be due to the nature of the injury, post-traumatic inflammation or post-injury infection (Thompson et al, 2003). Hyperthermia is a risk factor for secondary injury. This includes rebound hyperthermia, which is a possible consequence of rewarming after induced hypothermia (Gowda et al, 2018; Childs & Lunn, 2013). Clinicians regularly induce hypothermia soon after the initial brain injury to prevent secondary brain injury and improve other outcomes. Thermoregulatory impairment may be more common in some people with brain injury, though affected sub-groups have not been identified (Gowda et al, 2018).

4.3 Parkinson's Disease

Thermoregulation difficulties are common in people with Parkinson's disease and may lead to difficulties with sweating, sleep, and altered perception of heat and cold (Pfeiffer, 2020; Coon & Low, 2018; Zhong et al, 2013). The presence of peripheral neuropathy in people with Parkinson's disease can result in impairments to thermoeffectors such as vasoconstriction/dilation, sweating and piloerection (Coon & Low, 2018). Around 30-70% of people with Parkinson's experience problems with sweating, including hyperhidrosis (increased sweating) and hypohidrosis (reduced sweating). This may be related to neurological changes or to medications used to treat the core symptoms of Parkinson's disease. Hypohidrosis can increase risk of overheating, while hyperhidrosis can be uncomfortable and lead to sleep difficulties (Pfeiffer, 2020; Jost, 2017). Thermoregulation impairment can affect well-being and quality of life for people with Parkinson's disease:

> Patients are often bothered by heat intolerance which may influence activity levels and social endeavors. Needing to frequently change clothing or bedding due to excessive sweating episodes is also problematic for patients and their caregivers, particularly when motor function is compromised. Temperature intolerance or night sweats may impair a patient's sleep, which is often affected due to motor dysfunction or concomitant sleep disorders. Social function is also affected by sweating episodes, leaving some patients to feel embarrassed and contributing to social isolation (Coon & Law, 2018, p.271).

4.4 Multiple Sclerosis

Thermoregulation impairment is more researched in multiple sclerosis than for any other condition. Around 60-80% of people with multiple sclerosis experience temperature sensitivity. Thermoregulatory difficulties in people with multiple sclerosis, especially susceptibility to hyperthermia, may be due to impaired sweating function, decreased sensitivity of thermoreceptors or hypothalamic dysfunction. Hyperthermia is a significant risk as it can exacerbate symptoms including muscle weakness, spasticity, fatigue, blurred vision and pain, as well as worsening existing difficulties with balance, processing speed, concentration, and attention (Osila et al, 2023; Christogianni et al, 2022; Razi et al, 2022; Davis et al, 2018; Christogianni et al, 2018; Allen et al, 2017). Hyperthermia may be induced by environmental increases in temperature, hot baths or exercise (Razi et al, 2022; Christogianni et al, 2022; Davis et al, 2018; Christogianni et al, 2018). However, there is evidence that regular exercise for people with multiple sclerosis can improve symptoms and quality of life. Therefore, heat management strategies should be in place when clinicians recommend an exercise program for people with multiple sclerosis (Huang et al, 2015). Cold temperatures can also lead to a worsening of symptoms, though this is less common and less studied (Christogianni et al, 2018).

4.5 Peripheral neuropathy

Peripheral neuropathy is a general term for conditions that cause damage to the nerves of the peripheral nervous system. Damage can occur to large-diametre or small-diametre nerve fibres. Large fibres mediate motor and sensory functions, while small fibres mediate autonomic functions, pain and temperature (Novello & Pobre, 2023; Castelli et al, 2020).

Conditions that can result in peripheral neuropathy include Guillaine-Barre syndrome, diabetes mellitus, Fabry disease, Parkinson's disease, Ehlers Danlos syndrome, postural orthostatic tachycardia syndrome (POTS) and Sjögren syndrome. Diabetes related peripheral neuropathy is the most prevalent form of the peripheral neuropathy in developed countries (Osila et al, 2023; Fealey, 2018; Cheshire, 2016).

There is evidence that most people with some form of peripheral neuropathy experience abnormalities in core body temperature. Common thermoregulatory concerns for people with peripheral neuropathy include impairments to physiological thermoeffectors such as vasoconstriction/dilation, sweating, piloerection and shivering (Fealey, 2018; Cheshire, 2016). As peripheral neuropathy is associated with reduced sensitivity of thermoreceptors, there is also reason to believe the condition may lead to disruption of behavioural thermoeffectors (Fealey, 2018).

Heat intolerance is a possible symptom of POTS. High ambient temperatures may also exacerbate core symptom of orthostatic intolerance. (Fedorowski, 2018; Landero, 2014; Goodkin & Bellew, 2014). These symptoms may be associated with the presence of small fibre neuropathy. In a study of 276 participants with POTS, Angeli et al (2024) found 35% showed altered sweat patterns, which characterised the neuropathic phenotype. A small study of 30 people with POTS found significant differences in thermal perception and pain threshold (Billig et al, 2020). POTS is also a common co-occurring condition in Ehlers Danlos syndrome, which itself can present with thermoregulatory difficulties (Colman et al, 2023; Thwaites et al, 2022; Hakim et al, 2017).

4.6 Psychosocial conditions

While there is preliminary evidence that some people with anxiety disorders show abnormalities in physiological thermoeffectors such as vasodilation and sweating (Fischer et al, 2021), psychosocial conditions may coincide with thermoregulatory impairments in the form of altered sensation or disrupted behavioural thermoeffectors (due to altered cognition, judgement or executive control). RES 319 Weather and Bipolar Disorder contains some discussion of the effects of temperature on outcomes for people with bipolar and other psychosocial conditions.

4.7 Epilepsy and seizure disorders

Temperature may affect epilepsy and seizure activity differently, depending on the individual, the type of epilepsy or type of seizure.

Hyperthermia is both a possible trigger and a possible consequence of seizure. It may be a consequence of seizure due to excessive muscle activity or activation of the autonomic system (Pollandt & Bleck, 2018; Cheshire, 2016). Hyperthermia can also cause seizures, as in the case of febrile seizures experienced mainly by children during episodes of fever. In Dravet syndrome, seizures can follow even small temperature increases caused by higher ambient temperatures, fever, cold-warm shifts, warm baths or exercise (Gulcebi et al, 2021; Pollandt & Bleck, 2018).

However, colder temperatures may also increase risk of seizure in epilepsy. Hospital admission studies in Taiwan, Germany and Korea found that seizure risk increases in colder temperatures (Chang et al, 2019; Kim et al, 2017; Rakers et al, 2017). However, these studies take place in climates that tend to have mild summers and may not generalise to Australia. For example, Rakers et al (2017) found that ambient temperatures higher than 20°C decrease the risk of seizure, though the highest recorded temperature in the study was 28°C.

Epilepsy Action Australia (n.d.) states:

> Whilst research related to weather and seizures has been limited, and based in the northern hemisphere, there is no scientific evidence that hot weather itself causes seizures to occur in people with epilepsy. In Australia it appears most people report that the heat, or becoming overheated, tends to increase the likelihood of seizures. Becoming severely overheated can cause seizures, but an average hot day is not in itself the culprit.
>
> Obviously, heat can be a major contributor to dehydration. If someone is exposed to heat for a long period of time and does not drink enough fluid, this can cause dehydration which can increase the risk of a seizure in someone with epilepsy, sometimes later in the day. When fluid loss from the body (mostly perspiration) is greater than fluid intake, it causes a change in electrolytes – a drop in sodium (salt) and glucose (sugar) levels in the body. Ultimately, this can lead to low blood sugar levels (hypoglycemia) which can also trigger seizures for some people.

4.8 Autism

People with autism may experience sensory differences such as hypo or hypersensitivity to heat or cold (Raising Children Network, 2024; Zaniboni et al, 2023; Hidaka et al, 2023). Based on their review, Zaniboni et al suggest the following sensory differences with respect to perception of heat and cold:

- Different tactile sensitivity, as well as higher variability in warm and cold detection: paradoxical heat sensation (the perception of heat when it should not be perceived, hyper-sensitivity), lower thresholds in heat and cold detection (hypo-sensitivity).
- Thermal processing might be related with environment adoption or self-injury.
- Difficulties with interoception (heart-rate and body-temperature perception) and self-regulation and identification of emotions.
- Differences in hypothalamus development (related to homeostasic regulation, including metabolic rate, temperature and emotion). This can also lead to depression, anxiety, sleep disorders and obesity (2023, p.10).

4.9 Motor neurone disease / Amyotrophic lateral sclerosis

There is a lack of evidence regarding thermoregulatory impairments in motor neurone diseases such as amyotrophic lateral sclerosis (ALS). It is likely that behavioural thermoeffectors are impaired in ALS considering symptoms related to mobility and cognitive functions. There is minimal evidence that people with ALS experience altered heat sensation and that hypothalamus volume may be reduced. Physiological thermoeffectors such as shivering may be affected by progressive impairment in skeletal muscles (Dupuis et al, 2018). Much of the evidence for involvement of thermoregulatory systems in ALS comes from studies of animal models (Rodríguez-Sánchez et al, 2022; Braun et al, 2019). In their review of the subject, Dupuis et al state:

> In our clinical experience, we observed that ALS patients often complain of feeling hot, or conversely of being unable to warm up, and some patients develop low body temperature. Also, some patients report a worsening of symptoms in cold weather. However, these symptoms are generally not considered as being part of the core clinical picture, mostly because they are attributed to muscle atrophy and/or nerve degeneration. Therefore, potential thermoregulatory defects to the best of our knowledge have never been systematically studied in ALS patients (2018, p.750).

Since then, at least one study has shown a high rate of hypothermia in people with ALS who have had tracheostomy or invasive ventilation for longer than five years (Nakayama et al, 2018).

4.10 Huntington's disease

Thermoregulation problems are sometimes reported by people with Huntington's disease:

> some clinicians do occasionally report anecdotally that some of their [Huntington's disease] patients seem to have a striking indifference to cold and that they will dress too lightly for the weather, while others will sweat so profusely that they resort to wearing cooling vests (Weydt et al, 2018, p.766).

The first case study of a person with Huntington's disease presenting with hypothermia was submitted in 2020 (Altiner et al, 2020). Most of the evidence of thermoregulation impairment in

Huntington's disease comes from animal models. These studies have shown evidence of hypothermia, weight loss, involuntary movements, as well as differences in circadian rhythms, brown adipose tissue, skeletal muscle and the hypothalamus. This suggests a possible effect of Huntington's disease on heat retention, shivering and non-shivering thermogenesis. Development of psychiatric conditions and problems with mobility and cognitive function may also contribute to disruption of behavioural thermoeffectors. There are few studies directly investigating thermoregulation associated with Huntington's disease in humans (Altiner et al, 2020; Weydt et al, 2018).

4.11 Severe burns

The skin plays an important role in thermoregulatory processes including heat retention, sensation, sweating, piloerection, vasodilation and vasoconstriction. When large parts of the skin are lost or damaged, this enables increased heat loss and contributes to difficulties sensing changes in temperature, thereby increasing the risk of hypothermia. People with severe burns are also at risk of hypermetabolism, which can lead to hyperthermia, excessive sweating, weight loss, muscle wasting and other symptoms (Radzikowska-Büchner et al, 2023; Mertin et al, 2022). In cases of severe burn injury, metabolic changes can last up to three years after the initial injury and function of damaged skin may not return (Radzikowska-Büchner et al, 2023; Jeshke et al, 2011).

5. Management of thermoregulation impairment

Researchers and clinicians have recommended behavioural strategies to manage thermoregulation impairment in people with multiple sclerosis (Christogianni et al, 2022; Davis et al, 2018), autism (Zaniboni et al, 2023), and spinal cord injury (Girard, 2015). Behavioural strategies can include moving to a cooler area, planning activities for cooler times of the day, taking regular breaks from strenuous activity, choosing weather appropriate clothing, or gradual acclimatisation in warmer or colder temperatures (Healthdirect, 2024; Zaniboni et al, 2023; Grossman et al, 2021; Davis et al, 2018; Girard, 2015; Australian Red Cross, n.d.).

Standard first line treatment for hyperthermia includes cooling strategies that are usually low cost or readily accessible: air conditioning, misting fans, cold bath or shower, drinking cold water and applying cold packs or ice packs (Healthdirect, 2024; Grossman et al, 2021; Christogianni et al, 2022; Davis et al, 2018; Gowda et al, 2018; Hopkins et al, 2018; Zawardska et al, 2017; Cheshire, 2016; Australian Red Cross, n.d.). These non-invasive methods are less easy to control than invasive cooling strategies such as intravenous injection of cooling substances. Where non-invasive strategies succeed in lowering body temperature, they are not easily able to maintain a stable target temperature and therefore require monitoring and adjustment (Gowda et al, 2018).

There is evidence of effectiveness of non-invasive cooling strategies to improve exercise performance and lower the risk of heat related effects of exercise in the general population (Heydenreich et al, 2023; Douzi et al, 2019). There is mixed evidence for the effectiveness of

non-invasive strategies in people with thermoregulatory impairment. The inconsistency in the evidence may be due to the frequency of small, low powered studies and the heterogeneity of climatic conditions and outcome measures (Grossman et al, 2021).

In a review of cooling strategies for people with spinal cord injury, Grossman et al (2021) found inconsistent evidence for the temperature reducing effects of cooling garments, cold drinks and misting fans. Some studies show cooling garments reduce skin temperature but not core body temperature, whereas a consistent effect across several studies showed pre-cooling using cooling garments or other methods could improve endurance during exercise and lower rate of increase of body temperature (Grossman et al, 2021; Davis et al, 2018).

A 2023 systematic review into the use of cooling garments for people with Multiple Sclerosis found that cooling garments are effective in reducing body temperature and improving walking capacity and functional mobility (Stevens et al, 2023). The authors found no significant differences between types of cooling garment. Active treatment groups were compared with either other cooling garments, sham active controls or passive controls. No study was reviewed that compared cooling garments with other cooling strategies such as air conditioning.

5.1 Air conditioning compared to other cooling strategies

Researchers and clinicians have recommended reducing the ambient temperature of the environment with space cooling strategies/devices as a way of managing thermoregulation impairment in people with multiple sclerosis (Christogianni et al, 2022; Davis et al, 2018), autism (Zaniboni et al, 2023), spinal cord injury (Price & Trbovich, 2018), epilepsy (Epilepsy Action Australia, n.d.), and severe burns (Radzikowska-Büchner et al, 2023).

Existing evidence indicates that air conditioning has a role in managing thermoregulation impairment. Hospital studies show air conditioning can improve or maintain patients' thermal comfort, recovery rates and well-being, and reduce infections and length of hospital stays. However, more research is required to determine the optimum ambient temperature to maximise patient outcomes (Lenzer et al, 2020; Shajahan et al, 2019). In the case of severe burns, raising the ambient temperature of the room to 24°C – 38°C may prevent or reduce the risk of a hypermetabolic reaction (Radzikowska-Büchner et al, 2023).

There are very few studies in which air conditioning is assessed as an intervention aimed to manage thermoregulation impairment. In a survey study of 438 heat-sensitive people with multiple sclerosis, Christogianni et al (2022) found that around three quarters used air conditioning to manage risks of overheating. However, in a review of cooling therapies/interventions for people with multiple sclerosis, Bilgin et al (2022) did not find any studies that used any conditioning as an intervention.

No studies were found comparing the use of air conditioning with other cooling methods in illness management or treatment. One study compared the use of air conditioning with electric fans in the general population (Morris et al, 2021). The authors found that electric fans are an

appropriate way to manage risk of heat stress for adults in Australia when the ambient temperature is under 38°C. However, the authors also examined older people taking medication that may impair sweating function. They found impaired sweating function lowers the effectiveness of electric fans. This is because fans cool by both convection and evaporation (refer to 6.2 Fans for more detail). Therefore, the authors recommend supplementing electric fan use with air conditioning systems for people with impaired sweating function.

Most recommendations cited above are based on clinical opinion. Furthermore, the recommendations focus on achieving or maintaining cool indoor air temperatures, and rarely mention the means to achieve those temperatures. They do not differentiate between air conditioning and other space cooling strategies (evaporative cooling, ceiling fans, passive cooling).

6. Air conditioning and other cooling systems

Common home cooling systems include fans, evaporative cooling or refrigerated cooling. Sometimes the term air conditioning is used to refer to all these systems. Most often it is used to refer only to refrigerated cooling systems.

Not all systems will be appropriate in all circumstances. The most appropriate air conditioning system for a person will depend on factors including:

- environment – regional climate, average temperature, humidity
- building – size, layout, solar power, air flow and other passive cooling features
- occupancy – whole house or single room, rent or own, number of residents
- lifestyle – budget, habits, cooling needs, sustainability preferences (Wrigsley, 2023; Barnes, 2023; Lockyer, 2023; Milne et al, 2020; Gilmour & Steen, n.d.).

6.1 Cooling garments

Cooling garments can include jackets, vests, hats, hoods, gloves, wrist bands and thigh straps (Stevens et al. 2023; Laique & Hussain, 2018). Ren et al (2022) identify six types of cooling mechanism used in garments:

- ice cooling – garment contains insulated pockets to hold ice
- phase change materials cooling – made from a designed material that uses the latent heat from the body to lower the temperature of the microclimate between the body and the garment
- radiative cooling – made from a designed material that aims to maximise heat loss allowing more infrared radiation to escape the body
- thermo-electric cooling – garment contains conductors which can be used to directly draw heat energy from the body as an electric current is passed through the conductor
- liquid cooling – garment contains pipes carrying cold liquid and a pump to ensure liquid is spread over the garment

- air-cooling – garment that maximises ventilation through the use of design and small electric fans.

6.2 Fans

Fans work by moving air around a room more quickly. They do not cool the air, but rather aid the body's thermoregulatory processes. Faster moving air helps sweat evaporate more quickly (evaporation) and blows cooler air at the skin (convection). Fans are less effective in higher temperatures, though the exact threshold is still being debated in the literature (Morris et al, 2021; Milne et al, 2020; Iorio, 2019). Fans can be effective for healthy adults in temperatures up to 38°C (Morris et al, 2021) and may help to a lesser extent up to 42°C (Iorio, 2019). The Australian government's Your Home site states:

> Fans should be the first appliance of choice for cooling. They are cheap to run and generally use less energy than evaporative coolers or air-conditioners. Typically, the air flow created by a fan provides a similar improvement to comfort as reducing the temperature by around 3°C. With good design and insulation, fans can often supply adequate cooling for acclimatised residents in all Australian climates (Department of Climate Change, Energy, the Environment and Water; n.d).

Fans are most effective when aimed directly toward the body, in humid climates or when used in combination with water spray, wet clothing or wraps (Morris et al, 2021; Milne et al, 2020; Iorio, 2019; Department of Climate Change, Energy, the Environment and Water; n.d.).

6.3 Evaporative cooling

An evaporative cooler blows cool, humid air into a space by drawing outside air through a wet filter which is then expelled by a fan. An evaporative cooler may be less expensive to purchase and run than an air conditioning system, but this depends on the model. Evaporative coolers are less effective in humid environments and require large amount of water to operate (Milne et al, 2020; Department of Climate Change, Energy, the Environment and Water; n.d.).

6.4 Air conditioning (refrigerated cooling)

An air conditioning system that operates by refrigerated cooling draws warm air from inside the space and cools it via contact with a refrigerant gas. The cool air is blown back into the space and the extracted heat is expelled outside (Barnes, 2023; Milne et al, 2020; Barnes, 2019; Department of Climate Change, Energy, the Environment and Water; n.d.). Air conditioning systems can vary by cost, size, energy efficiency and type of refrigerant used. Air conditioners can be:

- fixed or portable
- single unit, split system, or multi-split system
- ducted or non-ducted

- reverse cycle or cooling only (Wrigsley, 2023; Barnes, 2023; Department of Climate Change, Energy, the Environment and Water, n.d.; Milne et al, 2020).

For comparison of purchase and running costs of different air conditioning systems in Australia, refer to 7. Air conditioning in Australia.

Reverse cycle air conditioning

A reverse cycle air conditioner operates in a similar way to a cooling-only system. However, a reverse cycle system is also able to reverse the refrigeration process, sending cold air outside and warm air inside. Reverse cycle air conditioners are often considered the most efficient systems because they can provide both heating and cooling. However, energy efficiency ultimately depends on a range of factors (Department of Climate Change, Energy, the Environment and Water, n.d.; Milne et al, 2020; Barnes, 2019).

Single unit, split system, or multi-split system air conditioning

Split system air conditioners have an outside unit and an inside unit. They are the most common fixed air conditioning systems and are usually more energy efficient than single unit systems. Split systems can be ducted or non-ducted. Multi-split systems have an outside unit and multiple indoor units, which can be placed in different rooms. They are an alternative to ducted systems (Barnes, 2023; Department of Climate Change, Energy, the Environment and Water, n.d.; Milne et al, 2020; Barnes, 2019).

Single unit systems are generally suited to smaller areas. They are generally less energy efficient than split systems. They can be fixed or portable. Portable systems are generally less expensive to purchase than fixed systems. They may be appropriate for smaller areas or when the system needs to be moved to different areas. They may also be appropriate when installing a fixed unit is not feasible, such as in rental properties (Lockyer, 2023; Milne et al, 2020; Barnes, 2019).

Ducted air conditioning

A ducted system is a central heating or cooling system, which means it is designed to warm or cool a whole house or building rather than a single room. Ducted systems can be evaporative coolers, reverse-cycle split systems or cooling only split systems. There is usually an outdoor unit on the roof and an indoor unit under the floor or in the ceiling. Ducts extend from the indoor unit and into multiple rooms or multiple areas of a bigger space (Department of Climate Change, Energy, the Environment and Water, n.d.; Milne et al, 2020; Barnes, 2019).

In terms of cost, ducted systems are generally more expensive to purchase, install and run, and therefore are generally less cost effective than non-ducted systems (refer to Table 2).

Installation is a significant upfront cost for ducted systems as work is required to install the roof unit as well as ducts throughout the home. Furthermore, ducted systems are not possible in some houses due to lack of space or other architectural features (King, 2023; Mullane, 2023).

Running costs are generally higher for ducted systems. Ducted systems may cool a large area faster than non-ducted single unit split systems, because the ductwork distributes the warm/cool air from a central unit. However, they are usually less energy efficient as they require a larger fan and some energy is lost as the warm/cool air travels through the ducts. As a central heating system, ducted air conditioning may waste energy if it is cooling or heating rooms that are not in use. Running costs may be partially addressed with well insulated ducting that limits energy loss. Running costs may also be reduced by using a zoned system that allows the user to switch on or off different sections of the home (Milne et al, 2020).

While ducted systems are generally less cost-effective than non-ducted systems, upfront and running costs vary widely depending on several factors (climate, temperature setting, maintenance schedule, system quality and features etc.). There may be circumstances in which ducted systems are ultimately more cost-effective. For example, if the user needs to cool a large house with multiple rooms or with very large rooms, a ducted system may end up less costly than installing multiple indoor units of a non-ducted split system. In one study based in Texas, a ducted system was compared to a non-ducted multiple split system. The authors found the ducted system was better at maintaining a constant temperature, better at humidity control and used almost 30% less energy (Bandari & Fumo, 2022). However, this study was conducted in a single house with only one model of each air conditioning system. It therefore cannot account for variables such as room size, insulation, climate etc.

