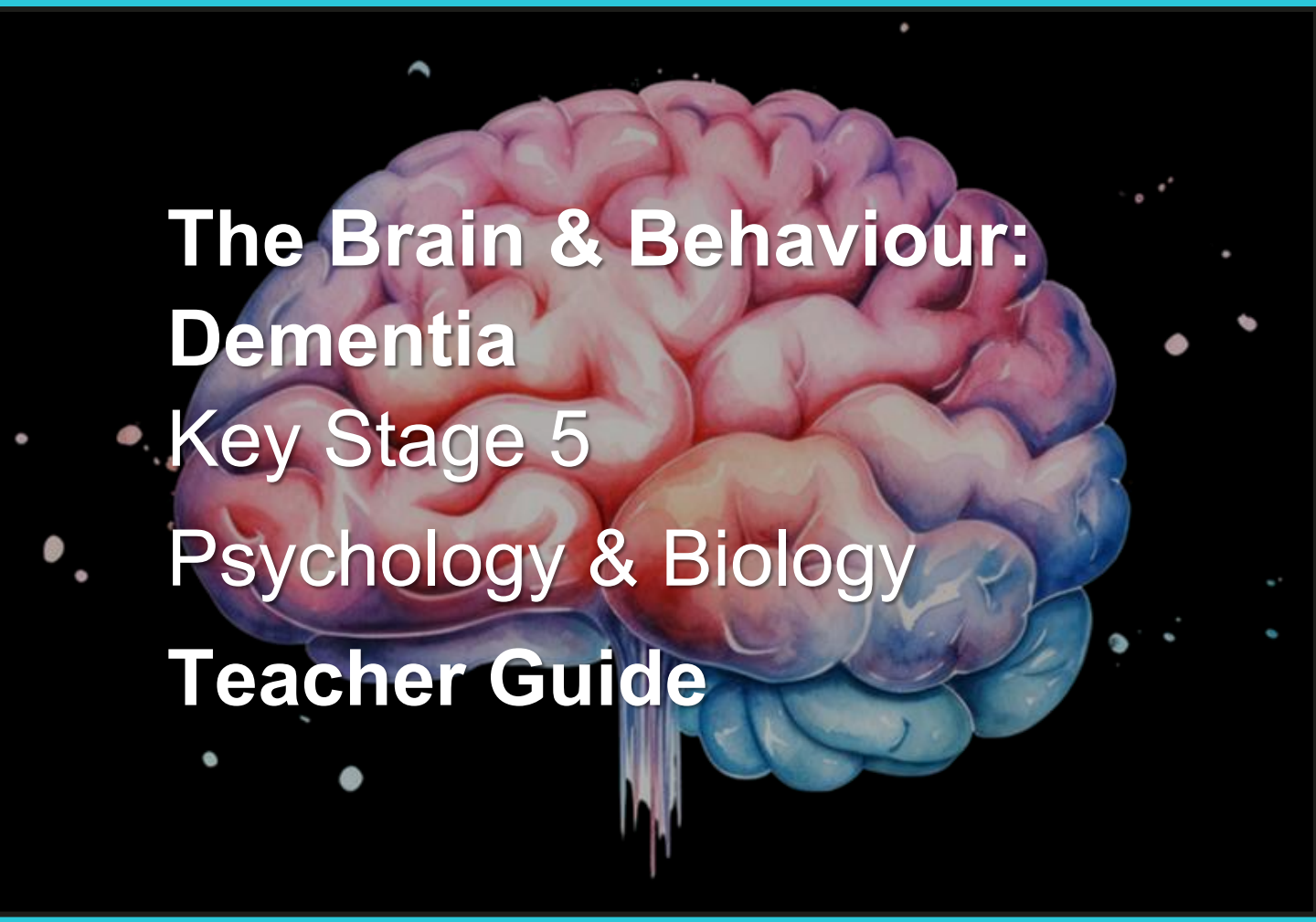
# Research-Based Curricula



## The Brain & Behaviour: Dementia

Key Stage 5

Psychology & Biology

**Teacher Guide**

2026

access ed
Building global university access programmes

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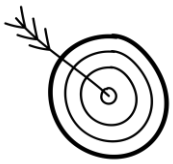
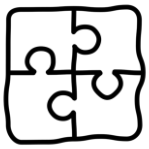


The Research-Based Curriculum is resources based on cutting-edge research, tailored for KS3, KS4 or KS5. The resources:

- *Support student attainment and progression*
- *Promote intellectual curiosity in students of all prior attainment*
- *Build understanding for more accessible 'stretch' beyond the curriculum*
- *Develop core academic skills that aid progression, including critical thinking, metacognition, and written and verbal communication*
- *Encourage students to see these subjects as engaging, worthwhile and inspiring for continued study*

## Content



Each RBC pack contains 1) Six resources that function as subject 'lessons'; 2) Activities at the end of each resource for students to test their learning; 3) Further Reading links related to the subject; 4) Final Reflection Activity as the final assignment; and 5) Teacher Guide and model activity answers (this document).

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# For Teachers

## Using RBC packs

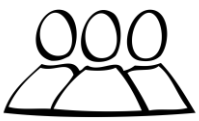
### Suggested School Use

Teachers can use these resources flexibly. Students can complete