Table 2 Cost comparison of ducted and non-ducted air conditioning systems

Costs	Ducted	Non-ducted
Purchase and installation cost	$9,000-$20,000 (King, 2023; Mullane, 2023)	• $600-$2800 (small) • $700-$3000 (med) • $1000-$5500 (large) (Richard & Iredale, 2023)
Running Costs (refer to Table 4)	• Cooling: $383-$1964 • Heating: $87-$1628	• Cooling: $30-$396 • Heating: $18-$528

7. Air conditioning use in Australia

Air conditioner use is increasing in Australia, up to a 2023 estimate of 86% of Australian households using air conditioning to cool their homes (Zander et al; 2023; Savvy, 2023; Godfrey, 2023; ABS, 2014). Half of households use fixed, wall mounted systems, which are evenly split between ducted and non-ducted systems (Energy Consumer Australia, 2023; refer to Table 3 for further details).

A non-ducted, reverse cycle, split system air conditioner can cost $500-$2000 plus installation costs of $600-$800. Annual running cost of a reverse cycle air conditioner at $30-$396 for

cooling and $18-$528 for heating, depending on location and the size of the room being affected. Multiple units or ducted air conditioning may be required for bigger houses, in which case the purchase, installation and running costs could be significantly greater. Portable air conditioners can be cheaper to purchase ($500-$900) but are less energy efficient than split systems and will likely cost more to run (Lockyer, 2023; Wrigsley, 2023; refer to Table 4 for further details).

Table 3 – Percentage of households with heating or cooling systems (Source: Energy Consumers Australia, 2023)

Heating/cooling system	%
Wall mounted unit	50%
Ceiling fans	42%
Portable cooling	27%
Ducted air conditioning	26%
Portable heater	21%
Portable electric or gas heaters	16%
Gas central heating^	13%
Wood burning heater	9%
Ducted evaporative cooling	8%
Fixed fire	7%
Outdoor electric or gas heaters	4%
Electric panel heaters	3%
Electric underfloor heating	2%
Hydronic heating system	1%
None of these	3%

Table 4 – Comparison of average annual air conditioner (split system, reverse-cycle) running cost for ducted and non-ducted small, medium and large rooms in Australian capital cities (Source: Wrigsley, 2023)

City	Average Usage Rates (non-ducted)	Cool/Heat (Small)	Cool/Heat (Medium)	Cool/Heat (Large)	Cool/Heat (Ducted)
Brisbane	31.2c/kWh	$155/$46	$258/$20	$396/$30	$1964/$97
Darwin	28.1c/kWh	$140/$41	$232/$18	$357/$27	$1770/$87
Sydney	35.3c/kWh	$61/$193	$101/$153	$154/$232	$780/$726
Adelaide	44.9c/kWh	$78/$246	$128/$194	$195/$295	$992/$924
Perth	30.8c/kWh	$54/$169	$88/$133	$134/$203	$681/$634
Melbourne	26.3c/kWh	$30/$206	$48/$306	$73/$471	$383/$1451
Hobart	29.5c/kWh	$33/$231	$54/$343	$81/$528	$429/$1628
Canberra	26.4c/kWh	$30/$206	$48/$307	$73/$473	$384/$1457

8. References

Allen, D. R., Huang, M., Parupia, I. M., Dubelko, A. R., Frohman, E. M., & Davis, S. L. (2017). Impaired sweating responses to a passive whole body heat stress in individuals with multiple sclerosis. Journal of neurophysiology, 118(1), 7–14. https://doi.org/10.1152/jn.00897.2016

Altıner, Ş., Ardic, S., & Çebi, A. H. (2020). Extending the Phenotypic Spectrum of Huntington Disease: Hypothermia. *Molecular syndromology*, *11*(1), 56–58. https://doi.org/10.1159/000505887

Angeli, A. M., Salonen, B. R., Ganesh, R., Hurt, R. T., Abdalrhim, A., Mueller, M., Volcheck, M., & Aakre, C. (2024). Symptom presentation by phenotype of postural orthostatic tachycardia syndrome. *Scientific reports*, *14*(1), 205. https://doi.org/10.1038/s41598-023-50886-8

Australian Bureau of Statistics. (2014). *Environmental Issues: Energy Use and Conservation (4602.0.55.001)*. Australian Government.

https://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/4602.0.55.001Mar%202014?OpenDocument

Australian Red Cross. (n.d.). *Heatstroke and heat exhaustion*. https://www.redcross.org.au/emergencies/prepare/heatstroke-and-heat-exhaustion/

Barnes, C. (2023). *What to know before buying an air conditioner*. CHOICE. https://www.choice.com.au/home-and-living/cooling/air-conditioners/buying-guides/air-conditioners

Barnes, C. (2019). *How to buy ducted reverse-cycle air conditioning*. CHOICE. https://www.choice.com.au/home-and-living/cooling/ducted-reverse-cycle-air-conditioning/buying-guides/ducted-reverse-cycle-air-conditioning

Bennetts, H., Arakawa Martins, L., van Hoof, J., & Soebarto, V. (2020). Thermal Personalities of Older People in South Australia: A Personas-Based Approach to Develop Thermal Comfort Guidelines. International journal of environmental research and public health, 17(22), 8402. https://doi.org/10.3390/ijerph17228402

Bhandari, M. & Fumo, N. (2022). Comparison Study of a Traditional Ducted Heat Pump and a Multi Split Heat Pump. *International Refrigeration and Air Conditioning Conference*. Paper 2321. https://docs.lib.purdue.edu/iracc/2321

Billig, S. C. I., Schauermann, J. C., Rolke, R., Katona, I., Schulz, J. B., & Maier, A. (2020). Quantitative sensory testing predicts histological small fiber neuropathy in postural tachycardia syndrome. *Neurology, Clinical practice*, *10*(5), 428–434. https://doi.org/10.1212/CPJ.0000000000000770

Braun, M. C., Castillo-Ruiz, A., Indic, P., Jung, D. Y., Kim, J. K., Brown, R. H., Jr, Swoap, S. J., & Schwartz, W. J. (2019). Defective daily temperature regulation in a mouse model of amyotrophic lateral sclerosis. *Experimental neurology*, 311, 305–312. https://doi.org/10.1016/j.expneurol.2018.07.008

Castelli, G., Desai, K. M., & Cantone, R. E. (2020). Peripheral Neuropathy: Evaluation and Differential Diagnosis. *American family physician*, 102(12), 732–739.

Chang, K. C., Wu, T. H., Fann, J. C. Y., Chen, S. L. S., Yen, A. M. F., Chiu, S. Y. H., ... & Liou, H. H. (2019). Low ambient temperature as the only meteorological risk factor of seizure occurrence: A multivariate study. *Epilepsy & Behavior*, 100, 106283.

Cheshire W. P., Jr (2016). Thermoregulatory disorders and illness related to heat and cold stress. *Autonomic neuroscience: basic & clinical*, 196, 91–104. https://doi.org/10.1016/j.autneu.2016.01.001

Childs, C., & Lunn, K. W. (2013). Clinical review: brain-body temperature differences in adults with severe traumatic brain injury. *Critical Care*, 17, 1-17.

Christogianni, A., O'Garro, J., Bibb, R., Filtness, A., & Filingeri, D. (2022). Heat and cold sensitivity in multiple sclerosis: A patient-centred perspective on triggers, symptoms,

and thermal resilience practices. *Multiple sclerosis and related disorders*, 67, 104075. https://doi.org/10.1016/j.msard.2022.104075

Christogianni, A., Bibb, R., Davis, S. L., Jay, O., Barnett, M., Evangelou, N., & Filingeri, D. (2018). Temperature sensitivity in multiple sclerosis: An overview of its impact on sensory and cognitive symptoms. Temperature (Austin, Tex.), 5(3), 208–223. https://doi.org/10.1080/23328940.2018.1475831

Colman, M., Syx, D., De Wandele, I., Rombaut, L., Wille, D., Malfait, Z., ... & Malfait, F. (2023). Sensory profiling in classical Ehlers-Danlos syndrome: a case-control study revealing pain characteristics, somatosensory changes, and impaired pain modulation. The Journal of Pain, 24(11), 2063-2078 https://doi.org/10.1016/j.jpain.2023.06.015

Connor, N. (2022). *What is Radiation Heat Transfer – Definition*. thermal-engineering.org. https://www.thermal-engineering.org/what-is-radiation-heat-transfer-definition/

Coon, E. A., & Low, P. A. (2018). Thermoregulation in Parkinson disease. *Handbook of clinical neurology*, 157, 715–725. https://doi.org/10.1016/B978-0-444-64074-1.00043-4

Davis, S. L., Jay, O., & Wilson, T. E. (2018). Thermoregulatory dysfunction in multiple sclerosis. Handbook of clinical neurology, 157, 701–714. https://doi.org/10.1016/B978-0-444-64074-1.00042-2

Department of Climate Change, Energy, the Environment and Water. (n.d.). *Heating and cooling*. Australian Government. https://www.energy.gov.au/households/heating-and-cooling

Douzi, W., Dugué, B., Vinches, L., Al Sayed, C., Hallé, S., Bosquet, L., & Dupuy, O. (2019). Cooling during exercise enhances performances, but the cooled body areas matter: A systematic review with meta-analyses. *Scandinavian journal of medicine & science in sports*, *29*(11), 1660–1676. https://doi.org/10.1111/sms.13521

Dupuis, L., Petersen, Å., & Weydt, P. (2018). Thermoregulation in amyotrophic lateral sclerosis. *Handbook of clinical neurology*, 157, 749–760. https://doi.org/10.1016/B978-0-444-64074-1.00046-X

Energy Consumers Australia. (2023). *Energy Consumer Behaviour Survey*. https://ecss.energyconsumersaustralia.com.au/wp-content/uploads/2023/10/ECBS-October-2023-Household-Toplines.pdf

Epilepsy Action Australia. (n.d.). *E-360 Edition 20: Weather conditions & epilepsy*. https://www.epilepsy.org.au/e-360-edition-20-weather-conditions-epilepsy/

Fealey R. D. (2018). Thermoregulation in neuropathies. *Handbook of clinical neurology*, 157, 777–787. https://doi.org/10.1016/B978-0-444-64074-1.00048-3

Fischer, S., Haas, F., & Strahler, J. (2021). A Systematic Review of Thermosensation and Thermoregulation in Anxiety Disorders. *Frontiers in physiology*, 12, 784943. https://doi.org/10.3389/fphys.2021.784943

Gilmour, K., & Steen, M. (n.d.). *Portable air conditioner buying guide*. CHOICE. https://www.choice.com.au/home-and-living/cooling/portable-air-conditioners/buying-guides/portable-air-conditioners

Girard O. (2015). Thermoregulation in wheelchair tennis-How to manage heat stress?. *Frontiers in physiology*, 6, 175. https://doi.org/10.3389/fphys.2015.00175

Godfrey, J. (2023). *Air conditioning usage statistics 2023-2024*. Finder. https://www.finder.com.au/energy/aircon-statistics

Goodkin, M. B., & Bellew, L. J. (2014). Osteopathic manipulative treatment for postural orthostatic tachycardia syndrome. *The Journal of the American Osteopathic Association*, *114*(11), 874–877. https://doi.org/10.7556/jaoa.2014.173

Gowda, R., Jaffa, M., & Badjatia, N. (2018). Thermoregulation in brain injury. *Handbook of clinical neurology*, 157, 789–797. https://doi.org/10.1016/B978-0-444-64074-1.00049-5

Grossmann, F., Flueck, J. L., Perret, C., Meeusen, R., & Roelands, B. (2021). The Thermoregulatory and Thermal Responses of Individuals With a Spinal Cord Injury During Exercise, Acclimation and by Using Cooling Strategies-A Systematic Review. *Frontiers in physiology*, 12, 636997. https://doi.org/10.3389/fphys.2021.636997

Gulcebi, M. I., Bartolini, E., Lee, O., Lisgaras, C. P., Onat, F., Mifsud, J., Striano, P., Vezzani, A., Hildebrand, M. S., Jimenez-Jimenez, D., Junck, L., Lewis-Smith, D., Scheffer, I. E., Thijs, R. D., Zuberi, S. M., Blenkinsop, S., Fowler, H. J., Foley, A., Epilepsy Climate Change Consortium, & Sisodiya, S. M. (2021). Climate change and epilepsy: Insights from clinical and basic science studies. *Epilepsy & behavior*, 116, 107791. https://doi.org/10.1016/j.yebeh.2021.107791

Hakim, A., O'Callaghan, C., De Wandele, I., Stiles, L., Pocinki, A., & Rowe, P. (2017). Cardiovascular autonomic dysfunction in Ehlers–Danlos syndrome—hypermobile type. *American Journal of Medical Genetics Part C: Seminars in Medical Genetics*, *175*(1) 168-174 https://doi.org/10.1002/ajmg.c.31543

Healthdirect. (2024). *How to take care of yourself in a heatwave*. Australian Government. https://www.healthdirect.gov.au/blog/how-to-handle-the-heatwave

Heydenreich, J., Koehler, K., Braun, H., Grosshauser, M., Heseker, H., Koenig, D., Lampen, A., Mosler, S., Niess, A., Schek, A., & Carlsohn, A. (2023). Effects of internal cooling on physical performance, physiological and perceptional parameters when exercising in the heat: A systematic review with meta-analyses. *Frontiers in physiology*, 14, 1125969. https://doi.org/10.3389/fphys.2023.1125969

Hidaka, S., Gotoh, M., Yamamoto, S., & Wada, M. (2023). Exploring relationships between autistic traits and body temperature, circadian rhythms, and age. *Scientific reports*, *13*(1), 5888. https://doi.org/10.1038/s41598-023-32449-z

Hopkins, P., Gupta, P. K., & Bilmen, J. G. (2018). *Malignant hyperthermia*. *Handbook of clinical neurology*, 157, 645–661. https://doi.org/10.1016/B978-0-444-64074-1.00038-0

Huang, M., Jay, O., & Davis, S. L. (2015). Autonomic dysfunction in multiple sclerosis: implications for exercise. Autonomic neuroscience: basic & clinical, 188, 82–85. https://doi.org/10.1016/j.autneu.2014.10.017

Iorio, K. (2019). *Do fans make any difference in a heatwave?* ABC News. https://www.abc.net.au/news/2019-12-20/are-fans-completely-useless-in-a-heatwave3f/11808944

Jeschke, M. G., Gauglitz, G. G., Kulp, G. A., Finnerty, C. C., Williams, F. N., Kraft, R., Suman, O. E., Mlcak, R. P., & Herndon, D. N. (2011). Long-term persistance of the pathophysiologic response to severe burn injury. *PloS one*, *6*(7), e21245. https://doi.org/10.1371/journal.pone.0021245

Jost W. H. (2017). Autonomic Dysfunction in Parkinson's Disease: Cardiovascular Symptoms, Thermoregulation, and Urogenital Symptoms. *International review of neurobiology*, 134, 771–785. https://doi.org/10.1016/bs.irn.2017.04.003

Kim, S. H., Kim, J. S., Jin, M. H., & Lee, J. H. (2017). The effects of weather on pediatric seizure: a single-center retrospective study (2005–2015). *Science of the total environment*, 609, 535-540.

King, K. (2023). *Ducted Air Conditioning vs Split System*. Canstar Blue. https://www.canstarblue.com.au/appliances/ducted-vs-split-system-air-con-the-pros-and-cons/

Laique, M. B., & Hussain, T. (2018). Comparison Between Traditional Air Conditioning System and Wearable Cooling/Heating Devices. *International Journal of Engineering Research and Technology*, *7*(1), 111-113.

Landero J. (2014). Postural orthostatic tachycardia syndrome: a dermatologic perspective and successful treatment with losartan. *The Journal of clinical and aesthetic dermatology*, *7*(8), 41–47 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4142820

Lenzer, B., Rupprecht, M., Hoffmann, C., Hoffmann, P., & Liebers, U. (2020). Health effects of heating, ventilation and air conditioning on hospital patients: a scoping review. *BMC public health*, *20*(1), 1287. https://doi.org/10.1186/s12889-020-09358-1

Lockyer, K. (2023). *Portable Air Conditioner vs Split System*. Canstar Blue. https://www.canstarblue.com.au/appliances/portable-or-split-system-air-conditioning-the-pros-and-cons/

Lohner, S. (2017). *Chilling Science: Evaporative Cooling with Liquids*. Scientific American. https://www.scientificamerican.com/article/chilling-science-evaporative-cooling-with-liquids/

Milne, G., Reardon, C., Ryan, P., Pavia, M., & Wyndham, J. (2020). *Heating and cooling*. Your Home: Australian Government. https://www.yourhome.gov.au/energy/heating-and-cooling

Morris, N. B., Chaseling, G. K., English, T., Gruss, F., Maideen, M. F. B., Capon, A., & Jay, O. (2021). Electric fan use for cooling during hot weather: a biophysical modelling study. The Lancet Planetary Health, 5(6), e368-e377. https://doi.org/10.1016/S2542-5196(21)00136-4

Mullane, J. (2023). *How Much Does Ducted Air Conditioning Cost?* Canstar Blue. https://www.canstarblue.com.au/appliances/ducted-air-conditioning-cost

Nakayama, Y., Shimizu, T., Matsuda, C., Haraguchi, M., Hayashi, K., Mochizuki, Y., Nagao, M., Kawata, A., & Isozaki, E. (2018). Non-motor manifestations in ALS patients with tracheostomy and invasive ventilation. *Muscle & nerve*, *57*(5), 735–741. https://doi.org/10.1002/mus.26004

Novello, B. J., & Pobre, T. (2023). Electrodiagnostic Evaluation of Peripheral Neuropathy. In StatPearls. StatPearls Publishing.

Osilla, E. V., Marsidi, J. L., Shumway, K. R., & Sharma, S. (2023). *Physiology, Temperature Regulation*. In StatPearls. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK507838/

Pfeiffer R. F. (2020). Autonomic Dysfunction in Parkinson's Disease. Neurotherapeutics : the journal of the American Society for Experimental NeuroTherapeutics, 17(4), 1464–1479. https://doi.org/10.1007/s13311-020-00897-4

Pollandt, S., & Bleck, T. P. (2018). Thermoregulation in epilepsy. Handbook of clinical neurology, 157, 737–747. https://doi.org/10.1016/B978-0-444-64074-1.00045-8

Price, M. J., & Trbovich, M. (2018). Thermoregulation following spinal cord injury. *Handbook of clinical neurology*, 157, 799–820. https://doi.org/10.1016/B978-0-444-64074-1.00050-1

Raising Children Network. (2024). Sensory sensitivities & autism: 4-18 years. https://raisingchildren.net.au/autism/behaviour/understanding-behaviour/sensory-sensitivities-asd

Rakers, F., Walther, M., Schiffner, R., Rupprecht, S., Rasche, M., Kockler, M., ... & Schwab, M. (2017). Weather as a risk factor for epileptic seizures: a case-crossover study. *Epilepsia*, *58*(7), 1287-1295.

Razi, O., Tartibian, B., Teixeira, A. M., Zamani, N., Govindasamy, K., Suzuki, K., Laher, I., & Zouhal, H. (2022). Thermal dysregulation in patients with multiple sclerosis during SARS-CoV-2 infection. The potential therapeutic role of exercise. *Multiple sclerosis and related disorders*, 59, 103557. https://doi.org/10.1016/j.msard.2022.103557

Ren, S., Han, M., & Fang, J. (2022). Personal Cooling Garments: A Review. *Polymers*, *14*(24), 5522. https://doi.org/10.3390/polym14245522

Richard, A., & Iredale, A. (2023). *What size air conditioner do you need?* CHOICE https://www.choice.com.au/home-and-living/cooling/air-conditioners/articles/what-size-air-conditioner-do-i-need

Rodríguez-Sánchez, S., Valiente, N., Seseña, S., Cabrera-Pinto, M., Rodríguez, A., Aranda, A., Palop, L., & Fernández-Martos, C. M. (2022). Ozone modified hypothalamic signaling enhancing thermogenesis in the TDP-43A315T transgenic model of Amyotrophic Lateral Sclerosis. *Scientific reports*, *12*(1), 20814. https://doi.org/10.1038/s41598-022-25033-4

Romanovsky, A. A. (2018). *Thermoregulation Part I: From basic neuroscience to clinical neurology*. Elsevier.

Savvy. (2023). Air Conditioning Use in Australia. https://www.savvy.com.au/energy/air-conditioning-use-in-australia

Shajahan, A., Culp, C. H., & Williamson, B. (2019). Effects of indoor environmental parameters related to building heating, ventilation, and air conditioning systems on patients' medical outcomes: A review of scientific research on hospital buildings. *Indoor air*, *29*(2), 161–176. https://doi.org/10.1111/ina.12531

Stevens, C. J., Singh, G., Peterson, B., Vargas, N. T., & Périard, J. D. (2023). The effect of cooling garments to improve physical function in people with multiple sclerosis: A systematic review and meta-analysis. *Multiple sclerosis and related disorders*, 78, 104912. https://doi.org/10.1016/j.msard.2023.104912

Strengers, Y., Dahlgren, K., Nicholls, L., Pink, S., Martin, R. (2021). *Digital Energy Futures: Future Home Life*. Emerging Technologies Research Lab (Monash University). Melbourne, Australia. https://www.monash.edu/__data/assets/pdf_file/0011/2617157/DEF-Future-Home-Life-Full-Report.pdf

Thompson, H. J., Tkacs, N. C., Saatman, K. E., Raghupathi, R., & McIntosh, T. K. (2003). Hyperthermia following traumatic brain injury: a critical evaluation. *Neurobiology of disease*, *12*(3), 163-173.

Thwaites, P. A., Gibson, P. R., & Burgell, R. E. (2022). Hypermobile Ehlers-Danlos syndrome and disorders of the gastrointestinal tract: What the gastroenterologist needs to know. *Journal of gastroenterology and hepatology*, *37*(9), 1693–1709. https://doi.org/10.1111/jgh.15927

Weydt, P., Dupuis, L., & Petersen, Å. (2018). Thermoregulatory disorders in Huntington disease. *Handbook of clinical neurology*, 157, 761–775. https://doi.org/10.1016/B978-0-444-64074-1.00047-1

Wrigsley, K. (2023). *How much electricity does an air conditioner use?* Canstar Blue. https://www.canstarblue.com.au/appliances/how-much-electricity-does-aircon-use/

Zander, K. K., Mathew, S., & Carter, S. (2024). Behavioural (mal) adaptation to extreme heat in Australia: Implications for health and wellbeing. *Urban Climate*, 53, 101772. https://doi.org/10.1016/j.uclim.2023.101772

Zander, K. K., van Hoof, J., Carter, S., & Garnett, S. T. (2023). Living comfortably with heat in Australia–preferred indoor temperatures and climate zones. *Sustainable Cities and Society*, 104706. https://doi.org/10.1016/j.scs.2023.104706

Zaniboni, L., & Toftum, J. (2023). Indoor environment perception of people with autism spectrum condition: a scoping review. *Building and Environment*, 110545.

Zawadzka, M., Szmuda, M., & Mazurkiewicz-Bełdzińska, M. (2017). Thermoregulation disorders of central origin - how to diagnose and treat. *Anaesthesiology intensive therapy*, *49*(3), 227–234. https://doi.org/10.5603/AIT.2017.0042

Zhang Y. (2019). Thermoregulation following Spinal Cord Injury: Theory and Fact. *Medicine and science in sports and exercise*, *51*(11), 2425. https://doi.org/10.1249/MSS.0000000000002086

Zhong, G., Bolitho, S., Grunstein, R., Naismith, S. L., & Lewis, S. J. (2013). The relationship between thermoregulation and REM sleep behaviour disorder in Parkinson's disease. *PloS one*, *8*(8), e72661. https://doi.org/10.1371/journal.pone.0072661

Long COVID-19

The content of this document is OFFICIAL.

Please note:

The research and literature reviews collated by our TAB Research Team are not to be shared external to the Branch. These are for internal TAB use only and are intended to assist our advisors with their reasonable and necessary decision-making.

Delegates have access to a wide variety of comprehensive guidance material. If Delegates require further information on access or planning matters, they are to call the TAPS line for advice.

The Research Team are unable to ensure that the information listed below provides an accurate & up-to-date snapshot of these matters

Research question: Based on latest research, would long covid be considered permanent and what is the prognosis? What are the most effective treatments? What are the outcomes of these treatments? What are the more common longer lasting effects? What is the prevalence of long COVID-19?

Date: 15/07/22

Reviewed: 20/09/23

Requestor: Jenni s47F - personal privac

Endorsed by (EL1 or above): Andrea s47F - persona

Researcher: Stephanie s47F - personal privacy

Cleared by: Stephanie s47F - personal privacy

Reviewed by: Aaron s47F - personal privacy

Next review date: 18/09/24

1. Contents

2. Summary

Update (September, 2023): Heterogeneity of research data is still a significant barrier to determining the prevalence and incidence of long COVID, the persistence of disability associated with long COVID and any effective treatment and management techniques. Estimates of activity limitation for people with long COVID vary between 16% and 80%. Estimates of prevalence vary considerably, though studies coalesce around estimates in the range of either 10%-20% or 40%-55%. The evidence base for treatment and management techniques is growing with some evidence supporting physical therapy, multimodal and personalised approaches. No pharmacological or non-pharmacological technique has emerged as a preferred pathway.

Long COVID-19 is a collection of symptoms that persist after the initial acute phase of COVID-19 infection. While some consider 4 weeks the start of prolonged symptomology, 12 weeks is emerging as the point where long COVID-19 can be diagnosed. The prevalence of long COVID-19 is difficult to determine due to heterogeneity in the research data, however it is suggested to effect between 10-20% of people who survive a COVID-19 infection.

Management of long COVID-19 will likely follow the management protocols for other post-viral syndromes, such as myalgic encephalitis/chronic fatigue syndrome, or critical illness recovery paths, for example post-intensive care syndrome.

Permanence of long COVID-19 is difficult to determine at this point as the disease is in its infancy, however most people are expected to make a recovery over many months. Nonetheless, it is expected some people will continue to have physical and/or mental impairment that significantly impacts their functional capacity. As a consequence, the United States Department of Health and Human Services advises that long COVID-19 can be considered a disability after patients complete an individualised assessment that indicates they have severely impaired functional capacity.

3. Review, September 2023

3.1 Prolonged disability

Estimates of prevalence and incidence of long COVID, and estimates of the presence of impairment or activity limitation still vary widely. Centers for Disease Control and Prevention found approximately one quarter of adults with long COVID report significant activity limitations (Ford et al, 2023). Reviewing 35 studies, Oliveira-Almeida et al (2023) found activity limitations in between 16% and 80% of subjects.

The World Health Organisation still endorses a prevalence estimate of 10-20% (WHO, 2022). Woodrow et al (2023) reviewed 73 studies and found prevalence estimates between 0% and 93%. An international systematic review considered 194 studies including 735,006 participants and found 45% of COVID-19 survivors experience ongoing symptoms at 4 months (O'Mahoney et al, 2022). In a review involving 120,970 patients, Di Gennaro et al (2023) found an incidence of 56.9%. In contrast, 10 longitudinal studies from the UK found continuation of symptoms after 12 weeks in 8-17% of cases (Hallek et al, 2023). Hallek et al (2023) found 15% of unvaccinated adults infected with SARS-CoV-2 met criteria for post-COVID syndrome, with lower incidence among vaccinated COVID-19 survivors. Evidence from the US also supports the rate of around 15%. Between 14% and 16% of respondents to the Household Pulse Survey report experiencing long COVID (National Centre for Health Statistics, 2023).

Centers for Disease Control and Prevention found approximately 16% of adults with COVID-like symptoms reported ongoing symptoms after 12 months (Montoy et al, 2023). In contrast, Woodrow et al (2023) found prevalence estimate of 48.5% after 12 months. Woodrow et al conclude that the way in which long COVID is defined and measured affects prevalence estimates. Estimates are lower in studies using routine health records (13.6%) compared with self-report studies (43.9%). The highest estimates were found in studies systematically investigating pathology (51.7%).

3.2 Treatment and management

No pharmacological or non-pharmacological treatment or management strategy has emerged as the favoured method among researchers or clinicians (Chandon et al, 2023; Chee et al, 2023; Fawzy et al, 2023; Marshall-Andon et al, 2023; Hallek et al, 2023).

In their review of 37 practice guidelines, Marshall-Andon et al (2023) found some consensus around education, shared decision making and personalised care for patients with long COVID, including tailoring the modality and setting of treatment or management to the patient's situation.

A recent review of 12 studies found physical therapy (especially, moderate exercise and interventions related to respiratory muscles) was associated with a significant improvement in fatigue, dyspnea and quality of life in patients with long COVID (Sánchez-García et al, 2023).

4. What is long COVID-19?

There is no internationally agreed definition of long COVID-19, however signs and symptoms beyond 4 weeks is considered ongoing COVID-19 (Molhave et al, 2022). The World Health Organisation (WHO) recognised the existence of continuing symptoms and effects of COVID-19 after the initial infection period in September 2020, stating long COVID-19 is:

> an illness that occurs in people who have a probable or confirmed SARS-CoV-2 infection; usually within 3 months of onset of the infection, with symptoms and effects that last for at least 3 months. These symptoms and effects cannot be explained by an alternative diagnosis (WHO, 2021a).

In the United States, Centers for Disease Control and Prevention (CDC) advise that post-COVID-19 conditions can be identified at least 4 weeks after the initial COVID-19 diagnosis (CDC, 2022). In the United Kingdom, the National Institute for Health Care and Excellence (NICE) proposes that 'acute COVID-19' is the period up to 4 weeks post infection diagnosis, 'COVID in progress' is the experience of signs and symptoms between 4-12 weeks post infection diagnosis, and post-COVID-19 syndrome are signs and symptoms that continue for more than 12 weeks after the initial infection and are not attributable to another diagnosis (NICE, 2022). A formal definition of long COVID-19 by the Australian Health Department could not be found.

The CDC suggests long COVID-19 is more common for people who had severe symptoms of COVID-19 during their initial infection (CDC, 2022), however the WHO advise there is no clear evidence of a relationship between initial severity of COVID-19 infection and the likelihood of developing long COVID-19 (WHO, 2021b). What is known, is people can suffer long COVID-19 regardless of whether they had mild or severe symptoms with the initial COVID-19 infection (Berger et al, 2021).

While research is continuing to try to identify those most at risk of long COVID-19, some risk factors may include (Berger et al, 2021; CDC, 2022):

- people who were in intensive care units during their initial COVID-19 infection
- people who have underlying health conditions prior to the infection including diabetes, heart failure, asthma, hypertension and epilepsy

- demographics with health inequities such as ethnic minority groups and people with disability
- people unvaccinated against COVID-19
- adults appear more vulnerable to long COVID-19 than children.

5. What is the prevalence of long COVID-19?

Despite the body of research emerging around long COVID-19, the prevalence is difficult to determine due to differences in study methodology, different outcome definitions and time frames, and different symptoms and levels of severity surveyed (Emecen et al, 2022). Additionally, prevalence is influenced by social determinants, such as poverty, racism and disability (Berger et al, 2021), therefore there is considerable inconsistency in the literature depending on participant demographics.

In one United Kingdom study, 18.2% of participants reported at least one symptom 6 months post initial COVID-19 infection (Emecen et al, 2022). This is in line with the WHO (2021b) estimate that around 10-20% of COVID-19 survivors experience mid- and long-term effects after the acute phase of illness has passed. However, a systematic review cited by Maglietta et al (2022), involving 57 studies and over 250,000 survivors of COVID-19, demonstrated more than half of these survivors experienced post-acute symptoms at 6 months post initial infection. I was unable to source clear data for the persistence of symptoms beyond this time point.

Further complicating prevalence data is the influence of different variants on recovery from COVID-19. Research from the United Kingdom comparing the Delta and Omicron variants suggests an increased risk of ongoing symptoms at 4 weeks post infection with Delta infection (10.8% participants) than an Omicron infection (4.5% participants) (Antonelli et al, 2022).

6. What are the most common symptoms of long COVID-19?

It has been noted that symptoms of long COVID-19 are similar to other post-viral fatigue syndromes such as myalgic encephalitis/chronic fatigue syndrome (Boaventrua et al, 2022; CDC, 2022), although the multisystem complications from long COVID-19 maybe broader and more intense than other post-viral syndromes (Boaventrua et al, 2022).

The most common symptoms of long COVID-19 reported in the literature include (Berger et al, 2021; CDC, 2022; Maglietta et al, 2022; Scordo et al, 2021; WHO, 2021b):

- Physical and mental fatigue that interferes with daily life
- Shortness of breath
- Memory and concentration problems (‘brain fog’)

- Headache
- Mental health impairment (e.g., anxiety, depression, mood swings)
- Abdominal pains
- Muscle weakness and joint pain
- Palpitations and chest pain
- Dizziness
- Gastrointestinal issues (e.g., diarrhea, stomach pain)
- Sleep problems
- Change in smell and/or taste
- Pins and needles feeling
- Skin lesions similar to chilblains

Long COVID-19 may affect people differently as different organ systems become involved, and an individual's symptoms may fluctuate or relapse over time (Berger et al, 2021; WHO, 2021b).

Figure 1 below, an excerpt from research by Yong and Liu (2021), highlights the different organ systems that may be affected by long COVID-19:

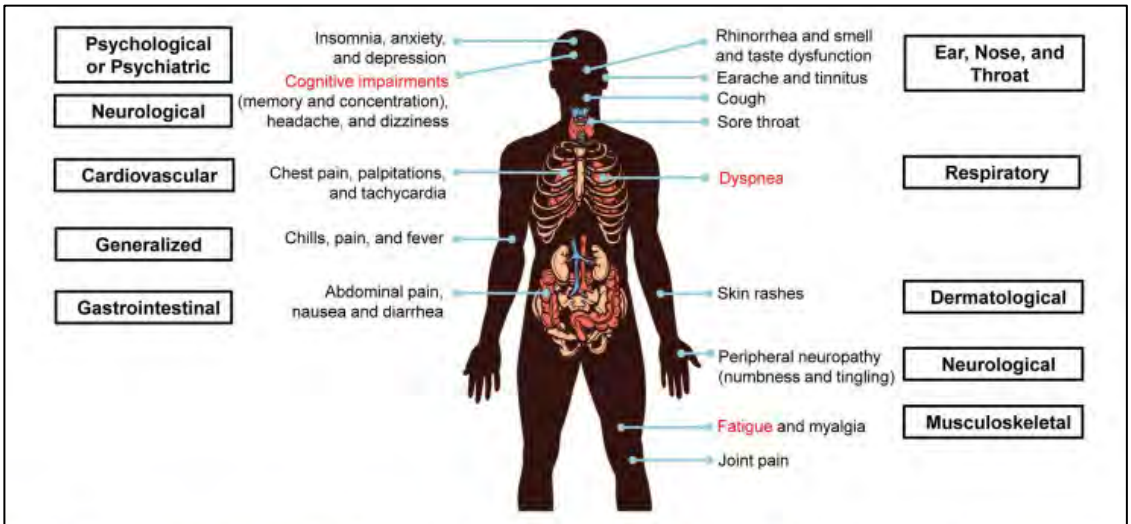


FIGURE 1 Symptoms of the post-COVID-19 syndrome (PCS). Multiple organ systems are affected, such as psychological or psychiatric (insomnia, anxiety and depression), neurological (cognitive impairments involving memory and concentration, headache, dizziness and peripheral neuropathy), ear, nose, and throat (rhinorrhoea, smell and taste alterations, earache, tinnitus, cough, and sore throat), respiratory (dyspnoea and cough), cardiovascular (chest pain, palpitations and tachycardia), gastrointestinal (abdominal pain, nausea and diarrhoea), generalised (chills, pain and fever) and musculoskeletal (fatigue, myalgia and joint pain). Dyspnoea, fatigue, and cognitive impairments are among the topmost common PCS symptoms (references in Table 1), as reviewed in Shah et al.,[3] Nasserie et al.,[10] Michelen et al.[11] and Lopez-Leon et al[19]. (The human body vector was licenced from Freepik.com.)

7. What is the current management for long COVID-19?

There is no documented specific medication to treat long COVID-19 (Molhave et al, 2022) and much of the current literature describes medical management of long COVID-19. Effective management of long COVID-19 involves symptom relief and rehabilitation (Molhave et al, 2022; WHO, 2021b), and involvement of a multidisciplinary team for patients with multiple organ systems impacted may be required (Berger et al, 2021; Kokhan et al, 2022; Molhave et al, 2022; Scordo et al, 2021; Sundar Srethstha & Love, 2021).

Particular rehabilitation programs mentioned in the literature include physical rehabilitation to improve respiratory and cardiovascular function, which is best performed within 2 months of initial diagnosis of COVID-19 (Kokhan et al, 2022; Molhave et al, 2022). Also, cognitive therapy has been shown to be effective for patients with mental fatigue (Molhave et al, 2022).

As the existence of long COVID-19 is in its infancy, there is speculation, for example by Dr Anthony Fauci of the National Institute of Allergy and Infectious Diseases, that long COVID-19 may have a similar aetiology to other post-infectious conditions such as myalgic encephalomyelitis/chronic fatigue syndrome (Scordo et al, 2021; Sundar Shrestha & Love, 2021). Therefore, exploring the aetiology and management of myalgic encephalomyelitis/chronic fatigue syndrome may give some insight into long COVID-19 syndrome (Scordo et al, 2021). Current literature indicates cognitive behaviour therapy and graded exercise therapy are important in the management of myalgic encephalitis/chronic fatigue syndrome (Sharpe et al, 2021; Snook & Slowman, 2019). Cognitive behaviour therapy focusses on challenging fatigue related cognitions and planning social and occupational rehabilitation, while graded exercise therapy involves determining baseline ability and slowly increasing intensity and duration without exacerbating symptoms (Sharpe et al, 2021; Snook & Slowman, 2019).

Another significant medical condition that may be relevant to the understanding and management of long COVID-19 is post-intensive care syndrome (PICS) - the presence of health problems common to patients who have recovered from critical illness in intensive care units (Parker et al, 2021). Similar to long COVID-19, cognitive impairment ('brain fog'), extreme fatigue, muscle weakness, and shortness of breath are among the most common symptoms of PICS (Parker et al, 2021). Parker et al (2021) suggests applying the PICS post-acute phase framework to long COVID-19 patients could involve:

- Occupational therapy – provide energy conservation and work simplification strategies; address impact of cognitive impairments on work performance; monitor for residual impairment in gross and fine motor function, sensory integration or pain related to positioning (such as prolonged proning in ICU); strengthening and fine motor training using writing aids or assistive technology.
- Physical therapy – ICU acquired weakness can persist for years after the acute illness has resolved, therefore physical therapy can be beneficial to improve strength and physical function.

- Speech therapy – intubation injuries can extend from the voice and airway to dysphagia; dysphagia can persist for months, but most patients will recover with support.
- Social workers – many patients report persistent symptoms that impact their ability to return to work. Social workers can connect patients with job resources, conduct screening for mental health impairments, and provide psychoeducation and referrals.
- Primary health care – primary health practitioners should provide aftercare and care coordination for long COVID-19 patients.

8. Permanence of long COVID-19

For most people, the natural history of long COVID-19 appears to be a gradual improvement of symptoms over many months (Berger et al, 2021; CDC, 2022; WHO, 2021b). However, the long-term prognosis for some people is unknown, as it is not known whether damaged organ systems will fully recover or if there will be lasting effects (Berger et al, 2021; Scordo et al, 2021). Unfortunately, it appears some people with long COVID-19 will continue to have long-term organ compromise, long-term complex immune and homeostatic dysfunction with disabling symptoms and impaired functional levels (Sundar Srethstha & Love, 2021).

In July 2021, long COVID-19 became a recognised disability under the Americans with Disabilities Act, Section 504 and Section 1557 (CDC, 2022; United States Department of Health and Human Services, 2021). In the United States, as long COVID-19 causes physical and/or mental impairment, it can be considered a disability if it substantially limits one or more major life activities such as caring for oneself, performing manual tasks, eating, walking or concentrating. Table 1 summarises further information from the United States Department of Health and Human Services (2021) regarding long COVID-19 as a disability.

However, whether long COVID-19 can be considered a permanent disability requires an individualised assessment to determine if the long COVID-19 symptoms and effects substantially impact the individual's functional capacity (United States Department of Health and Human Services, 2021).

Table 1

*Information regarding long COVID-19 as a disability (*United States Department of Health and Human Services, 2021)

ADA, Section 504, and Section 1557 if it substantially limits one or more major life activities. These laws and their related rules define a person with a disability as an individual with a physical or mental impairment that substantially limits one or more of the major life activities of such individual ("actual disability"); a person with a record of such an impairment ("record of"); or a person who is regarded as having such an impairment ("regarded as"). A person

with long COVID has a disability if the person's condition or any of its symptoms is a "physical or mental" impairment that "substantially limits" one or more major life activities.

a. Long COVID is a physical or mental impairment.

A physical impairment includes any physiological disorder or condition affecting one or more body systems, including, among others, the neurological, respiratory, cardiovascular, and circulatory systems. A mental impairment includes any mental or psychological disorder, such as an emotional or mental illness.

Long COVID is a physiological condition affecting one or more body systems. For example, some people with long COVID experience:

- Lung damage
- Heart damage, including inflammation of the heart muscle
- Kidney damage
- Neurological damage
- Damage to the circulatory system resulting in poor blood flow
- Lingering emotional illness and other mental health conditions

Accordingly, long COVID is a physical or mental impairment under the ADA, Section 504, and Section 1557.

b. Long COVID can substantially limit one or more major life activities

"Major life activities" include a wide range of activities, such as caring for oneself, performing manual tasks, seeing, hearing, eating, sleeping, walking, standing, sitting, reaching, lifting, bending, speaking, breathing, learning, reading, concentrating, thinking, writing, communicating, interacting with others, and working. The term also includes the operation of a major bodily function, such as the functions of the immune system, cardiovascular system, neurological system, circulatory system, or the operation of an organ.

The term "substantially limits" is construed broadly under these laws and should not demand extensive analysis. The impairment does not need to prevent or significantly restrict an individual from performing a major life activity, and the limitations do not need to be severe, permanent, or long-term. Whether an individual with long COVID is substantially limited in a major bodily function or other major life activity is determined without the benefit of any medication, treatment, or other measures used by the individual to lessen or compensate for symptoms. Even if the impairment comes and goes, it is considered a disability if it would substantially limit a major life activity when the impairment is active.

Long COVID can substantially limit a major life activity. The situations in which an individual with long COVID might be substantially limited in a major life activity are diverse. Among possible examples, some include:

- A person with long COVID who has lung damage that causes shortness of breath, fatigue, and related effects is substantially limited in respiratory function, among other major life activities
- A person with long COVID who has symptoms of intestinal pain, vomiting, and nausea that have lingered for months is substantially limited in gastrointestinal function, among other major life activities
- A person with long COVID who experiences memory lapses and “brain fog” is substantially limited in brain function, concentrating, and/or thinking.

9. References

Antonelli, M., Pujol, J. C., Spector, T. D., Ourselin, S., & Steves, C. J. (2022). Risk of long COVID associated with delta versus omicron variants of SARS-CoV-2. *Lancet (London, England)*, *399*(10343), 2263–2264. https://doi.org/10.1016/S0140-6736(22)00941-2

Berger, Z., Altiery de Jesus, V., Assoumou, S. A. & Greenhalgh, T. (2021). Long COVID and health inequities: The role of primary care. *The Milbank Quarterly, 99*(2), 519-541. https://doi.org/10.1111%2F1468-0009.12505

Boaventura, P., Macedo, S., Ribeiro, F., Jaconiano, S., & Soares, P. (2022). Post-COVID-19 condition: where are we now? *Life, 12*(4), 517. https://doi.org/10.3390/life12040517

Centre for Disease Control and Prevention. (2022). *Long COVID or Post-COVID Conditions*. Accessed from https://www.cdc.gov/coronavirus/2019-ncov/long-term-effects/index.html

Chandan, J. S., Brown, K. R., Simms-Williams, N., Bashir, N. Z., Camaradou, J., Heining, D., Turner, G. M., Rivera, S. C., Hotham, R., Minhas, S., Nirantharakumar, K., Sivan, M., Khunti, K., Raindi, D., Marwaha, S., Hughes, S. E., McMullan, C., Marshall, T., Calvert, M. J., Haroon, S., … TLC Study (2023). Non-Pharmacological Therapies for Post-Viral Syndromes, Including Long COVID: A Systematic Review. *International journal of environmental research and public health*, *20*(4), 3477. https://doi.org/10.3390/ijerph20043477

Chee, Y. J., Fan, B. E., Young, B. E., Dalan, R., & Lye, D. C. (2023). Clinical trials on the pharmacological treatment of long COVID: A systematic review. *Journal of medical virology*, *95*(1), e28289. https://doi.org/10.1002/jmv.28289

de Oliveira Almeida, K., Nogueira Alves, I. G., de Queiroz, R. S., de Castro, M. R., Gomes, V. A., Santos Fontoura, F. C., Brites, C., & Neto, M. G. (2023). A systematic review on physical function, activities of daily living and health-related quality of life in COVID-19 survivors. *Chronic illness*, *19*(2), 279–303. https://doi.org/10.1177/17423953221089309

Emecen, A. N., Keskin, S., Turunc, O., Suner, A. F., Siyve, N., Basoglu Sensoy, E., Dinc, F., Kilinc, O., Avkan Oguz, V., Bayrak, S., & Unal, B. (2022). The presence of symptoms within 6 months after COVID-19: a single-center longitudinal study. *Irish Journal of Medical Science,* 1–10. https://doi.org/10.1007/s11845-022-03072-0

Fawzy, N. A., Abou Shaar, B., Taha, R. M., Arabi, T. Z., Sabbah, B. N., Alkodaymi, M. S., Omrani, O. A., Makhzoum, T., Almahfoudh, N. E., Al-Hammad, Q. A., Hejazi, W., Obeidat, Y., Osman, N., Al-Kattan, K. M., Berbari, E. F., & Tleyjeh, I. M. (2023). A systematic review of trials currently investigating therapeutic modalities for post-acute COVID-19 syndrome and registered on WHO International Clinical Trials Platform. *Clinical microbiology and infection*, *29*(5), 570–577. https://doi.org/10.1016/j.cmi.2023.01.007

Ford N.D., Slaughter D., Edwards D., Dalton, A., {errine, C., Vahratian, A., & Saydah, S. (2023). Long COVID and Significant Activity Limitation Among Adults, by Age — United States, June 1–13, 2022, to June 7–19, 2023. *Morbidity and Mortality Weekly Report*, *72*(32), 866–870. http://dx.doi.org/10.15585/mmwr.mm7232a3

Hallek, M., Adorjan, K., Behrends, U., Ertl, G., Suttorp, N., & Lehmann, C. (2023). Post-COVID Syndrome. *Deutsches Arzteblatt international*, *120*(4), 48–55. https://doi.org/10.3238/arztebl.m2022.0409

Kokhan, S., Vlasava, S., Kolokoltsev, M., Bayankin, O., Kispayev, T., Tromfimova, N., Suslina, I., & Romanova, E. (2022). Postcovid physical rehabilitation at the sanatorium. *Journal of Physical Education and Sport, 22*(3), Art 76, 607-613. DOI:10.7752/jpes.2022.03076

Maglietta, G., Diodati, F., Puntoni, M., Lazzarelli, S., Marcomini, B., Patrizi, L., & Caminiti, C. (2022). Prognostic Factors for Post-COVID-19 Syndrome: A Systematic Review and Meta-Analysis. *Journal of clinical medicine*, *11*(6), 1541. https://doi.org/10.3390/jcm11061541

Marshall-Andon, T., Walsh, S., Berger-Gillam, T., & Pari, A. A. A. (2023). Systematic review of post-COVID-19 syndrome rehabilitation guidelines. Integrated healthcare journal, 4(1), e000100. https://doi.org/10.1136/ihj-2021-000100

Mølhave, M., Agergaard, J., & Wejse, C. (2022). Clinical Management of COVID-19 Patients - An Update. *Seminars in nuclear medicine*, *52*(1), 4–10. https://doi.org/10.1053/j.semnuclmed.2021.06.004

Montoy, J. C. C. (2023). Prevalence of Symptoms≤ 12 Months After Acute Illness, by COVID-19 Testing Status Among Adults—United States, December 2020–March 2023. *Morbidity and Mortality Weekly Report*, *72*(32), 859-865. https://www.cdc.gov/mmwr/volumes/72/wr/mm7232a2.htm

National Centre for Health Statistics. (2023). Long COVID – Household Pulse Survey 2022-2023. US Census Bureau. https://www.cdc.gov/nchs/covid19/pulse/long-covid.htm.

National Institute for Health and Care Excellence. (2022). *COVID-19 rapid guideline: managing the long-term effects of COVID-19.* Accessed from https://www.nice.org.uk/guidance/ng188/resources/covid19-rapid-guideline-managing-the-longterm-effects-of-covid19-pdf-51035515742

O'Mahoney, L. L., Routen, A., Gillies, C., Ekezie, W., Welford, A., Zhang, A., Karamchandani, U., Simms-Williams, N., Cassambai, S., Ardavani, A., Wilkinson, T. J., Hawthorne, G., Curtis, F., Kingsnorth, A. P., Almaqhawi, A., Ward, T., Ayoubkhani, D., Banerjee, A., Calvert, M., Shafran, R., … Khunti, K. (2022). The prevalence and long-term health effects of Long Covid among hospitalised and non-hospitalised populations: A systematic review and meta-analysis. *EClinicalMedicine*, 55, 101762. https://doi.org/10.1016/j.eclinm.2022.101762

Parker, A. M., Brigham, E., Connolly, B., McPeake, J., Agranovich, A. V., Kenes, M. T., Casey, K., Reynolds, C., Schmidt, K., Kim, S. Y., Kaplin, A., Sevin, C. M., Brodsky, M. B., & Turnbull, A. E. (2021). Addressing the post-acute sequelae of SARS-CoV-2 infection: a multidisciplinary model of care. *The Lancet. Respiratory medicine*, *9*(11), 1328–1341. https://doi.org/10.1016/S2213-2600(21)00385-4

Sánchez-García, J. C., Rentero Moreno, M., Piqueras-Sola, B., Cortés-Martín, J., Liñán-González, A., Mellado-García, E., & Rodriguez-Blanque, R. (2023). Physical Therapies in the Treatment of Post-COVID Syndrome: A Systematic Review. *Biomedicines*, *11*(8), 2253. https://doi.org/10.3390/biomedicines11082253

Scordo, K. A., Richmond, M. M., & Munro, N. (2021). Post-COVID-19 Syndrome: Theoretical Basis, Identification, and Management. *AACN advanced critical care*, *32*(2), 188–194. https://doi.org/10.4037/aacnacc2021492

Sharpe, M., Chalder, T., & White, P. D. (2022). Evidence-Based Care for People with Chronic Fatigue Syndrome and Myalgic Encephalomyelitis. *Journal of general internal medicine*, *37*(2), 449–452. https://doi.org/10.1007/s11606-021-07188-4

Snook, A., & Slowman, L. (2019). Chronic Fatigue Syndrome: Occupational Therapy. *CINAHL Rehabilitation Guide.*

Sundar Shrestha, D., & Love, R. (2021). Long COVID Patient Symptoms and its Evaluation and Management. *JNMA; journal of the Nepal Medical Association*, *59*(240), 823–831. https://doi.org/10.31729/jnma.6355

United States Department of Health and Human Services. (2021). *Guidance on "Long COVID" as a disability under the ADA., Section 504, and Section 1557*. United States of America Government. Accessed from https://www.hhs.gov/civil-rights/for-providers/civil-rights-covid19/guidance-long-covid-disability/index.html#footnote10_0ac8mdc

Woodrow, M., Carey, C., Ziauddeen, N., Thomas, R., Akrami, A., Lutje, V., Greenwood, D. C., & Alwan, N. A. (2023). Systematic Review of the Prevalence of Long COVID. *Open forum infectious diseases*, *10*(7), ofad233. https://doi.org/10.1093/ofid/ofad233

World Health Organisation. (2022, December). Post COVID-19 condition (Long COVID) [Fact sheet]. https://www.who.int/europe/news-room/fact-sheets/item/post-covid-19-condition

World Health Organisation. (2021a). *A clinical case definition of post COVID-19 condition by a Delphi consensus, 6 October 2021*. Accessed from https://www.who.int/publications/i/item/WHO-2019-nCoV-Post_COVID-19_condition-Clinical_case_definition-2021.1

World Health Organisation. (2021b). *Coronavirus disease (COVID-19): Post COVID-19 condition*. Accessed from https://www.who.int/news-room/questions-and-answers/item/coronavirus-disease-(covid-19)-post-covid-19-condition

Yong, S. J., & Liu, S. (2022). Proposed subtypes of post-COVID-19 syndrome (or long-COVID) and their respective potential therapies. *Reviews in Medical Virology, 32*(4), e2315. https://doi.org/10.1002/rmv.2315

10. Version control

Version	Amended by	Brief Description of Change	Status	Date
1.0	SJP131	Document creation	Cleared	15/07/22
2.0	AHR908	Review	Cleared	20/09/23

Applying to the NDIS

Quick summary: If you want to become an NDIS participant you'll need to apply to the NDIS. There are some requirements you need to meet to be eligible for the NDIS.

First, you need to be younger than 65 when you apply, be an Australian citizen or permanent resident, and live in Australia. Then, you'll need to meet the requirements for disability, early intervention, or both.

You may be eligible under the disability requirements if you have one or more impairments that are or are likely to be permanent. And this substantially impacts your ability to do daily life activities. Your impairment must also affect your social life, or your ability to work and study. And, you must be likely to need NDIS supports for your lifetime.[1] NDIS Supports are the services, items and equipment that can be funded by the NDIS.

Or you may be eligible under the early intervention requirements if you have one or more impairments that are likely to be permanent or you have developmental delay and supports are likely to benefit you by reducing your need for supports in the future. We will also consider if these supports are NDIS supports.

Or you may be eligible under both the disability and early intervention requirements.

If you think you might be eligible, we can help you apply to the NDIS. We'll talk to you about your needs, current situation and what is important to you. We'll look at all the information you give us to decide if you're eligible. If you're eligible for the NDIS, you'll become a participant, and we'll work with you to start creating your plan. If you're not eligible, an early childhood partner or local area coordinator can help you explore and access government and community services. When we work with children younger than 9 and their families, we call this early connections. When we work with people aged 9–64, we call this community connections.

What's on this page?

- What about children younger than 6 with developmental delay?
- How do you apply to the NDIS?
- How do we decide if you're eligible?
- What happens after we decide?

What do we mean by applying to the NDIS?

Applying to the NDIS means doing things to find out if you can become a participant and start getting supports under the NDIS. There is a process to follow when you apply to the NDIS. After you apply, we'll look at all the information you provide and decide if you're eligible.

If we decide you're eligible, you'll become a participant in the NDIS. We'll then work with you to create your first plan, which will include funding for any reasonable and necessary supports.

If you're not eligible, there are other services available to you, such as other government and community services. We can help you connect to these other services.

For children younger than 9, we encourage families to talk to an early childhood partner before applying to the NDIS. They can help families connect to the right supports and let families know if the NDIS is right for their child. Learn more about our early childhood approach and early connections.

Are you eligible for the NDIS?

To be eligible for the NDIS, you first need to meet the age requirements and residence requirements. This means you need to be younger than 65 when you apply, live in Australia, and be an Australian citizen or permanent resident.[2]

You will need to meet the requirements for disability[3], early intervention[4], or both.

When we say impairment in this guideline, we mean a loss of, or damage to your body's function. Under the law for the NDIS, we check if you're eligible based on your impairments, not your type of disability or diagnosis.

Disability requirements

To meet the disability requirements, we must have evidence of **all** of the following:

- You have a disability attributable to one or more impairments. This means your disability and impairments are linked.
- Your impairment is likely to be permanent.

- Your impairment means you have a substantially reduced functional capacity to do one or more daily life activities. These activities include moving around, communicating, socialising, learning, undertaking self-care, or self-management tasks.
- Your impairment affects your ability to work, study or take part in social life.
- You'll likely need NDIS supports for your lifetime.

Early intervention requirements

To meet the early intervention requirements, we must have evidence of **all** of the following:

- You have an impairment that's likely to be permanent, or that you are a child younger than 6 with developmental delay.
- Early intervention will benefit you by reducing your need for supports in the future.
- Early intervention will benefit you by either reducing the impact your impairment has on your functional capacity or support your informal supports to build their skills to help you. Or the early intervention will prevent the deterioration of your functional capacity or improve it.
- The early intervention supports you need are NDIS supports.

We'll use information in your application to decide if you're eligible for the NDIS.

If you're eligible, you become a NDIS participant.[5] The length of time you'll be a participant will depend on your situation and NDIS support needs. Learn more about leaving the NDIS.

This guideline has detailed information on how we decide if you're eligible for the NDIS. For general information about who's eligible, read Am I eligible and Applying to the NDIS factsheet. Check out our website for information about children younger than 9.

We also have information on psychosocial disability. If you have a psychosocial disability, this webpage has information on whether the NDIS is right for you, and what information we need if you apply.

A psychosocial disability isn't a mental health condition or diagnosis. A psychosocial disability means you have reduced capacity to undertake tasks and activities of daily living due to your mental health.

Do you meet the age requirements?

To be eligible for the NDIS, you must first meet the age requirements. This means you are younger than 65 on the day you apply.

How old are you?

You must be younger than 65 on the day you make your NDIS application.[6]

This means your application needs to be complete, in the format we ask for, and received by the NDIA before you turn 65. Learn more about how to complete your NDIS application.

If you're turning 65 soon and want to apply, contact us so we can help you apply on time.

Do you meet the residence requirements?

Then you must meet the residence requirements. This means:

- You are an Australian citizen or permanent resident.
- You live in Australia.

Are you an Australian citizen or permanent resident?

You must be an Australian citizen,[7] or have one of two visa types that means you can live here:

- A permanent visa[8]
- A protected special category visa[9] – this is only for some citizens of New Zealand.

Do you live in Australia?

You must live in Australia.[10] This means Australia is your home and you spend most of your time here.

To help us decide, you need to give us evidence to show us you live here. If you give us consent to use your Centrelink record, that usually gives us enough evidence to decide if you live in Australia.

If you don't give us consent, you need to give us enough information to help us decide that you live in Australia. You'll need to answer these questions:

- Where do you live?
- Where is your family?
- Do you work in Australia?
- Do you own property in Australia?
- How much time do you spend outside Australia?

Some of these questions might not apply to you, but we must consider them when we decide whether you live in Australia.[11]

We may also ask you other questions to determine whether you live in Australia.[12] For example, your family might be deployed overseas in the Defence Force, meaning you need to leave Australia for a while. In these situations, we'll ask you for more information about why you're leaving and can't return.

If you apply, we look at your whole situation when we decide if there's enough evidence to show that you live here. This will be a simple decision for us in most situations. But sometimes we may need to look at the questions below.

Where do you live?

We consider where you live, and your living situation.[13] We'll look at whether you have more permanent accommodation in Australia than any other country.

For example, you might own a home or have a formal rental agreement in Australia. This is a good sign to us that you live in Australia.

Where is your family?

We also consider where your immediate family lives.[14]

We look at where you spend most of your time with them, face-to-face. We don't consider how you connect with your family on the phone or internet.

Do you work in Australia?

If you work, we look at where you normally work or make money.[15] If you work or make money in Australia, that's a good sign you live here. If you don't work or are unemployed, we'll consider the other questions to determine whether you live in Australia.

Do you own property in Australia?

We consider what assets or property you own in Australia.[16] We also see if you have an Australian bank account.

If you own assets or property here, it doesn't always mean you live in Australia. Your assets or property will need to show you have an ongoing connection to Australia. You don't live in Australia just because you own assets or property here.

How much time do you spend outside Australia?

We consider how often you go overseas, and how long you're outside Australia when you travel.[17] We also consider why you travel overseas, such as for work, holiday or to live with family.

This is usually the most important point to help us decide if you live in Australia. You need to show us that you have a long-term and meaningful connection to living in Australia.

You don't need to stay in Australia all the time. You can still work overseas or go on holiday.

You will need to show a stronger connection to Australia than other countries if you spend a lot of time overseas.

If we decide you're eligible and create your plan, there may be times you can't use your NDIS funding overseas. This is usually after you're overseas for more than 6 weeks unless we give you more time. Learn more about when you can't use your plan.

Do you meet the disability requirements?

You meet the disability requirements if we have evidence of all of the following:[18]

- Your disability is caused by an impairment.
- Your impairment is likely to be permanent.
- Your permanent impairment substantially reduces your functional capacity to undertake one or more of the following activities: moving around, communicating, socialising, learning, or undertaking self-care or self-management tasks.
- Your permanent impairment affects your ability to work, study or take part in social life.
- You'll likely need NDIS support for your lifetime.

If you give us evidence you have been diagnosed with one or more conditions on List A, we'll likely decide you meet the disability requirements.

If you meet the disability requirements, it's likely you'll need NDIS supports for your lifetime. This means you won't have to prove your disability every time we reassess your plan.

If at any time your NDIS support needs or situation changes, we may need to check your NDIS eligibility. We will talk with you if this happens.

Learn more about how we check if you are still eligible for the NDIS.

Is your disability related to an impairment?

When we consider your disability, we think about whether any reduction or loss in your ability to do things, across all life domains, is because of an impairment.

An impairment is a loss of or damage to your body's function.

We will look at:

- your body's functions
- your body structure
- how you think and learn.

To meet the disability requirements, we must have evidence your disability is attributable by at least one of the impairments below:[19]

- **intellectual** – how you speak and listen, read and write, solve problems, and process and remember information
- **cognitive** – how you think, learn new things, use judgment to make decisions, and pay attention
- **neurological** – how your body functions
- **sensory** – how you see or hear
- **physical** – the ability to move parts of your body.

You may also be eligible for the NDIS if you have a psychosocial disability.[20] This means you have reduced capacity to do daily life activities and tasks due to your mental health.

It doesn't matter what caused your impairment, for example if you've had it from birth, or acquired it from an injury, accident or health condition.

It also doesn't matter if you have one impairment, or more than one impairment.

Is your impairment likely to be permanent?

To meet the disability requirements, we need to know that your impairment is permanent, or likely to be permanent. Permanent under the law for the NDIS means enduring. This means we need to know whether your impairments are enduring so that you require NDIS supports on an ongoing basis.

We will focus on your impairments, and not on the cause of your impairments, or your diagnoses.

You might have some periods in your life where there is a smaller impact on your daily life, because your impairment may be episodic or fluctuate in intensity[21]. Your impairment can still be permanent due to the overall impact on your life, and the likelihood that you will be impacted across your lifetime.

Even when your condition or diagnosis is permanent, we'll check if your impairment is permanent too. For example, you may not be eligible if your impairment is temporary, or if

there are known, available and appropriate evidence-based clinical, medical or other remaining treatments options that are likely to remedy the impairment.

Generally, we'll consider whether your impairment is likely to be permanent if all available and appropriate treatment options are pursued.

If you give us evidence you have been diagnosed with a condition on List B, we'll likely decide your disability is from an impairment that's likely to be permanent.

Is there any medical treatment for your impairment?

We don't fund supports to treat your impairment. The Australian health system provides health services to treat illnesses or health conditions.

Your impairment will likely be permanent if your treating professional tells us there are no further treatments that could remedy it.

Your treating professional will tell us or be asked to certify if there are medical, clinical or other treatments that are likely to remedy your impairment. We need to understand whether there are treatments that are:[22]

- **known** – the treatment can be identified by an Australian medical practitioner as a suitable treatment for your impairment
- **available to you** – we need to take account of whether there are genuine barriers that prevent you from accessing treatment including, but not limited to, the nature of your impairment and your ability to access treatment
- **appropriate for you and your impairment** – we need to consider whether the treatment could remedy your impairment and is suitable and safe for you to undergo. Your ability to undergo treatment will be assessed according to your capabilities, your health and other personal circumstances, including your living arrangements
- **evidence-based** – there's proof the treatment is likely to be effective.

When we look at what treatments are available to you, we think about whether the treatment is suitable for your personal situation. The word treatment should be understood in a broadest sense and may include changes to your diet and lifestyle.

If you're still undergoing or have recently had treatment, we may not be sure you have a permanent impairment if that treatment could remedy the impairment.[23]

In some situations, it may be clear your impairment is likely to be permanent while you're still undergoing treatment or rehabilitation. For example, you may still need treatment and rehabilitation for a spinal cord injury, but it's clear you are likely to have a permanent impairment.

You might still have a permanent impairment, even if its effects may change over time.[24]

For degenerative impairments, or those that get worse over time, we consider them permanent if treatment isn't likely to remedy the impairment. That is, the treatment won't cure the impairment or come close to removing its effects.

Does your impairment substantially reduce your functional capacity?

Your permanent impairment needs **to substantially reduce your functional capacity or ability** to undertake activities in one of the following areas:

- **Communicating** – how you speak, write, or use sign language and gestures, to express yourself compared to other people your age. We also look at how well you understand people, and how others understand you.
- **Socialising** – how you make and keep friends, or interact with the community, or how a young child plays with other children. We also look at your behaviour, and how you cope with feelings and emotions in social situations.
- **Learning** – how you learn, understand and remember new things, and practise and use new skills.
- **Mobility**, or **moving around** – how easily you move around your home and community, and how you get in and out of bed or a chair. We consider how you get out and about and use your arms or legs.
- **Self-care** – personal care, hygiene, grooming, eating and drinking, and health. We consider how you get dressed, shower or bathe, eat or go to the toilet.
- **Self-management (if older than 6)** – how you organise your life. We consider how you plan, make decisions, and look after yourself. This might include day-to-day tasks at home, how you solve problems, or manage your money. We consider your mental or cognitive ability to manage your life, not your physical ability to do these tasks.

Your impairment substantially reduces your functional capacity if you usually need disability-specific supports to participate in or complete the above tasks.[25]

These disability-specific supports include:

- a high level of support from other people, such as physical assistance, guidance, supervision or prompting.
- assistive technology, equipment or home modifications that are prescribed by your doctor, allied health professional or other medical professional.

To help us decide if you're eligible, we need to know your capacity and where you need more help. We get this information from you when you apply to the NDIS.

If you have more than one permanent impairment, we will consider them together, to see if they substantially reduce your functional capacity.

We consider how you're involved in different areas of life like home, school, work and the community, and how you carry out tasks and actions. We also consider any other factors that may impact your day-to-day life.

Your needs might go up and down each day or each month. Progressive Multiple Sclerosis (MS) can be a good example of this. We consider your ability over time, taking into account your ups and downs.

How does a child's impairment affect their daily life?

To help us decide if a child's ability is substantially reduced, we compare their abilities with other children of the same age.

If a child's ability is much less than most other children the same age, they may meet the disability requirements. For example, if they:

- need assistive technology, equipment or home modifications to participate in daily activities – except for common items like glasses
- usually need more assistance to join activities, or they can't join in.

Sometimes when a child's impairment doesn't substantially reduce their ability right now, but might in the future, we will look at the early intervention requirements. Similarly, if a child's impairment currently substantially reduces their ability, but may not after receiving supports, we will look at the early intervention requirements. Early intervention can be for children of any age, however there are different requirements for children younger than 6 with developmental delay to meet the early intervention requirements.

Learn more about the early intervention requirements.

What if you have a hearing impairment?

Some hearing impairments may lead to a substantially reduced functional capacity.

We'll generally decide you have a substantially reduced functional capacity if your hearing loss is at least 65 decibels in your better ear. This is based on a pure tone average of 500Hz, 1000Hz, 2000Hz and 4000Hz.

We may also decide you have a substantially reduced functional capacity if your hearing loss is less than 65 decibels in your better ear. We may decide this if either:

- you also have another permanent impairment, such as a vision or cognitive impairment
- you give us evidence your speech detection and speech discrimination outcomes are significantly poorer than expected.

Does your impairment affect your social, work or study life?

Then, we look at how your impairments affect your ability to work, study or take part in social life.[26] This means your permanent impairments affect how you can find and keep a job, contribute to your community, or join social activities. We get this information from you when you apply to the NDIS.

We look at your ability to do things like:

- find and keep a job, or start your own business
- study
- spend and save money
- play sport
- go to the movies
- volunteer
- travel.

It doesn't matter how much your ability to work, study or socialise is affected by your impairment. It only needs to affect your social or work life in some way for you to meet the criteria.

Will you likely need NDIS supports for your lifetime?

You must be likely to need NDIS supports for your lifetime.[27] NDIS Supports are the services, items and equipment that can be funded by the NDIS.

NDIS supports are investments that help you build or maintain your functional capacity and independence, and help you work, study or take part in social life.

Even if your needs go up and down over time, or happen episodically[28], we may still consider it's likely you'll need NDIS supports for your lifetime.[29]

We consider your overall situation to answer this question.

When we decide if you'll likely need NDIS supports for your lifetime, we consider:

- your life circumstances
- the nature of your long-term support needs
- whether your needs could be best met by the NDIS, or by other government and community services.

For example, you may have an impairment that is caused by a chronic health condition. Many chronic health conditions are most effectively managed or remedied through medical management through the health system. If this is the case, we may decide that you don't need NDIS supports for your lifetime.

Learn more about reasonable and necessary supports and NDIS supports.

Do you need early intervention?

Early intervention is usually early access to support, to help reduce the functional impacts of your impairment.

Early intervention can be for both children or adults and may only be needed for a short time. You won't need these supports for your lifetime, so your treating professional or your early childhood partner will tell us how early intervention support could benefit you or your child.

You will meet the early intervention requirements if you meet all of the following:

- You have an impairment that's likely to be permanent.
- Early intervention supports will likely benefit you, for example if it means you'll need less disability support in the future and your functional capacity will improve.
- The early intervention supports you need are NDIS supports.

There are different requirements for children younger than 6 with developmental delay to meet the early intervention requirements.

If we have evidence a child younger than 7 has been diagnosed with a condition on List D, we'll decide they meet the early intervention requirements.

You may also meet the early intervention requirements if you're aged between 0 and 25 with a hearing impairment.

We also need to understand how NDIS supports benefit you, like building your skills and increasing your capacity, so that you may no longer need NDIS supports. If you meet the early intervention requirements, your support needs are more likely to change and you may

only need NDIS supports for a short time. We'll regularly check your eligibility when we reassess your plan, and at other times too.

If you no longer meet the early intervention requirements, we'll check if you meet the disability requirements. Learn more about leaving the NDIS.

Do you have an impairment that's likely to be permanent?

To meet the early intervention requirements, there must be enough evidence that you have at least one of the impairments below and your impairment is likely to be permanent.[30]

An impairment is a loss of or damage to your body's function.

We will look at:

- your body's functions
- your body structure
- how you think and learn.

An impairment could be:[31]

- **intellectual** – how you speak and listen, read and write, solve problems, and process and remember information
- **cognitive** – as how you think, learn new things, use judgment to make decisions, and pay attention
- **neurological** – how your body functions
- **sensory** – how you see or hear
- **physical** – the ability to move parts of your body.

We also need evidence at least one of your impairments will be permanent, or likely to be permanent.[32]

When we decide if your impairment is likely to be permanent, we consider the same things as in the disability requirements.

You may also be eligible for the NDIS if you have a psychosocial disability.[33] This means you have reduced capacity to do daily life activities and tasks due to your mental health. Your psychosocial disability might vary at different times in how much it impacts your daily life. Even if it fluctuates and you have some periods where there is a smaller impact on your daily life, you might have this impairment for your lifetime.

If you give us evidence you have been diagnosed with a condition on List B, we'll decide you have an impairment that's likely to be permanent.

How will early intervention benefit you?

We need to decide that getting early intervention supports means you'll likely need fewer supports in the future.[34]

We need to know that early intervention supports will help you with at least one of the following:[35]

- addressing the impact of your impairment on your ability to move around, communicate, socialise, learn, look after yourself and organise your life
- preventing your functional capacity from getting worse
- improving your functional capacity
- supporting your informal supports, which includes building their skills to help you.

To help us decide if the early intervention will help you in these ways, we look at:[36]

- how your impairment might change over time
- how long you've had your impairment
- if there's been a significant change to your impairment
- if your needs are likely to change soon, such as if you're finishing school.

Will the early intervention supports you need be NDIS supports?

The early intervention support that you would likely benefit from must be NDIS supports.[37] NDIS supports are the services, items and equipment that can be funded by the NDIS.

Learn more about reasonable and necessary supports and NDIS supports.

What about people aged between 0 and 25 with a hearing impairment?

If you're aged between 0 and 25 with a hearing impairment, you may meet the early intervention requirements. We'll decide you meet the early intervention requirements if you give us evidence of all of the following:

- You're aged between 0 and 25.
- You have auditory neuropathy or hearing loss of at least 25 decibels in either ear at 2 or more adjacent frequencies – see below.

We need evidence of your auditory neuropathy or hearing loss from a specialist audiological assessment. The assessment might include electrophysiological testing when required. The evidence must show your hearing loss is likely to be permanent.

If you're aged 26 or older with hearing loss, we'll check if you're eligible in the same way we consider all other impairments. You may be eligible under the disability requirements.

What about children younger than 6 with developmental delay?

Children younger than 6 with a developmental delay may be eligible for the NDIS under the early intervention requirements.[38]

Developmental delay is a term used to describe a delay in a child's development. It means that a child finds it much harder to do everyday things that other children their age can do, for example, dress themselves, talk or walk. A child with developmental delay needs lots of extra help to do everyday things compared to children of the same age.

First, we need to know the child:

- is younger than 6 on the day we decide whether they're eligible[39]
- lives in Australia[40]
- is an Australian citizen or permanent resident.[41]

Then, we need to know the child has a developmental delay.[42]

Finally, we need to know the child's supports will be NDIS supports. NDIS supports are the services, items and equipment that can be funded by the NDIS.

An early childhood partner can also provide supports to children who aren't eligible for the NDIS.

Learn more about the early childhood approach and early connections.

Does the child have a developmental delay?

When we decide if a child has developmental delay, we use the definition in the law for the NDIS.[43]

We need to know the delay:

- is due to mental or physical impairments
- substantially reduces the child's functional capacity compared with other children the same age.
- means the child needs specialist services from more than one professional working as a team to support the child and for longer than 12 months.

Is the delay due to mental or physical impairments?

First, we need to know the developmental delay is due to a mental or physical impairment, or a combination of mental or physical impairments.[44]

An impairment is a loss or significant change in at least one of:

- the child's body functions
- the child's body structure
- how the child thinks and learns.

Families, early childhood partners and other professionals can understand the child's body function by:

- observing their activities during play and daily tasks
- comparing their activities to other children of the same age.

For some very young children, problems in body function can't be easily measured. If so, the child may be eligible if there is significant risk of a future disability diagnosis or developmental delay. We need evidence of this from a health or allied health professional's judgment or informed clinical opinion.

Does the delay substantially reduce the child's functional capacity?

We need to know the delay substantially reduces the child's functional capacity compared to other children their age.[45]

This means the child has a significantly lower ability to do everyday activities, when compared to children of the same age. Or the child does things in a significantly different way to other children their age because of their reduced capacity.

The child would also need much more support to do the activity, compared to other children the same age.

The substantial reduction in functional capacity must be in at least one of the following areas of major life activity:

- **Self-care** – how children take care of themselves, shower, bathe, dress, eat, drink, toilet, groom, and sleep.
- **Receptive and expressive language** – this involves skills such as gesture, sign language, listening, giving and receiving information, communicating wants and needs through facial expressions, vocalisations or speech, and interaction with others.

A substantial reduction in functional capacity for either receptive language or expressive language will meet the criteria – it doesn't need to be both.

- **Cognitive development** – learning and applying knowledge. This includes areas such as:
  - understanding and remembering information
  - attention
  - learning new things
  - practising and using new skills
  - planning and making decisions
  - problem solving
  - developing pretend play skills
  - developing play interests
  - emotional and sensory regulation
  - developing emotional intelligence
  - social awareness
  - safety awareness.
- **Motor development** – this includes participation in everyday activities like moving around the home and community and manipulating objects.

We need evidence from a health, allied health or early childhood professional, who uses multiple sources of information about the child's ability to do everyday activities.

This will include information that parents or carers report about their child. It will also include a mix of standardised assessments of developmental and functional capacity, both in everyday activities and natural settings.

It should also include observations in everyday play, learning, activities or routines to better understand how the child participates in these everyday activities.

For very young children where functional capacity can't be measured, the child may be eligible if there is significant risk of a future disability diagnosis or developmental delay. We need evidence of this from a health or allied health professional's informed clinical opinion.

Does the child need specialist services from more than one type of professional and for longer than 12 months?

We need to know that the child needs a mix of specialist care, treatment or other services, due to their developmental delay. The child must also need these services for an extended duration – that is, longer than 12 months.[46]

We need to know the child needs all of the following:

- **A service response that involves more than one professional working as a team to support the child**. This means the child needs support for multiple activities, and across multiple natural settings such as the home, community and early childhood centres. The child must need more support than what's expected for a child the same age.
- **A team that works collaboratively, by communicating and sharing information, knowledge and skills**. The support must be individually planned and coordinated. The team will build the capacity of the child's family and other important people in the child's life, such as carers, educators and professionals, about the child's individual needs. This support should be embedded in everyday play, learning, activities and routines.
- **More support than an individual discipline providing a unilateral response to a single problem**. This means the child needs support from more than one professional supporting one area of delay. This is known as interdisciplinary care. For example, a child is unlikely to be eligible if a speech pathologist alone can help their language delay, without needing support or consultation from other professionals.
- **Supports for an extended duration**. This means a health, allied health or early childhood professional who knows the child determines they need support for more than 12 months. A child will likely meet this criteria if there is clear evidence that they'll need early intervention support for more than 12 months.

We need evidence from an early childhood professional, such as an early childhood teacher, educator or allied health professional who knows the child. They need to recommend that the child needs support for multiple activities and across multiple natural settings, from a team working together.

Some children in remote areas might not have access to a team of professionals. If so, they may still be eligible if the one professional needs to provide the supports to the child across multiple activities and across multiple natural settings.

We also need evidence from an early childhood professional, such as an early childhood teacher, educator, or allied health professional, that the child needs support for more than 12 months. The professional should consider multiple sources of information, including:

- parent or carer reports
- a mix of standardised and culturally appropriate developmental or functional assessments in everyday activities and natural settings
- observations in everyday play, learning, activities, and routines.

How do we work out if the child meets the criteria for developmental delay?

We'll need a range of information about the child, observed in everyday activities and settings they usually participate in. This should include parent or carer reports and standardised assessments of developmental and or functional capacity.

Early childhood partners are early childhood professionals who give us evidence of developmental delay to help us decide if the child is eligible. An early childhood partner will meet with children and families to better understand the child's day to day life, and any concerns about their development.

Early childhood partners will observe a child in familiar places like home and childcare and may complete assessments using screening tools. This information helps us decide if a child meets the early intervention requirements for developmental delay.

Families and carers can also provide copies of existing reports, assessments or letters about the developmental delay.

We may also ask for evidence from a variety of sources, including mainstream services. For example, we may also ask for evidence from your doctor, child health nurse, or other health professional.

Learn more about providing evidence of developmental delay.

What if there are no early childhood partners in your area?

If there are no early childhood partners in the child's area, a mainstream, community, or health service can give us a report for evidence of developmental delay.

If you're in one of these areas, learn more about what evidence we need for developmental delay.

Will the child's early intervention supports be NDIS supports?

To meet the early intervention requirements, the supports must be NDIS supports.[47] NDIS supports are the services, items and equipment that can be funded by the NDIS.

Learn more about reasonable and necessary supports and NDIS supports.

What happens if a child with developmental delay is eligible?

If we decide a child with developmental delay is eligible for the NDIS, they'll become a participant. But they're usually no longer eligible after they turn 6.

This is because they will no longer meet the early intervention requirements under developmental delay. To remain an NDIS participant after they turn 6, the child will need to have an impairment that's likely to be permanent and meet the requirements for disability, early intervention, or both.

We'll talk to families or carers before a child turns 6 and explain what information we need to decide if the child is still eligible. Learn more about leaving the NDIS.

Example

Hunter is 5 years old and became a participant under the early intervention requirements for developmental delay.

We give him a new 12-month NDIS plan in August. We also talk to Hunter's family about Hunter leaving the NDIS after he is 6 years old.

Hunter's family will be able to use his NDIS funding for the full 12 months, until August the next year. By then, he'll be aged 6 years.

At the end of the 12 months, we'll talk to Hunter's family about his progress and what outcomes have been achieved. We'll listen to understand if he built capacity to work towards his goals. If there's evidence that Hunter does not have an impairment that is likely to be permanent and he no longer meets the requirements for disability, early intervention, or both, we'll decide Hunter is no longer eligible. He will be supported to leave the NDIS. We'll help his family continue to stay connected to government and community services.

Learn more about leaving the NDIS and mainstream and community supports.

What if a child doesn't meet our criteria for developmental delay?

Early childhood partners provide supports to children younger than 6 who don't meet our criteria for developmental delay.

A child may have developmental concerns. This means a child younger than 6 is developing slower compared to other children their age, but the delay doesn't meet our definition for developmental delay.

For example, a child's functional capacity may be substantially reduced in one or more areas. But it's unclear if the child needs support from a team of professionals for more than 12 months.

An early childhood partner can provide **early supports** to children younger than 6 with developmental concerns. They can also help the child's family connect to other government and community supports.

Learn more about early connections.

How do you apply to the NDIS?

Applying to the NDIS is how you let us know you want to become an NDIS participant.

If you're aged 9 and older, there are a few ways you can apply:[48]

- Your local area coordinator can help you apply. They can help you through the application process and be your point of contact. Find your nearest location.
- Sometimes you may not have a local area coordinator in your area. You can contact us on 1800 800 110 to discuss other options available to you.

For children younger than 9, we encourage families to talk to an early childhood partner before applying to the NDIS. They can provide supports to children before they apply, and let families know if the NDIS is right for their child.

When you apply, you or your authorised representative will need to:

- give us the information and any documents we need to confirm your identity. Learn more about evidence of identity and privacy.
- give us the information and any documents we need to decide if you're eligible[49]
- sign or certify the NDIS application[50]
- talk to you about your needs and current situation.

When we talk to you, we'll listen to understand what is important to you. We'll also ask questions to make sure we know all the ways we can help.

We can use this information to help you make community connections if you want us to. Learn more about community connections and early connections.

Other people can help you apply if you want them to. Sometimes they can apply on your behalf. Learn more about who can help you apply.

Learn more about how to apply to the NDIS and in the Applying to the NDIS factsheet.

What information do we need in your application?

The Evidence of Identity factsheet shows what information we need to confirm your identity. When you apply for the NDIS, you'll need to give us copies of these documents. If you can't do this, let us know so we can work out what to do depending on your situation. We'll still need to check your identity before progressing your application.

To show us you're younger than 65 when you apply, live in Australia, and that you're an Australian citizen or permanent resident, you can give us either of the following:

- consent to access and use your Centrelink record
- copies of documents or other evidence that we ask for if you apply in person or over the phone.

In most cases, we can just use your identity documents.

Who can give us evidence of your impairments?

We need evidence of your impairments, to help us work out if you're eligible. To provide this, ask your treating professional. For children younger than 6 with developmental delay, an early childhood partner can provide evidence of developmental delay. Your treating professional or early childhood partner can contact us if they need to discuss what evidence to provide.

Your treating professional might be your doctor, specialist, or allied health service provider. You should use a professional who:

- has worked with you for a long time, usually for at least 6 months
- is the most appropriate type of professional to give evidence about your impairment
- is qualified and registered in their area of practice with the Australian Health Practitioner Regulation Agency or relevant professional authority.

If your treating professional doesn't meet these requirements, we may not be able to confirm the information in your application and may need to request further information.

When we check if you're eligible for the NDIS, we mainly consider the information you give us when you apply.

Learn more about who can give us evidence of your disability or impairment on the following pages:

- Providing evidence of your disability
- Providing evidence of disability for children
- Information for GPs and health professionals.

You can also learn more about how we use, collect and store your personal information.

What if you're in a remote or very remote area?

We understand it might be hard to get your treating professional to provide evidence of your impairments in a remote or very remote area. If it's hard to get your treating professional to do this, let us know.

You might not need to give us as much evidence about your impairment as people in big cities, depending on what services are available in your area. We use a technical definition for remote and very remote. You will need to live in an area that's classified as MM6 or MM7 on the Modified Monash Model to be considered remote or very remote.

How do we check your application?

Before we can accept your application, we make sure it's been made by the right person. That is, the application is from you, or someone who can apply for you.

We then check all the answers we need have been provided, and that it is the correct information.[51]

If you don't have all the answers at the meeting, we'll help you work out what to do. You can also let us know if there is a mistake. We can work with you to help complete the application properly. We can't decide if you're eligible until we have a complete application.

We'll also let you know if we need more information and, if so, what you need to give us.

Once you have completed your application with all the right information, we'll check whether you're eligible. That is, we'll check that:

- you meet the age and residence requirements
- you meet the requirements for disability, early intervention, or both.

Learn more in the Applying to the NDIS factsheet.

Who can help you apply?

You can ask someone to help you apply if you want to. They can help you:

- make your decision to apply to the NDIS
- gather the information we need.

You can choose who helps you. For example, you could ask for help from:

- a family member
- a friend
- a carer
- a partner
- a support worker or service provider
- staff in a residential aged care facility
- your treating health professional
- hospital staff.

With your permission, we can share information with these people during your application. For example, they could call us to check how your application is progressing. You can let us know if you would like us to share information.

Can someone else apply for you?

If someone else has legal authority to make decisions for you, they can apply to the NDIS on your behalf.

If you're younger than 18, the people with parental responsibility for you will apply for you.[52] This is often your parents or legal guardian. In some situations, we can decide someone else has parental responsibility.[53] Learn more about child representatives.

If you're an adult, these people may be able to apply to the NDIS on your behalf:

- a person you give consent to act as your authorised representative – this means you give them permission to apply for you
- your guardian
- a person with power of attorney who can make personal and health decisions for you
- a person with advance care health directive.

If you're an adult and want someone else to apply for you, you can tell us in person, or over the phone.

When do we contact you to help you apply?

Sometimes, we'll reach out to you to help you apply. This might be if you live in a:

- group home or supported accommodation
- large residential facility
- residential aged care facility – also read our page on younger people in residential aged care
- rural or remote area.

How do we decide if you're eligible?

Once we have your application, we review all the information we have in your application.

This will help us decide if you're eligible for the NDIS. As part of the process, we will also need to check your identity.

You are eligible for the NDIS if you meet the requirements for:

- age
- residence
- disability, early intervention, or both.

It's likely children younger than 6 with developmental delay won't meet the disability requirements. So, they may only be eligible under the early intervention requirements.

If you don't meet either the disability requirements or the early intervention requirements, you won't be eligible for the NDIS. But an early childhood partner or local area coordinator can help you connect with other government and community supports.

When will we decide if you're eligible?

Once we have your application, we have **21 days** to decide one of the following:[54]

- you're eligible for the NDIS
- you're not eligible for the NDIS
- we need more information.

We can make a decision quicker in urgent circumstances. Let us know if your situation is urgent, for example, if you're about to leave a hospital or custodial setting. Learn more about our timeframes for urgent decisions.

How do we consider your evidence of disability?

When we're deciding if you're eligible, we may look at things like:

- how old your evidence is
- who provided your evidence.

If we get more than one type of evidence from you, we might consider some evidence over others. We call this weighing evidence.

What if we need more information to decide if you're eligible?

When we decide if you're eligible, we look at:

- the information in your NDIS application
- any other information we have.

We need enough information in your application to show us you're eligible for the NDIS by meeting the requirements for disability, early intervention, or both.

Sometimes we might need to ask you for more information. For example, we may not have enough information about your functional capacity.

We'll ask you for more information if we need it to make sure we have the full picture.[55]

We might ask you for more information if:

- your application doesn't have all the information we need
- we need to answer a particular question.

We only ask for more information if we need it to decide if you're eligible.[56] If we need more information, we'll let you know:

- what you need to do
- what information we need
- when you need to give us the information.

If we ask for more information, you'll have at least **90 days** to give it to us. We can't decide if you're eligible until we have this. You can ask for more time if you need it. We can give you more time if we think it's reasonable for your situation.[57]

If we can't contact you within **90 days**, or you don't give us the information within the timeframe, we'll withdraw your application. This means we'll stop processing your application.

If you don't get the information to us in time, you can apply again.

What happens after we get your information?

Once you give us the information we need, we then have **14 days** to decide if:[58]

- you're eligible for the NDIS
- you're not eligible for the NDIS
- we need more information – for example, if the information you gave us isn't what we need.

What happens if we don't decide on time?

If we don't meet our decision-making timeframes, we have to treat this as if we decided you're not eligible.[59]

If this happens, we'll automatically review this decision that you're not eligible.[60] We'll send you a letter to explain this. You don't need to do anything.

We'll then make sure your application is reviewed by a staff member who wasn't involved in the original application. We'll contact you to let you know the outcome.

Learn more about reviewing our decisions.

What happens after we decide?

What happens if you're eligible?

On the day we decide you're eligible for the NDIS, you become a NDIS participant.[61]

The time that you remain eligible for the NDIS depends on your individual circumstances and NDIS support needs.

You'll need to continue to be eligible for the NDIS. This means you'll need to continue to live in Australia and be an Australian citizen or permanent resident. You'll also need to continue to meet requirements for disability, early intervention, or both. Learn more about whether you will always be eligible and leaving the NDIS.

We'll send you a letter to let you know:

- you are eligible

- if you met the requirements for disability, early intervention, or both
- the next steps.

Your letter will also confirm the date you became eligible for the NDIS.[62]

How will we create your first plan?

After you receive the letter confirming you're eligible, we'll contact you to organise your first planning conversation. We'll contact you within **21 days**.

We'll then work together to create your plan. If you received help to make community connections, we can build on the information and goals we talked about and include these supports in your plan. Learn more about community connections and early connections.

You'll receive a plan that sets out your NDIS supports. NDIS supports are the services, items and equipment that can be funded by the NDIS.

Your plan will include NDIS supports for the impairments that meet the disability or early intervention requirements.

For example, you may have many impairments, but only one meets our eligibility criteria. Or you might get another impairment after we decide you're eligible. If so, we only fund NDIS supports for impairments that meet the requirements for disability, early intervention, or both.

If you're aged 7 or older, we must approve your first plan within **56 days** after you become a participant.

For children younger than 7, we'll approve their first plan within **90 days** after they become a participant.

For more information, check out creating your plan.

Will you always be eligible for the NDIS?

There are many reasons for leaving the NDIS.

Some people decide they don't want to be a participant anymore.

You'll also leave the NDIS if you're no longer eligible.

When we reassess your plan, we check that all your details are correct and up to date. We also look at any new information we have received.

If you're eligible under the **early intervention requirements**, your support needs are more likely to change. We'll check at each plan reassessment and at other times, whether you still meet the early intervention requirements.

For example, during a plan reassessment it may show you no longer meet the early intervention requirements because you have built your skills and capacity and will no longer benefit from NDIS supports.

If you're eligible under the disability requirements, your disability is permanent. We don't expect your disability to change, and it's likely you'll need NDIS supports for your lifetime. We will only ask you for more information about your eligibility if there is evidence that you may no longer meet the disability requirements.

You can find out more about the eligibility requirements, and how we check these, at Am I eligible. Children with developmental delay will usually leave the NDIS after they turn 6.

Over time, you might develop your skills and independence and not need NDIS supports anymore.

If you met the requirements for early intervention and not disability, you usually won't be eligible after the early intervention supports, which are NDIS supports have benefitted you. For example, if you needed early intervention supports to achieve your goal to improve your functional capacity, and your functional capacity improves, you may no longer meet the early intervention requirements anymore.

If you're no longer eligible, we'll help you transition from the NDIS and make sure you are connected with other services in your community, if you need them. We'll also keep your information, so you can apply again if your situation changes.

Learn more about leaving the NDIS.

What happens if you're not eligible?

If you're not eligible, you can't become an NDIS participant.

We'll try and contact you by phone, or your preferred contact method,[63] to explain why you're not eligible. We'll give you reasons for our decision, and answer any questions you might have.

We'll also send you a letter with our decision, including the reasons you're not eligible and what to do next. Your letter will confirm the date we made the decision.

Even if you're not eligible for the NDIS, your early childhood partner or local area coordinator can help you explore and access government and community supports. When we work with children under 9 and their families, we call this early connections. When we work with people aged 9–64, we call this community connections.

Your early childhood partner or local area coordinator will use the information you shared to suggest supports in your community. They will work with you to see how these supports may

help you with what is important to you. We also have a list of other government and community supports you can get, even if you're not eligible for the NDIS.

What if you don't agree with our decision?

If you don't agree with our decision that you're not eligible, you should contact us. We can help explain our decision and what your options or next steps might be.

You can also ask for an internal review.[64] Another staff member, who wasn't involved in the original decision, will then check if we made the right decision. You need to ask for an internal review within **3 months** after receiving the decision.[65] Learn more about reviewing our decisions.

But you can't ask for an internal review if:

- you withdraw your application because you don't want to apply anymore
- a decision has not been made and we ask you for more information
- we withdraw your application because you didn't give us information on time
- it's been more than 3 months since you received our decision that you're not eligible.

If you don't agree with the internal review decision, you can ask the Administrative Appeals Tribunal to review it. We call this an external review. You can't ask for an external review until after we make the internal review decision.

You can contact us to discuss any concerns you may have about the process. You can also make a complaint if you're not happy with any part of the process.

Can you apply again?

Yes. If we decide you're not eligible, or you're no longer eligible, you can apply again, unless you have requested a review of that decision and are waiting for a decision to be made on the outcome of your review. This includes when you apply for the NDIS or leave the NDIS after your status as a participant has been revoked. Learn more about leaving the NDIS.[66]

You'll follow the same process to apply as you did the first time. Remember, you need to be younger than 65 on the day you make your new application. And children with developmental delay will need to be younger than 6 on the day they apply.

If you've asked for an internal review of the decision, you can't apply to the NDIS again until we've completed that review.[67] You can ask us to stop our internal review at any time.[68]

Also, if your review is with the Administrative Appeals Tribunal after an internal review, you can't apply again until it has made a decision.[69] You can also ask the Tribunal at any time to withdraw your application. Learn more on the Administrative Appeals Tribunal website.

Applying to the NDIS – Appendices

What's on this page?

List A: Conditions that are likely to meet the disability requirements

1. **Intellectual disability** diagnosed and assessed as moderate, severe or profound in accordance with current DSM criteria.
2. **Autism** diagnosed by a specialist multi-disciplinary team, paediatrician, psychiatrist or clinical psychologist experienced in the assessment of Pervasive Developmental Disorders and assessed using the current Diagnostic and Statistical Manual of Mental Disorders (DSM-V) diagnostic criteria as having severity of Level 2 (Requiring substantial support) or Level 3 (Requiring very substantial support).
3. **Cerebral palsy** diagnosed and assessed as severe (e.g. assessed as Level 3, 4 or 5 on the Gross Motor Function Classification System - GMFCS).
4. **Genetic conditions** that consistently result in permanent and severe intellectual and physical impairments:
    - Angelman syndrome
    - Coffin-Lowry syndrome in males
    - Cornelia de Lange syndrome
    - Cri du Chat syndrome
    - Edwards syndrome (Trisomy 18 – full form)
    - Epidermolysis Bullosa (severe forms):

  - YR
  - Autosomal recessive dystrophic epidermolysis bullosa
  - Hallopeau-Siemens type
  - Herlitz Junctional Epidermolysis Dystrophica
- Lesch-Nyhan syndrome
- Leigh syndrome
- Leukodystrophies:
  - Alexander disease (infantile and neonatal forms)
  - Canavan disease
  - Krabbe disease (globoid cell leukodystrophy) – Infantile form
  - Pelizaeus-Merzbacher Disease (Connatal form)
- Lysosomal storage disorders resulting in severe intellectual and physical impairments:
  - Gaucher disease Types 2 and 3
  - Niemann-Pick disease (Types A and C)
  - Pompe disease
  - Sandhoff disease (infantile form)
  - Schindler disease (Type 1)
  - Tay-Sachs disease (infantile form)
- Mucopolysaccharidoses – the following forms:
  - MPS 1-H (Hurler syndrome)
  - MPS III (San Fillipo syndrome)
  - Osteogenesis Imperfecta (severe forms):
  - Type II - with two or more fractures per year and significant deformities severely limiting ability to perform activities of daily living
- Patau syndrome
- Rett syndrome
- Spinal Muscular Atrophies of the following types:

- Werdnig-Hoffmann disease (SMA Type 1- Infantile form)
- Dubowitz disease (SMA Type II – Intermediate form)
- X-linked spinal muscular atrophy

5. **Spinal cord injury** or **brain injury** resulting in paraplegia, quadriplegia or tetraplegia.
6. **Hemiplegia** where there is severe or total loss of strength and movement in the affected limbs of the body.
7. **Permanent blindness** in both eyes, diagnosed and assessed by an ophthalmologist as follows:
   - Corrected visual acuity (extent to which an object can be brought into focus) on the Snellen Scale must be less than or equal to 6/60 in both eyes; or
   - Constriction to within 10 degrees or less of arc of central fixation in the better eye, irrespective of corrected visual acuity (i.e. visual fields are reduced to a measured arc of 10 degrees or less); or
   - A combination of visual defects resulting in the same degree of visual impairment as that occurring in the above points. (An optometrist report is not sufficient for NDIS purposes.)
8. **Permanent bilateral hearing loss** > 90 decibels in the better ear (pure tone average of 500Hz, 1000Hz, 2000Hz and 4000Hz).
9. **Deafblindness** confirmed by ophthalmologist and audiologist and assessed as resulting in permanent and severe to total impairment of visual function and hearing.
10. **Amputation** or congenital absence of 2 limbs – for example, 2 legs, 2 arms, or a leg and an arm (not a leg and a hand, or an arm and a foot).

List B: Conditions that are likely to result in a permanent impairment

Conditions primarily resulting in intellectual or learning impairment

- Intellectual disability
- Pervasive developmental disorders not meeting severity criteria in List A or List C, such as autism
- Asperger syndrome
- Atypical autism

- Childhood autism.

Chromosomal abnormalities resulting in permanent impairment and not specified on List A

- Aicardi-Goutières syndrome
- CHARGE syndrome
- Cockayne syndrome Types I and Type II/Cerebro-oculo-faciao-skeletal (COFS) syndrome /Pena Shokeir syndrome Type II/Weber-Cockayne syndrome/Neill-Dingwall syndrome)
- Cohen syndrome
- Dandy-Walker syndrome
- DiGeorge syndrome /22q11.2 deletion syndrome/Velocardiofacial syndrome/ Shprintzen syndrome/Conotruncal anomaly face syndrome
- Down syndrome/Trisomy 21
- Fragile X syndrome
- Kabuki syndrome
- Menkes disease
- Prader-Willi syndrome
- Seckel syndrome /microcephalic primordial dwarfism/Harper’s syndrome/Virchow-Seckel dwarfism
- Smith-Lemli-Optiz syndrome
- Smith-Magenis syndrome
- Spinal muscular atrophy Types III and IV
- Sturge-Weber syndrome
- Trisomy 9
- Tuberous sclerosis
- Turner syndrome
- Williams syndrome
- Wolf-Hirschhorn syndrome.

Conditions primarily resulting in Neurological impairment

- Alzheimer's dementia
- Creutzfeldt-Jakob disease
- HIV dementia
- Huntington's disease
- Multi-infarct dementia
- Parkinson's disease
- Post-polio syndrome
- Vascular dementia.

Systemic atrophies primarily affecting the central nervous system

- Abetalipoproteinaemia
- Adult-onset spinal muscular atrophy/late-onset SMA type III)
- Fazio-Londe disease/Progressive bulbar palsy of childhood
- Friedrich's ataxia
- Hereditary spastic paraplegia/ Infantile-onset ascending hereditary spastic paralysis/ L1 syndrome/ spastic paraplegias types 2 and 11Huntington's disease/Huntington's chorea
- Louis-Bar syndrome/Ataxia-telangiectasia
- Motor neuron disease/Motor neurone disease/ Lou Gehrig's disease /Amyotrophic lateral sclerosis
- Primary lateral sclerosis
- Progressive bulbar palsy
- Spinal muscular atrophy – all types
- Spinocerebellar Ataxia – all types, including Machado-Joseph disease.

Extrapyramidal and movement disorders

- Hallervorden-Spatz syndrome /Pantothenate kinase-associated neurodegeneration (PKAN)/neurodegeneration with brain iron accumulation 1 (NBIA 1)
- Parkinson's disease

- Shy-Drager syndrome /Multiple System Atrophy /Striatonigral degeneration (MSA-P)/ Sporadic olivopontocerebellar atrophy (MSA-C)
- Steele-Richardson-Olszewski syndrome/Progressive supranuclear ophthalmoplegia
- Stiff-man syndrome /Stiff-person syndrome.

Other degenerative diseases of the nervous system

- Alzheimer's disease
- Alpers disease/Grey-matter degeneration/Alpers syndrome/progressive sclerosing poliodystrophy/progressive infantile poliodystrophy
- Lewy body dementia
- Pick's disease.

Demyelinating diseases of the central nervous system

- Adrenoleukodystrophy
- Multiple sclerosis
- Schilder's disease /Diffuse myelinoclastic sclerosis – non-remitting.

Episodic and paroxysmal disorders

- Brain stem stroke syndrome
- Cerebellar stroke syndrome
- Motor and sensory lacunar syndromes
- Lennox syndrome /Lennox-Gastaut syndrome
- West's syndrome.

Polyneuropathies and other disorders of the peripheral nervous system

- Adult Refsum disease
- Charcot-Marie-Tooth disease/Hereditary motor and sensory neuropathy/ peroneal muscular atrophy
- Dejerine-Sottas disease /Dejerine-Sottas syndrome/Dejerine-Sottas neuropathy/progressive hypertrophic interstitial polyneuropathy of childhood/onion bulb neuropathy
- Infantile Refsum disease.

Other disorders of the nervous system

- Hydrocephalus
- Multiple system atrophy.

Conditions resulting in Physical impairment

- Amputation
- Congenital absence of limb or part thereof
- Epidermolysis bullosa
- Harlequin type icthyosis
- Juvenile arthritis / Stills Disease (excluding monocyclic/self-limited Adult Onset Stills disease)
- Rheumatoid arthritis.

Diseases of myoneural junction and muscle

- Andersen-Tawil syndrome/ Periodic paralysis /myoplegia paroxysmalis familiaris
- Becker muscular dystrophy
- Congenital muscular dystrophy
- Distal muscular dystrophy
- Duchenne muscular dystrophy
- Facioscapulohumeral muscular dystrophy
- Limb-girdle muscular dystrophy
- Mitochondrial myopathy
- Myotonic dystrophy /dystrophia myotonica
- Myotonic muscular dystrophy
- Myotubular myopathy
- Oculopharyngeal muscular dystrophy
- Paramyotonia Congenita
- Thomsens disease /Congenital myotonia/ Becker myotonia).

Cerebral palsy and other paralytic syndromes not meeting severity criteria on List A

- Cerebral palsy
- Diplegia
- Hemiplegia
- Monoplegia
- Paraplegia
- Quadriplegia
- Tetraplegia.

Conditions resulting in Sensory and/or Speech impairment

Disorders of the choroid and retina where permanent blindness diagnostic and severity criteria on List A are not met

- Behr's syndrome
- Kearns-Sayre syndrome
- Optic atrophy
- Retinitis pigmentosa
- Retinoschisis (degenerative and hereditary types/juvenile retinoschisis)
- Stargardt disease
- Usher syndrome.

Disorders resulting in hearing loss

- Cortical deafness
- Pendred syndrome
- Sensorineural hearing loss
- Stickler syndrome
- Usher syndrome
- Waardenburg syndrome.

Conditions resulting in multiple types of impairment

- Aceruloplasminemia

- Addison-Schilder disease /Adrenoleukodystrophy
- Albinism
- Arginosuccinic aciduria
- Aspartylglucosaminuria
- Cerebrotendinous xanthomatosis /cerebral cholesterosis
- Congenital cytomegalovirus infection
- Congenital iodine-deficiency syndrome /cretinism
- Congenital rubella syndrome
- Glycine encephalopathy /non-ketotic hyperglycinaemia
- GM1 gangliosidosis
- Hartnup disease
- Homocystinuria
- Lowe syndrome/ Oculocerebrorenal syndrome
- Mannosidosis
- Menkes disease
- Mucolipidosis II /I-cell disease
- Mucolipidosis III /pseudo-Hurler polydystrophy
- Mucolipidosis IV
- Neuronal ceroid lipofuscinosis (NCL)/ Adult type (Kuf's or Parry's disease)/ Juvenile (Batten disease)/ Late infantile (Jansky-Bielschowsky)
- Niemann-Pick disease
- Pyruvate carboxylase deficiency
- Pyruvate dehydrogenase deficiency
- Sialidosis
- Sulfite oxidase deficiency.

The following mucopolysaccharidoses

- Scheie syndrome /MPS 1-H
- Hurler-Scheie syndrome /MPS 1 H-S

- Hunter syndrome /MPS II
- Morquio syndrome /MPS IVA
- Maroteaux-Lamy syndrome /MPS VI
- Sly syndrome /MPS VII.

Congenital conditions – cases where malformations cannot be corrected by surgery or other treatment and result in permanent impairment but with variable severity

- Arnold-Chiari Types 2 and 3/Chiari malformation
- Microcephaly
- Fetal alcohol spectrum disorder
- Fetal hydantoin syndrome
- Spina bifida
- VATER syndrome /VACTERL association.

List C: What if you're receiving disability support in Western Australia?

Please note: the transition of people formerly in Western Australian government disability programs is now complete and List C is no longer in operation.

If you were a participant in a WA defined program and are in the process of applying to the NDIS before 3 October 2024, please contact 1800 800 110 or enquiries@ndis.gov.au to discuss whether List C arrangements still apply.

We have an agreement with the Western Australian government to bring Western Australians onto the NDIS. If you're receiving disability supports in Western Australia, you might already meet most of the eligibility criteria. We'll send you a letter with all the details on how to apply.

The Western Australian government will let us know if you're on a program for faster access to the NDIS. This is called a defined program.

If you're on one of these programs, you need to show us that you:

- are younger than 65 on the day you apply
- live in Australia permanently
- are an Australian citizen or permanent resident.

We'll let you know what evidence you need to give us when you apply.

If you show us you meet the above requirements, we'll decide you're eligible under the **disability requirements**.

Which Western Australian defined programs are eligible for the NDIS?

- WA state-administered National Disability Insurance Scheme
- Supported Community Living
- Community Residential
- Day Options
- Disability Professional Services
- Emergency Accommodation
- Respite
- LAC Coordination
- Recreation.

List D: Permanent impairment/Early intervention, under 7 years. No further assessment required.

Synonyms for conditions are also shown (e.g. condition / synonym / synonym).

Conditions primarily resulting in Intellectual/ learning impairment

Chromosomal abnormalities resulting in permanent impairment

- Global Developmental Delay
- Aicardi syndrome
- Aicardi-Goutières syndrome
- Angelman syndrome
- CHARGE syndrome
- Cockayne syndrome/ Types I and Type II / Cerebro-oculo-faciao-skeletal (COFS) syndrome/ Pena Shokeir syndrome Type II / Weber-Cockayne syndrome/ Neill-Dingwall syndrome
- Coffin-Lowry syndrome

- Cohen syndrome
- Cornelia de Lange syndrome
- Cri du Chat syndrome
- Dandy-Walker syndrome
- DiGeorge syndrome/ 22q11.2 deletion syndrome/ Velocardiofacial syndrome/ Shprintzen syndrome/ Conotruncal anomaly face syndrome
- Down syndrome/ Trisomy 21
- Edwards syndrome/ Trisomy 18
- Fragile X syndrome
- Kabuki syndrome
- Lesch-Nyhan syndrome/ Nyhan’s syndrome/ Kelley-Seegmiller syndrome/ Juvenile gout
- Leigh syndrome/ Leigh’s disease/ subacute necrotizing encephalomyelopathy
- Menkes disease
- Patau syndrome/ Trisomy 13
- Prader-Willi syndrome
- Rett syndrome
- Seckel syndrome/ microcephalic primordial dwarfism/ Harper’s syndrome/ Virchow-Seckel dwarfism
- Smith-Lemli-Optiz syndrome
- Smith-Magenis syndrome
- Sturge-Weber syndrome
- Trisomy 9
- Tuberous sclerosis
- Williams syndrome
- Wolf-Hirschhorn syndrome.

Conditions primarily resulting in Neurological impairment

Systemic atrophies primarily affecting the central nervous system

- Friedrich's ataxia
- Hereditary spastic paraplegia/ Infantile-onset ascending hereditary spastic paralysis/ L1 syndrome/ spastic paraplegias types 2 and 11
- Louis-Bar syndrome/ Ataxia-telangiectasia
- Niemann-Pick disease (Types A and C)
- Progressive bulbar palsy of childhood/ Fazio-Londe disease.

The following spinal muscular atrophies

- Spinal muscular atrophy Type I/ Werdnig Hoffmann disease/ infantile SMA
- Spinal muscular atrophy Type II/ Dubowitz disease
- Spinal muscular atrophy Type III Kugelberg-Welander disease/ juvenile SMA
- Spinal muscular atrophy lower extremity dominant/ SMA-LED
- X-linked spinal muscular atrophy.

Extrapyramidal and movement disorders

- Hallervorden-Spatz syndrome / Pantothenate kinase-associated neurodegeneration (PKAN)/ neurodegeneration with brain iron accumulation 1 (NBIA 1)
- Alpers disease/ Alpers syndrome/ Grey-matter degeneration/ Progressive sclerosing poliodystrophy/ Progressive infantile poliodystrophy
- Demyelinating diseases of the central nervous system
- Adrenoleukodystrophy / X-linked childhood cerebral form
- Alexander disease
- Canavan disease
- Krabbe disease/ Globoid cell leukodystrophy
- Pelizaeus-Merzbacher disease.

Episodic and paroxysmal disorders

- Lennox-Gastaut syndrome/ Lennox syndrome

- West's syndrome.

Polyneuropathies and other disorders of the peripheral nervous system

- Dejerine-Sottas disease/ Dejerine-Sottas syndrome/ Dejerine-Sottas neuropathy/ progressive hypertrophic interstitial polyneuropathy of childhood/onion bulb neuropathy
- Infantile Refsum disease.

Conditions primarily resulting in physical impairment

- Amputation
- Diamond-Blackfan anaemia
- Epidermolysis bullosa
- Harlequin type icthyosis
- Hay Wells syndrome/ ankyloblepharon/ ectodermal dysplasia/ clefting [AEC] syndrome
- Joint or limb deformities resulting in impaired mobility
- Juvenile arthritis/ Stills Disease
- Osteogenesis imperfecta
- Sjogren Larsson syndrome.

Diseases of myoneural junction and muscle

- Congenital muscular dystrophy
- Congenital myotonia / Thomsens disease/ Becker myotonia
- Distal muscular dystrophy
- Duchenne muscular dystrophy
- Emery-Dreifuss muscular dystrophy
- Facioscapulohumeral muscular dystrophy
- Myotubular myopathy
- Oculopharyngeal muscular dystrophy
- Paramyotonia Congenita.

Cerebral palsy and other paralytic syndromes

- Cerebral palsy
- Diplegia
- Hemiplegia
- Monoplegia
- Paraplegia
- Quadriplegia
- Tetraplegia.

Conditions resulting in sensory and/or speech impairment

- Permanent blindness in both eyes, diagnosed and assessed by an ophthalmologist as follows either:
    - Corrected visual acuity (extent to which an object can be brought into focus) on the Snellen Scale must be less than or equal to 6/60 in both eyes
    - Constriction to within 10 degrees or less of arc of central fixation in the better eye, irrespective of corrected visual acuity (i.e. visual fields are reduced to a measured arc of 10 degrees or less)
    - A combination of visual defects resulting in the same degree of visual impairment as that occurring in the above points.

  (An optometrist report is not sufficient for NDIS purposes.)
- Deafblindness confirmed by ophthalmologist and audiologist and assessed as resulting in permanent and severe to total impairment of visual function and hearing.

Conditions resulting in multiple types of impairment

- Aceruloplasminemia
- Addison-Schilder disease/ Adrenoleukodystrophy /
- Albinism
- Arginosuccinic aciduria
- Aspartylglucosaminuria
- Cerebrotendinous xanthomatosis/ cerebral cholesterosis

- Congenital cytomegalovirus infection
- Congenital hypothyroidism
- Congenital iodine-deficiency syndrome /cretinism
- Congenital rubella syndrome
- Galactosaemia with long term learning disabilities and neurological impairment
- Glycine encephalopathy/ non-ketotic hyperglycinaemia
- GM1 gangliosidosis
- Hartnup disease
- Homocystinuria
- Lowe syndrome/ Oculocerebrorenal syndrome
- Mannosidosis
- Menkes disease
- Mucolipidosis II / I-cell disease
- Mucolipidosis III / pseudo-Hurler polydystrophy
- Mucolipidosis IV
- Neuronal ceroid lipofuscinosis
- Niemann-Pick disease
- Phenylketonuria
- Pyruvate carboxylase deficiency
- Pyruvate dehydrogenase deficiency
- Sialidosis
- Sulfite oxidase deficiency.

The following mucopolysaccharidoses

- Hurler syndrome/MPS1-H
- Scheie syndrome/ MPS 1-S
- Hurler-Scheie syndrome/ MPS 1 H-S
- Hunter syndrome/ MPS II

- San Fillipo syndrome/ MPS III
- Morquio syndrome/ MPS IVA
- Maroteaux-Lamy syndrome/ MPS VI
- Sly syndrome/ MPS VII.

The following lysosomal storage disorders

- Gaucher disease Types 2 and 3
- Niemann-Pick disease (Types A and C)
- Pompe disease
- Sandhoff disease (infantile form)
- Schindler disease (Type 1)
- Tay-Sachs disease (infantile form).

Congenital conditions – cases where malformations cannot be corrected by surgery or other treatment and result in permanent impairment

- Chiari malformation/Arnold-Chiari malformation
- Congenital absence of limb(s)
- Congenital hydrocephalus
- Fetal alcohol spectrum disorder
- Fetal hydantoin syndrome
- Microcephaly
- Spina bifida
- VATER syndrome (VACTERL association).

When do we make priority eligibility decisions?

If you're in one of the following situations, we'll decide if you're eligible within **2 to 5 business days**.

- Child younger than 7 years with a hearing impairment, either:
  - Identified as Hearing Australia or Early Childhood Partner Priority
  - Identified as 'newly diagnosed'.

- A child is identified as having a developmental delay and is turning 6 years old within 30 days of a valid NDIS application.
- **Immediate risk** to self, others, community or agency where appropriate disability or informal supports are not in place.
- **Unexpected, significant deterioration** of disability-related functional capacity where appropriate disability or informal supports are not in place.
- **Rapid deterioration** in functional capacity of a person with one of the following permanent disabilities:
    - Amyotrophic Lateral Sclerosis (ALS or Lou Gehrig's Disease)
    - Brain Cancer
    - Motor Neurone Disease (MND)
    - Progressive Bulbar Palsy (PBP)
    - Primary Lateral Sclerosis (PLS)
    - Progressive Muscular Atrophy (PMA).
- A **terminal illness** and disability
- **Imminent risk** (within 1–14 days) of breakdown of either:
    - Accommodation – risk of homelessness
    - Caring arrangements, including informal supports, due to death, serious illness or injury of informal supports, or significant and unexpected deterioration of disability-related functional capacity.
- Appropriate disability supports are not in place and are re-entering the community after a long-term residence or hospital stay (specific release date not required):
    - A person with a **newly acquired, significant disability**, such as spinal cord injury, being discharged from hospital
    - A **younger person living in residential aged care**
    - A person being **discharged from an inpatient mental health facility**
    - A person due to be **released from correctional facility**.

How do we weigh evidence of disability?

We understand that you may have evidence of your disability from different health professionals at different times. When we're deciding if you're eligible for the NDIS, we look at:

- how old your evidence is
- who provided your evidence.

We weigh evidence based on what we consider best practice, or highest quality. We consider this evidence most strongly when we make a decision.

What type of evidence should you provide?

We need evidence to help us consider if you meet the disability or early intervention requirements.

For the disability requirements, we need evidence to confirm your permanent impairment and evidence about how this impacts your functional capacity.

For the early intervention requirements, we need evidence to confirm your permanent impairment and evidence that confirms you need early intervention.

It's important to understand the type of evidence that you are providing us. You may have evidence from a doctor or specialist confirming your **permanent impairment** or you may have evidence from an allied health professional or other medical professional that tells us about **impacts to your functional capacity** (your ability to do daily life activities). These are different types of evidence which will often be provided by different health professionals based on their qualifications.

How old should your evidence be?

How old should your evidence be to confirm your permanent impairment?

We need evidence from your doctor or specialist to confirm your permanent impairment. You can give us evidence confirming this **from any age**. However, evidence about how your impairment impacts your functional capacity should be from the last 12 months.

How old should your evidence be to confirm your functional capacity?

Generally, we need evidence about how your impairment impacts your functional capacity from **the last 12 months**. This is because your functional capacity may change over time, even if your impairment doesn't. It's important that we have evidence of your current circumstances to ensure we understand your support needs.

If you give us more than one type of evidence, we might weigh the newer evidence over the older evidence. If you give us older evidence, we will generally give this less weighting when we make our decision. In these cases, we will generally ask for more information. If this is not provided, we may decide you are not eligible for the NDIS.

How old should your evidence be to confirm you need early intervention?

We need evidence from your doctor or specialist to confirm your permanent impairment and that you need early intervention.

Generally, we need evidence about that confirms you need early intervention from **the last 12 months**. This is because your functional capacity may change over time – even if your impairment does not. It's important that we have evidence of your current circumstances to ensure we understand your support needs.

Who should provide evidence?

We generally prefer evidence that comes from a treating professional who:

- is the most **appropriately qualified** person to provide evidence of your primary disability
- has treated you for a significant period of time (at least six months)
- is registered to practise in Australia or New Zealand
- provides disability evidence (such as a medical report) that is original, genuine and specific to you.

Depending on your situation, you might get your evidence of **permanent impairment** from a different treating professional than your **evidence of functional capacity**.

If you need help to get your evidence together, your local area coordinator or early childhood partner can help you.

Who should provide evidence of your permanent impairment?

We generally prefer evidence from your doctor or specialist to confirm your permanent impairment.

Examples of common doctors or specialists include:

- General Practitioner (GP)
- Paediatrician
- Orthopaedic surgeon
- Neurologist

- Psychiatrist.

Who should provide evidence of your functional capacity?

We generally prefer evidence from a doctor, specialist, allied health or other medical professional for confirm how your permanent impairment impacts your functional capacity.

In addition to doctors and specialists, examples of common allied health or other medical professionals include:

- Occupational Therapist
- Speech Pathologist (Therapist)
- Psychologist
- Physiotherapist.

Who should provide evidence that you need for early intervention?

We generally prefer evidence from your doctor or specialist to confirm your permanent impairment.

Whereas a doctor, specialist, allied health or other medical professional can give us evidence to confirm you need early intervention.

In addition to doctors and specialists, examples of common allied health or other medical professionals include:

- Occupational Therapist
- Speech Pathologist (Therapist)
- Psychologist
- Physiotherapist.

Health professionals registered to practise in Australia and New Zealand

We strongly prefer evidence of your disability to come from a registered Australian or New Zealand health professional. Most Australian health professionals are registered with the Australian Health Practitioner Regulation Agency (AHPRA).

We will still consider evidence from non-Australian or New Zealand health professionals, or unregistered health professionals. However, this evidence will be given less weight.

If we cannot confirm the registration of your health professional, we will ask you (and your health professional) for more information in the first instance. If we still cannot confirm their registration, we will likely decide that you are not eligible for the NDIS.

Reference list

[1] NDIS Act s 24(1)(e).
[2] NDIS Act ss 22-23.
[3] NDIS Act s 24.
[4] NDIS Act s 25.
[5] NDIS Act s 28(1).
[6] NDIS Act s 22.
[7] NDIS Act s 23(1)(b)(i).
[8] NDIS Act s 23(1)(b)(ii).
[9] NDIS Act s 23(1)(b)(iii).
[10] NDIS Act s 23(1)(a).
[11] NDIS Act s 23(2).
[12] NDIS Act s 23(2)(f).
[13] NDIS Act s 23(2)(a).
[14] NDIS Act s 23(2)(b).
[15] NDIS Act s 23(2)(c).
[16] NDIS Act s 23(2)(d).
[17] NDIS Act s 23(2)(e).
[18] NDIS Act s 24.
[19] NDIS Act ss 24(1)(a); 25(1)(a).
[20] NDIS Act ss 24(1)(a); 25(1)(a)(ii).
[21] NDIS Act ss 24(3)
[22] NDIS (Becoming a Participant) Rules rr 5.4, 6.4.
[23] NDIS (Becoming a Participant) Rules rr 5.6, 6.6.
[24] NDIS (Becoming a Participant) Rules rr 5.5, 6.5.
[25] NDIS Act s 25(3). NDIS (Becoming a Participant) Rules r 6.8.
[26] NDIS Act s 24(1)(d).
[27] NDIS Act s 24(1)(e).
[28] NDIS Act s24(3)
[29] NDIS Act s 24(2).
[30] NDIS Act ss 25(1)(a)(i)-(ii).
[31] NDIS Act s 25(1)(a)(i).
[32] NDIS Act ss 25(1)(a)(i)-(ii).
[33] NDIS Act s 25(1)(a)(ii).
[34] NDIS Act s 25(1)(b).
[35] NDIS Act s 25(1)(c).
[36] NDIS (Becoming a Participant) Rules r 6.9.
[37] NDIS Act s 25(1)(d)
[38] NDIS Act s 25.
[39] NDIS Act ss 9 (definition of ‘developmental delay’), 21(1)(c), 25(1)(a)(iii).
[40] NDIS Act s 23(1)(a).
[41] NDIS Act s 23(1)(b).
[42] NDIS Act ss 9 (definition of ‘developmental delay’), 25(1)(a)(iii).
[43] NDIS Act ss 9 (definition of ‘developmental delay’), 25(1)(a)(iii).
[44] NDIS Act s 9 (definition of ‘developmental delay’ para (a)).
[45] NDIS Act s 9 (definition of ‘developmental delay’ para (b)).
[46] NDIS Act s 9 (definition of ‘developmental delay’ para (c)).
[47] NDIS Act s 25(1)(d); NDIS (Becoming a Participant) Rules rr 6.1, 8.4.
[48] NDIS Act s 19(1)(a).
[49] NDIS Act s 19(1)(b).
[50] NDIS Act s 19(1)(c).

[51] NDIS Act s 197(1).
[52] NDIS Act s 74(1)(a).
[53] NDIS Act s 74(1)(b).
[54] NDIS Act s 20.
[55] NDIS Act s 26(2)(d).
[56] NDIS Act s 26.
[57] NDIS Act s 26(3)
[58] NDIS Act s 26(2).
[59] NDIS Act s 21(3).
[60] NDIS Act s 100(5)(b).
[61] NDIS Act s 28(1).
[62] NDIS Act s 28(2).
[63] NDIS Act s 7(2).
[64] NDIS Act s 100(2).
[65] NDIS Act s 100(2)
[66] NDIS Act s 19(2).
[67] NDIS Act s 19(2)(c).
[68] NDIS Act s 102.
[69] NDIS Act s 19(2)(d).

Make an access decision - post legislation changes

SGP KP Publishing

Exported on 2024-10-08 15:18:45

Table of Contents

This article provides guidance for an access delegate (assessor) to:

- check legislative timeframes
- complete disability evidence
- request further information
- approve or override the streaming case
- record access decision
- submit decision for quality check
- complete access decision correspondence
- notify early childhood partner of access not met decision (developmental delay only)
- check disabilities tab (access met only)
- assign plan approval case (Motor neurone disease only).

The legislation you need to use automatically populates in the access decision case based on what is selected in the access request case.

Go to article Complete pre-assessment of access decision to decide which version of the legislation and article you need to use to make an access decision.

Use this article if you need to use the **post-legislation changes** version of the legislation to make an access decision.

1 Recent updates

3 October 2024
Updated guidance to:

- reflect legislation changes from 3 October 2024
- Update title from Make access decision to make it clear to use this article when making a decision using the post-legislation changes version of the legislation.
- Remove step to consider if the person is from a defined program as defined programs no longer apply.
- To be eligible under the disability requirements, there must be evidence the person requires NDIS supports for their lifetime. NDIS supports are the services, items and equipment that can be funded by the NDIS.
- To be eligible under the early intervention requirements, there must be evidence that supports that are likely to benefit the person will be NDIS supports.
- If the age and residence requirements are met, you must consider both the disability and early intervention requirements. The person may meet both, rather than only one or the other.
- If eligible, you must select the impairments that met the requirements for disability, early intervention requirements or both. Impairments include intellectual, cognitive, neurological, sensory, physical, or impairments to which a psychological disability is attributable. Or you can select developmental delay, and you do not need to select impairments.
- New step to advise the justice liaison officer (JLO) when requesting more evidence (if relevant)
- New step to notify early childhood partner of access not met decision (developmental delay only)
- New step to assign plan the approval case (Motor neurone disease only).

2 Before you start

You have:

- completed the pre-assessment and checked you need to use the **post-legislation changes** version of the legislation to make an access decision using article Complete pre-assessment for an access decision
- checked there is evidence of identity or an internal note to explain why it can't be provided
- checked the application is complete with evidence of consent to apply, age, residence and disability
- read article Check eligibility – age and residence requirements
- read article Understand disability requirements
- read article Understand early intervention requirements.

3 Check legislative timeframes

You have **21 days** to make a decision or request further information.

When a request for further information has been made, you have **14 days** from the date that the last information or report was received to make a decision.

1. Use article Check decision – legislative timeframes to check if your decision or request for further information will be outside legislative timeframes. If:
    - inside legislative timeframes: go to section **Complete disability evidence**.
    - outside legislative timeframes: use article Check decision – legislative timeframes to record an internal note. Then go to section **Complete disability evidence**.

4 Complete disability evidence

Links to evidence documents in the access request case will display here.

1. At **Have you verified the Evidence of Disability?**, select **Yes**. This is based on section **Check evidence of disability** in article Complete pre-assessment of access decision.
2. At **Select assessed Impairment**, enter text into the field and a list will display. Select relevant option.
3. Make a note of the assessed impairment name and ICD code. You will need this for section **Decision Main Criteria**.
4. At **What evidence was used to confirm the Impairment?**, select the appropriate option.

Note: **Defined Program** no longer populates the access decision.

5. At **Does the applicant's impairment belong to List A, B or D?**, select the appropriate option.

Note: This no longer populates the access decision.

6. For more information about list conditions, go to articles:
    - Check eligibility – List A condition
    - Check eligibility – List B condition
    - Check eligibility – List D condition.
7. Select **Add** next to **Impairment Assessment** to record additional impairments. If an:
    - **access met** decision, add all impairments that meet eligibility requirements
    - **access not met** decision, add all reported impairments.

5 Request further information

1. Decide if you need more information to make an access decision. Only request more information if:
    - the application is complete
    - you need more evidence after receiving the applicant's disability evidence.
2. If the applicant is a child with developmental delay turning 6 within the next 90 days, ask your team leader before requesting more information.

In this situation, it may be more appropriate to make an access not met decision. For an access request, the child must be younger than 6 on the day the access decision is made. For an internal review, the child must have been younger than 6 at the time of the access not met decision. This reduces the risk of the child turning 6 before an access decision is made.

3. At **Do you require further information from the applicant?**, select **Yes** or **No**. If:
    - **No**, select **Next**, then go to section **Approve or override streaming case**.
    - **Yes**, continue to next step.
4. At **Enter the Required Information below**, copy relevant templates from Templates for requesting further information to make a decision – Post-legislation changes.
5. Select **Next**.
6. Select one or more of the criteria for the requested information from the provided tick boxes.
7. Select **Next**.
8. Select **Done**.
9. Select **Next**.
10. At **Do you want to preview the document before sending it?**, select **Yes**.
11. Complete **Select a Recipient for preview** to preview the letter.
12. If correct, select **Next**. If incorrect, select **Previous** to update the fields that populate the letter.
13. Select **Submit Correspondence**.
14. Select **Next**. A letter will automatically be sent to the applicant or their authorised representative to request further information. The access decision case is automatically closed.

5.1 Check correspondence

1. From the **Person Account** case, select the **Documents** tab.
2. Check for **Further Information Requested** letter. **Category** will be **Outbound Correspondence**. If:
    - **Yes**, if there is a justice liaison officer, go to section **Notify the justice liaison officer (JL) (if relevant)**. If not, go to section **Next steps**.
    - **No**, go to next step.
3. Submit a Report a defect with PACE, my NDIS Provider or Participant Portal, or my NDIS App ticket
4. Share the ticket with your team leader and assistant director.

5. If there is a justice liaison officer, go to section **Notify the justice liaison officer (JL) (if relevant)**. If not, go to section **Next steps**.

5.2 Notify the justice liaison officer (JLO) (if relevant)

5.2.1 Create the enquiry case

1. Create an **Enquiry** case and reassign to the **MyNDIS Contact** using article Create an enquiry case.
2. At **Requested By**, select **General Enquiry Only**.
3. At **Case Origin**, select **Internal**.
4. At **Enquiry Type**, select **Access**.
5. At **Category**, select **Access Request**.
6. At **Sub Category**, select **Escalation Enquiry**.
7. In **Enquiry Notes**, use the following template:

Further information requested to support **<Applicant's name>** NDIS application.

More information is needed about <permanency/functional capacity/lifetime supports/future support needs/whether the NDIS is the most appropriate service> of <Impairment/Impairments>. <Reason this information is being requested>.

Refer to Request for More Evidence letter in the person account for more information.

Support the applicant to provide more evidence by **<Date>** (90 days).

8. At **Enquiry Outcome** select **Keep enquiry open - Do not re-assign**. This will allow you to assign the case to the JLO later with an email notification.

5.2.2 Assign the enquiry case

1. From the **Enquiry** case, select the **Change Owner** icon next to the current **Case Owner**.
2. At the **Search Users** free text field, search for the **MyNDIS Contact**.
3. Select the tick box next to **Send notification email**.
4. Select **Change Owner**.
5. Go to section **Next Steps**.

6 Approve or override the streaming case

The streaming case is critical to make sure the Typical Support Package (TSP) is generated for eligible applicants, and they're assigned to the correct team. This is important to make sure they receive the support level they need to engage with the NDIS.

Generally, the streaming case is completed by a local area coordinator, early childhood partner or planner and the case routes to an access delegate for approval.

1. Check for a streaming case. A streaming case **must** be completed and approved **before** completing the access decision case. If:
    - **Yes**, continue to next step.
    - **No**, create a streaming case. Go to article Complete a streaming case (Streaming and Restreaming). Then continue to next step.
2. Go to article Approve or override a streaming case (Streaming and Restreaming) to approve or override the streaming case.

7 Record access decision

7.1 Age and Residency Sub Criterion

1. Use article Check eligibility – age and residence requirements to help you decide if the age and residence requirements are met.

7.1.1 Age Criterion 1: (Section 22(1)(a))

1. At **Is the applicant under the age of 65 years old?** select **Yes** or **No**. If
    - **Yes**, go to section **Residency Criterion 1: (Section 23(1)(a))** to consider the next criteria.
    - **No**, continue to next step.
2. Select **Next**.
3. Select **N/A** for all early intervention and disability requirements. You don't need to consider the remaining eligibility requirements.
4. Go to section **Evidence Used for Decisions**.

7.1.2 Residency Criterion 1: (Section 23(1)(a))

1. At **Is the applicant currently living in Australia?** select **Yes** or **No**. If:
    - **Yes**, go to section **Residency Criterion 2: (Section 23(1)(b))** to consider the next criteria.
    - **No**, continue to next step.
2. Select **Next**.
3. Select **N/A** for all early intervention and disability requirements. You don't need to consider the remaining eligibility requirements.
4. Go to section **Evidence Used for Decisions**.

7.1.3 Residency Criterion 2: (Section 23(1)(b))

1. At **Is the applicant an Australian Citizen or a Current Eligible Visa Holder?** select **Yes** or **No**. If:
    - **Yes**, select **Next** and go to section **Early Intervention Sub Criteria** to consider the next criteria.
    - **No**, continue to next step.
2. At **Why does the applicant not meet the sub criteria?**, select the relevant option.
3. Select **Next**.
4. Select **N/A** for all early intervention and disability requirements. You don't need to consider the remaining eligibility requirements.
5. Go to section **Evidence Used for Decisions**.
6. Select **Next**.

7.2 Early Intervention Sub Criteria

1. Use article Understand early intervention requirements to help you decide if the early intervention requirements are met.
2. Consider if the applicant has an impairment on List B or D. This no longer populates in the access decision. You need to select the relevant criteria manually. If:
    - **list D** and the child is younger than 7, select **Yes** to all early intervention requirements and select **Next**.
    - **list B**, select **Yes** to **Early Intervention Criterion 1: (Section 25(1)(a))** and **Early Intervention Criterion 2: (Section 25(1)(b))**. Then consider the remaining early intervention requirements based on the evidence.
3. If none of the above, consider each early intervention requirement based on the evidence. All early intervention requirements must be answered.
4. If you select **No** for any criteria, select the reason the early intervention requirement is not met.

7.2.1 Early Intervention Criterion 1: (Section 25(1)(a))

1. At **Is there one or more identified intellectual, cognitive, neurological, sensory or physical impairments that are, or are likely to be permanent (section 25(1)(a)(i) or is there one or more identified impairments that are attributable to a psychiatric condition that are, or are likely to be, permanent (section 25(1)(a)(ii))?**:
    - Select **Yes** or **No**.
    - If **No** and the applicant is younger than 6, there will be questions to consider if the child has a developmental delay that meets the early intervention requirements.

7.2.2 Early Intervention Criterion 2: (Section 25(1)(b))

1. At **Is the provision of early intervention supports likely to benefit the person by reducing the person's future needs for supports in relation to disability?**
1.
    - Select **Yes** or **No**.

7.2.3 Early Intervention Criterion 3: (Section 25(1)(c))

1. At **Is the provision of early intervention supports likely to improve, or reduce deterioration, of functional capacity or strengthen sustainability of informal supports?**:
    - Select **Yes** or **No**.

7.2.4 Early Intervention Criterion 4: (Section 25 (d))

1. At **Are any early intervention supports that would be likely to benefit the person as mentioned in paragraphs 25(1)(b) and (c) NDIS supports?**:
    - Select **Yes** or **No**.
2. Select **Next**.

7.3 Disability Sub Criterion

1. Use article Understand disability requirements to help you decide if the disability requirements are met.
2. Consider if the applicant has an impairment on List A or B. This no longer populates in the access decision. You need to select the relevant criteria manually. If:
    - **list A**, the disability requirements are met. Select **Yes** to all disability requirements and select **Next**.
    - **list B**, select **Yes** to **Disability Criterion 1: (Section 24 (1)(a))** and **Disability Criterion 2: (Section 24 (1)(b))**. Then consider the remaining disability requirements based on the evidence.
3. If none of the above, consider each disability requirement based on the evidence. All disability requirements must be answered.
4. If you select **No** for any criteria, select the reason the disability requirement is not met.

7.3.1 Disability Criterion 1: (Section 24 (1)(a))

1. At **Are the impairment(s) attributable to one or more intellectual, cognitive, neurological, sensory or physical impairments or to psychiatric condition(s)?**:
    - Select **Yes** or **No**.

7.3.2 Disability Criterion 2: (Section 24 (1)(b))

1. At **Are the impairment(s) permanent, or are they likely to be permanent?**:
    - Select **Yes** or **No**.

7.3.3 Disability Criterion 3: (Section 24 (1)(c))

1. At **Does the impairment(s) result in substantially reduced functional capacity?**:
    - Select **Yes** or **No**.

7.3.4 Disability Criterion 4: (Section 24 (1)(d))

1. At **Does the impairment affect the person's capacity for social and economic participation?**:
    - Select **Yes** or **No**.

7.3.5 Disability Criterion 5: (Section 24 (1)(e))

1. At **Is the person likely to require support under the NDIS for their lifetime**?:
    - Select **Yes** or **No**.
2. Select **Next**.

7.4 Evidence Used for Decisions (access not met only)

1. For any access criteria the person does not meet, record:
    - **Evidence type**
    - **Evidence** (if required) – start to enter the linked evidence name, then select relevant document.

- **Explanation** – record Criteria not met – see linked evidence.

2. Select **Next**.

7.5 Decision Main Criteria

1. These questions will automatically be completed with **Access Met**, **Access Not Met** or **N/A**:
   - **Does the person meet Age Criteria**
   - **Does the person meet Residency Criteria**
   - **Does the person meet Disability Criteria**
   - **Does the person meet Early Intervention Criteria**
   - **Overall decision**.

   Note: An applicant may meet both the disability and early intervention requirements.
2. If **Overall decision** is:
   - **Access Not Met**, go to step 9.
   - **Access Met**, go to next step.
3. Open the Impairment Categories Guide to complete the next steps.
4. Search the **ICD 10 Code** or **Condition** name for **all** impairments that meets the requirements for disability, early intervention, or both.
5. Note the **Required impairment category** column.
6. Consider if any of the **Optional impairment categories** in the guide apply based on the evidence provided:
   - **Intellectual** – such as how they speak and listen, read and write, solve problems, and process and remember information.
   - **Cognitive** – such as how they think, learn new things, use judgment to make decisions, and pay attention.
   - **Neurological** – such as how their body functions.
   - **Sensory** – such as how they see or hear.
   - **Physical** – such as the ability to move parts of their body.
   - **Psychosocial** - This means they have reduced capacity to do daily life activities and tasks due to your mental health.
7. If the early intervention requirements are met, at **Which of the following meet the eligibility criteria for access?**, select options that apply for **all eligible** impairments that met the early intervention requirements:
   - **Intellectual**
   - **Cognitive**
   - **Neurological**
   - **Sensory**
   - **Physical**
   - **One or more impairments to which a psychological disability is attributable**
   - **Developmental Delay**.

8. If the disability requirements are met, at **Which of the following meet the eligibility criteria for access?**, select options that apply for **all eligible** impairments that met the disability requirements:
    - **Intellectual**
    - **Cognitive**
    - **Neurological**
    - **Sensory**
    - **Physical**
    - **One or more impairments to which a psychological disability is attributable**.

    Note: This is important as we only fund NDIS supports for eligible impairments that meet the requirements for disability, early intervention or both.
9. If the decision isn't correct: Select **Back**.
    - Re-check and update **previous** criteria as per above steps.
10. If the decision is correct:
    - **Select Save for later**.

7.5.1 If access met for applicant with chronic health condition

You **must** request technical advice from the Technical Advisory and Practice Improvement branch (TAPIB).

1. First send a technical support request to SEB.QUALITY to request Quality Development Officer (QDO) feedback.
2. If the QDO agrees, request technical advice from TAPIB using the following articles:
    - Create a technical advice case
    - Complete the risk matrix for a technical advice case
    - Review and action returned technical advice case.

    Note: You can review relevant article in the TAPIB Digest to help you refine your request.

If access met for applicant younger than 25 with primary psychosocial disability

You **must** receive endorsement from your team leader or assistant director.

- First send a technical support request (Review Request: Under 25 Psychosocial) to SEB.QUALITY to request QDO feedback.
- If the QDO agrees, they will email your team leader or assistant director to request endorsement.

7.5.2 If access met and the applicant resides in hospital

You **must** ask for QDO feedback.

1. Send a technical support request to SEB.QUALITY to request QDO feedback. They will decide if TAPIB advice is required.

8 Submit decision for quality check

8.1 Submit proposed decision for potential quality check

1. Complete the Access Assessor Outcome Form to submit the proposed access decision for a potential quality check. An email will advise you if the proposed decision is sent for quality check. If:
    - **not sent** for quality **check**, go to section **Approve access decision**
    - **sent** for quality check, go to section **Review quality check feedback**.

8.2 Review quality check feedback

1. Review quality check feedback when received by email. If:
    - **no adjustment** required and **you** agree with the feedback, go to section **Approve access decision**
    - **adjustment required** and you **agree** with the feedback, continue to next step.
2. Re-assess and amend access decision in line with the feedback.
3. From the **Decision Main Criteria** screen, select **Save for later**.
4. Go to section above **Submit proposed decision for potential quality check** to re-submit for a potential quality check.

Note: For technical support to understand the legislative criteria, please send a technical support request to SEB.QUALITY.
If you **don't agree** with the feedback, contact your team leader about the reconsideration process.

8.3 Approve access decision

1. Return to **Decision Main Criteria** view.
2. Select **Approve** to submit decision.
3. Go to section **Complete access decision correspondence**.

9 Complete access decision correspondence

9.1 Automated Access Met letter

1. Check the correspondence preferences of the applicant are correct. Select **Next**.
2. At **Do you want to preview the document before sending it?** select **Yes** or **No**.
3. If you select **Yes**, you need to **Select a Recipient for preview?**. Select the applicant you wish to preview the correspondence for and select **Next**.
4. A letter preview will generate.
5. If correct, select **Next**. If incorrect, select **Previous** to update the fields that populate the letter.
6. Select **Submit Correspondence**. An automatic access met decision letter will be sent to the person or authorised representative.
7. At **Correspondence Summary** select **Next**.
8. Select **Done** to close the access decision case.

9.1.1 Check correspondence

1. In the **Access Decision** case, select the **Documents** tab.
2. Check for **Outcome of Application – Eligible letter**. **Category** is **Outbound Correspondence**. If:
    - **Yes**, go to section **Check disabilities tab**
    - **No**, go to next step.
3. Submit a Report a defect with PACE, my NDIS Provider or Participant Portal, or my NDIS App ticket,
4. Share the ticket with your team leader and assistant director.
5. Go to section **Next steps**.

9.2 Manual Access Not Met letter

PACE **won't** generate an automatic **Access Not Met letter**. You need to complete and send a manual letter.

1. Select **Done** to close the access decision case.
2. Complete the manual access not met letter using these articles:
    - Letter –Access not met decision
    - Template – Access not met decision – post legislation changes.

    Note: Only the letter text is required from this resource. **Interactions are not required**.
3. Use article Send a manual letter to complete this process.

10 Notify early childhood partner of access not met decision (developmental delay only)

If the child is younger than 6 with a developmental delay, you need to notify the early childhood partner of the access not met decision.

1. Create an **Enquiry** case and reassign to the **MyNDIS Contact** using article Create an enquiry case.
2. At **Requested By**, select **General Enquiry Only**.
3. At **Case Origin**, select **Internal**.
4. At **Enquiry Type**, select **Partner Supported Access**.
5. At **Category**, select **General Information**.
6. In **Enquiry Notes**, use the following template:
7. Access not met decision for developmental delay made on **<Date>**.

Evidence provided does not support the developmental delay criteria because <provide a summary of the justification for the decision e.g. the applicant does not have a significantly lower ability to do everyday activities, when compared to children of the same age>.

<If the child is turning 6 within 90 days: The child is turning 6 within 90 days. It may be more appropriate to support the applicant with an internal review, rather than a new access request. For an access request, the child must be younger than 6 on the day the access decision is made. For an internal review, the child must have been younger than 6 at the time of the access not met decision. This reduces the risk of the child turning 6 before an access decision is made.**>**
Access request outcome will be communicated to the applicant's authorised representative – refer to case activity for updates.

Please support applicant with next steps such as early connections.

8. At **Enquiry Outcome** select **Re-assign** this enquiry to another user.
9. At **Case Re-assignment Reason** select **Referral to Partner**.
10. At **Select User or Queue** select **User**.
11. At **Case Owner** enter the applicant's **MyNDIS Contact**.

11 Check disabilities tab (access met only)

The **Disabilities** tab **must** include all impairments that meet the requirements for disability, early intervention or both as this affects planning.

There may be impairments that you need to add or remove.

1. From the **Person Account**, select **My Profile**.
2. Select the **Disabilities** tab.
3. Review **Active status** to see what impairments are current.
4. If required, use article Update a person account to:
    - add **impairments** that meet the eligibility requirements
    - **remove** any impairments that don't meet the eligibility requirements by adding an **End Date**.

12 Assign plan approval case (Motor neurone disease only)

If you are in the Priority Health Access Team and have made an access met decision for an applicant with Motor neurone disease (also known as Lou Gehrig's disease or Amyotrophic lateral sclerosis), assign the **Plan Approval** case to the **Aged Care Referral Routing Queue**.

In all other situations, a plan approval case will automatically route to the relevant queue.

1. In the **Plan Approval** case, select the **Change Owner** icon next to the current **Case Owner**.
2. Select the **Users** icon (on the left of the search bar) then select **Queues**.
3. At the **Search Users** free text field, search and select **Aged Care Referral Routing Queue**.
4. Select **Change Owner**.

13 Next steps

1. After you complete the access decision case, an automatic contact applicant task will create. Contact the applicant or their authorised representative. If:
    - **Access met** or **access not met**, go to article Contact to advise outcome of access decision.
    - **Further information requested**, go to article Contact to request further information for access decision.
2. For an access met decision, a plan approval case will automatically route to the relevant queue for a planner to develop their first participant NDIS plan.

The contents of this document are OFFICIAL.

Impairment categories guide

Guidance in this document is not approved for use unless you view it in PACE.

This article provides guidance for an access delegate to:

- understand what impairment categories to select for a person that meets the eligibility requirements.

Recent updates

4 October 2024

New guidance to:

- reflect legislation changes from 3 October 2024
- decide what impairment categories to select for a person that meets the eligibility requirements.

Impairments categories guide

Impairments are a loss of or damage to a body's function. When we assess an impairment to meet access, we look at:

- the body's function
- the body's structure
- how they think and learn

The following list provides you with information on:

- the condition
- the ICD 10 Code
- the **required** impairment category for access assessors to select
- **optional** impairment categories to select as relevant, based on evidence provided with access request.

Conditions

Autism (ASD)

1. **Autism** – includes Rett and Asperger Syndrome

   ICD 10 Codes: F84.0, F84.2, F84.5

   Required impairment category: Neurological

   Optional impairment categories: Intellectual, cognitive, physical, psychosocial

Acquired brain injury

1. **Glioblastoma**

   ICD 10 Code: G71.9

   Required impairment category: Neurological

   Optional impairment categories: Cognitive, physical, psychosocial

2. **Hypoxic brain injury**

   ICD 10 Code: 93.1

   Required impairment category: Neurological

   Optional impairment categories: Physical, psychosocial

3. **Traumatic brain injury** – also called head injury and acquired brain damage

   ICD 10 Code: T90

   Required impairment category: Neurological

   Optional impairment categories: Cognitive, physical, psychosocial

Intellectual disability

1. **Mild intellectual disability**

   ICD 10 Codes: F70

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive

2. **Moderate intellectual disability**

   ICD 10 Codes: F71

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive, physical, psychosocial

3. **Severe intellectual disability**

ICD 10 Codes: F72

Required impairment category: Intellectual

Optional impairment categories: Cognitive, physical, psychosocial

4. **Profound intellectual disability**

   ICD 10 Codes: F73

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive, physical, psychosocial

5. **Unspecified intellectual disability**

   ICD 10 Codes: F79

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive, sensory, physical, psychosocial

6. **Pervasive developmental disorder**

   ICD 10 Codes: F84.8

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive, sensory, psychosocial

7. **Microcephaly**

   ICD 10 Codes: Q02

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive, neurological, sensory, physical

8. **Other congenital brain conditions –** for example, tuberous sclerosis

   ICD 10 Codes: Q04

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive, neurological, physical

9. **Spina bifida**

   ICD 10 Codes: Q05

   Required impairment category: Physical

   Optional impairment categories: Intellectual, cognitive, sensory, neurological

10. **Foetal alcohol syndrome**

    ICD 10 Codes: Q86.0

Required impairment category: Neurological

Optional impairment categories: Intellectual, cognitive, sensory, physical, psychosocial

11. **Foetal alcohol spectrum disorder (FASD)**

    ICD 10 Codes: Q86.0D

    Required impairment category: Neurological

    Optional impairment categories: Intellectual, cognitive, sensory, physical, psychosocial

12. **Cornelia de Lange syndrome**

    ICD 10 Codes: Q87.1

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, neurological, sensory, physical, psychosocial

13. **Prader Willi syndrome**

    ICD 10 Codes: Q87.1

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, neurological, physical, psychosocial

14. **Coffin-Lowry syndrome**

    ICD 10 Codes: Q87.8

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, neurological, sensory, physical, psychosocial

15. **Other congenital conditions (causing intellectual disability)**

    ICD 10 Codes: Q89

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, neurological, sensory, physical, psychosocial

16. **Edwards syndrome**

    ICD 10 Codes: Q91

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, neurological, sensory, physical, psychosocial

17. **Patau syndrome**

    ICD 10 Codes: Q91

    Required impairment category: Intellectual

Optional impairment categories: Cognitive, neurological, sensory, physical, psychosocial

18. **Cri du Chat syndrome**

    ICD 10 Codes: Q93.4

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, sensory, physical

19. **Angelman syndrome**

    ICD 10 Codes: Q93.5

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, sensory, physical

20. **Other chromosomal syndromes (including Kabuki & Williams syndromes)**

    ICD 10 Codes: Q99

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, neurological, sensory, physical, psychosocial

21. **Fragile X syndrome**

    ICD 10 Codes: Q99.2

    Required impairment category: Intellectual

    Optional impairment categories: Cognitive, neurological, sensory, physical, psychosocial

Cerebral Palsy

1. **Cerebral palsy**

   ICD 10 Codes: G80

   Required impairment category: Physical

   Optional impairment categories: Intellectual, cognitive, neurological, sensory

Down Syndrome

1. **Down syndrome**

   ICD 10 Codes: Q90

   Required impairment category: Intellectual

   Optional impairment categories: Cognitive, physical

Hearing Impairment

1. **Hearing loss**

   ICD 10 Codes: H90

   Required impairment category: Sensory

   Optional impairment categories: Cognitive

2. **Congenital hearing condition**

   ICD 10 Codes: Q16.9

   Required impairment category: Sensory

   Optional impairment categories: Cognitive

Visual Impairment

1. **Albinism**

   ICD 10 Codes: E70.3

   Required impairment category: Sensory

   Optional impairment categories: Not applicable

2. **Visual impairment (including blindness)**

   ICD 10 Codes: H54

   Required impairment category: Sensory

   Optional impairment categories: Not applicable

3. **Congenital eye conditions**

   ICD 10 Codes: Q15.9

   Required impairment category: Sensory

   Optional impairment categories: Not applicable

Other Sensory - Speech

1. **Other sensory - speech**

   ICD 10 Codes: R47

   Required impairment category: Sensory

   Optional impairment categories: Physical

Multiple Sclerosis

1. **Multiple Sclerosis**

ICD 10 Codes: G35

Required impairment category: Neurological

Optional impairment categories: Cognitive, physical, psychosocial

Other Neurological

1. **Alzheimer's disease**

   ICD 10 Codes: F00

   Required impairment category: Cognitive

   Optional impairment categories: Neurological, physical, psychosocial

2. **Unspecified dementia**

   ICD 10 Codes: F03

   Required impairment category: Cognitive

   Optional impairment categories: Neurological, physical, psychosocial

3. **Huntington disease**

   ICD 10 Codes: G10

   Required impairment category: Physical

   Optional impairment categories: Cognitive, neurological, psychosocial

4. **Motor neurone disease**

   ICD 10 Codes: G12.2

   Required impairment category: Physical

   Optional impairment categories: Cognitive, neurological, sensory, psychosocial

5. **Parkinson's disease**

   ICD 10 Codes: G20

   Required impairment category: Neurological

   Optional impairment categories: Cognitive, physical, psychosocial

6. **Epilepsy – Mandatory TAPIB**

   ICD 10 Codes: G40

   Required impairment category: Neurological

   Optional impairment categories: Intellectual, cognitive, psychosocial

7. **Muscular dystrophy**

ICD 10 Codes: G71.0

Required impairment category: Physical

Optional impairment categories: Neurological

8. **Other Neurological – List A and List C**

   ICD 10 Codes: G99

   Required impairment category: Neurological

   Optional impairment categories: Cognitive, sensory, physical

Stroke

1. **Stroke**

   ICD 10 Codes: I69

   Required impairment category: Physical

   Optional impairment categories: Cognitive, neurological, sensory, psychosocial

Other Physical

1. **Rheumatoid arthritis**

   ICD 10 Codes: M05

   Required impairment category: Physical

   Optional impairment categories: Not applicable

2. **Other arthritis – mandatory TAPIB**

   ICD 10 Codes: M12

   Required impairment category: Physical

   Optional impairment categories: Not applicable

3. **Other physical**

   ICD 10 Codes: M95

   Required impairment category: Physical

   Optional impairment categories: Psychosocial

4. **Multiple traumatic amputations**

   ICD 10 Codes: T05

   Required impairment category: Physical

Optional impairment categories: Psychosocial

5. **Myopathy**

   ICD 10 Codes: G72.9

   Required impairment category: Physical

   Optional impairment categories: Not applicable

Psychosocial disability

1. **Schizophrenia**

   ICD 10 Codes: F20

   Required impairment category: Psychosocial

   Optional impairment categories: Cognitive

2. **Schizoaffective disorder**

   ICD 10 Codes: F25.9

   Required impairment category: Psychosocial

   Optional impairment categories: Cognitive

3. **Bipolar affective disorder**

   ICD 10 Codes: F31

   Required impairment category: Psychosocial

   Optional impairment categories: Cognitive

4. **Major depressive illness**

   ICD 10 Codes: F32

   Required impairment category: Psychosocial

   Optional impairment categories: Cognitive

5. **Other anxiety disorders**

   ICD 10 Codes: F41

   Required impairment category: Psychosocial

   Optional impairment categories: Cognitive

6. **Obsessive-compulsive disorder**

   ICD 10 Codes: F42

   Required impairment category: Psychosocial

Optional impairment categories: Not applicable

7. **Post traumatic stress disorder**

   ICD 10 Codes: F43

   Required impairment category: Psychosocial

   Optional impairment categories: Cognitive

8. **Borderline personality disorder**

   ICD 10 Codes: F60.3

   Required impairment category: Psychosocial

   Optional impairment categories: Not applicable

9. **Tourette syndrome**

   ICD 10 Codes: F95.2

   Required impairment category: Neurological

   Optional impairment categories: Cognitive, physical, psychosocial

10. **Other psychosocial disorders**

    ICD 10 Codes: F99

    Required impairment category: Psychosocial

    Optional impairment categories: Cognitive

11. **Anorexia**

    ICD 10 Codes: R63

    Required impairment category: Psychosocial

    Optional impairment categories: Cognitive, physical

Spinal Cord Injury

1. **Malignant neoplasm of spinal cord complete and incomplete**

   ICD 10 Codes: C72.5, C72.7

   Required impairment category: Physical

   Optional impairment categories: Neurological, sensory

2. **Spinal cord injury (complete)**

   ICD 10 Codes: T09.5

   Required impairment category: Physical

Optional impairment categories: Neurological, sensory, psychosocial

3. **Spinal cord injury (incomplete)**

   ICD 10 Codes: T09.7

   Required impairment category: Physical

   Optional impairment categories: Neurological, sensory, psychosocial

Other

1. **Malignant neoplasm of brain**

   ICD 10 Codes: C71

   Required impairment category: Neurological

   Optional impairment categories: Cognitive, psychosocial

2. **Metastatic cancer**

   ICD 10 Codes: C79.9

   Required impairment category: Physical

   Optional impairment categories: Cognitive

3. **Malignant neoplasm of blood or immune disease**

   ICD 10 Codes: C96

   Required impairment category: Physical

   Optional impairment categories: Cognitive

4. **Autoimmune disorders**

   ICD 10 Codes: D89.9

   Required impairment category: Physical

   Optional impairment categories: Cognitive

5. **Obesity – mandatory TAPIB**

   ICD 10 Codes: E66

   Required impairment category: Physical

   Optional impairment categories: Psychosocial

6. **Classical phenylketonuria**

   ICD 10 Codes: E70.0

   Required impairment category: Cognitive

Optional impairment categories: Psychosocial

7. **Disorders of pyruvate metabolism and gluconeogenesis**

   ICD 10 Codes: E74.4

   Required impairment category: Intellectual

   Optional impairment categories: Neurological, physical

8. **Other metabolic disorders**

   ICD 10 Codes: E88

   Required impairment category: Intellectual

   Optional impairment categories: Neurological, physical

9. **Dementia – rapidly progressing**

   ICD 10 Codes: F03.9

   Required impairment category: Cognitive

   Optional impairment categories: Not applicable

10. **Functional neurological disorder (FND) – mandatory TAPIB**

    ICD 10 Codes: F44.4

    Required impairment category: Neurological

    Optional impairment categories: Cognitive, sensory, physical

11. **Other language disorder**

    ICD 10 Codes: F80

    Required impairment category: Cognitive

    Optional impairment categories: Not applicable

12. **Peripheral neuropathy – does NOT require TAPIB**

    ICD 10 Codes: F90.0

    Required impairment category: Neurological

    Optional impairment categories: Sensory, physical

13. **Oppositional defiant disorder (ODD)**

    ICD 10 Codes: F91.3

    Required impairment category: Cognitive

    Optional impairment categories: Psychosocial

14. **Other hereditary ataxias**

    ICD 10 Codes: G11.8

    Required impairment category: Neurological

    Optional impairment categories: Cognitive, sensory, physical

15. **Dementia – early onset**

    ICD 10 Codes: G30.0

    Required impairment category: Cognitive

    Optional impairment categories: Not applicable

16. **Plegia**

    ICD 10 Codes: G83.1

    Required impairment category: Physical

    Optional impairment categories: Neurological

17. **Chronic pain – mandatory TAPIB**

    ICD 10 Codes: G89.4

    Required impairment category: Physical

    Optional impairment categories: Sensory, psychosocial

18. **Postural Orthostatic Tachycardia Syndrome (POTS) – mandatory TAPIB**

    ICD 10 Codes: I49.8

    Required impairment category: Neurological

    Optional impairment categories: Physical

19. **Lymphoedema – mandatory TAPIB**

    ICD 10 Codes: I89.0

    Required impairment category: Physical

    Optional impairment categories: Not applicable

20. **Chronic lung disease**

    ICD 10 Codes: J44.9

    Required impairment category: Physical

    Optional impairment categories:

21. **Chronic Obstructive Pulmonary Disease (COPD) – mandatory TAPIB**

ICD 10 Codes: J44.9A

Required impairment category: Physical

Optional impairment categories: Not applicable

22. **Osteoarthritis – mandatory TAPIB**

    ICD 10 Codes: M19.9

    Required impairment category: Physical

    Optional impairment categories: Not applicable

23. **Systemic lupus erythematosus**

    ICD 10 Codes: M32

    Required impairment category: Physical

    Optional impairment categories: Not applicable

24. **Ankylosing spondylitis**

    ICD 10 Codes: M45

    Required impairment category: Physical

    Optional impairment categories: Not applicable

25. **Fibromyalgia**

    ICD 10 Codes: M79.7

    Required impairment category: Physical

    Optional impairment categories: Sensory

26. **Renal failure**

    ICD 10 Codes: N18

    Required impairment category: Physical

    Optional impairment categories:

27. **Ehlers Danlos – does NOT require TAPIB**

    ICD 10 Codes: Q79.6

    Required impairment category: Physical

    Optional impairment categories: Not applicable

28. **Dyslexia**

    ICD 10 Codes: R48

Required impairment category: Cognitive

Optional impairment categories: Psychosocial

29. **Childhood apraxia of speech**

    ICD 10 Codes: R48.2

    Required impairment category: Neurological

    Optional impairment categories: Cognitive

30. **Short stature**

    ICD 10 Codes: R62.5

    Required impairment category: Physical

    Optional impairment categories: Not applicable

31. **Amputation – single limb or upper/lower limb**

    ICD 10 Codes: Z89

    Required impairment category: Physical

    Optional impairment categories: Cognitive, sensory, psychosocial

32. **Amputation – multiple**

    ICD 10 Codes: Z89.1

    Required impairment category: Physical

    Optional impairment categories: Cognitive, sensory, psychosocial

Article labels

PACE user role names

Add: User role name label

Delete: User role name label

No change.

Topics

Add: Topic label

Delete: Topic label

No change.

Case names

Add: Case name label

Delete: Case name label

No change.

Ownership

Add: Ownership label

Delete: Ownership label

No change.

Version control

Version	Amended by	Brief Description of Change	Status	Date
0.1	VFK746	Draft transfer of endorsed Attachment A – Descriptions of impairment categories to KA – required for legislation updates	DRAFT	2024-10-03
0.2	TCM150	Peer review	DRAFT	2024-10-03
0.3	VFK746	Implementation of Peer Review	DRAFT	2024-10-03
0.4	JM0122	EL1 review and approval	DRAFT	2024-10-07
0.5	VFK746	Class 1 endorsement – progress to BM	DRAFT	2024-10-08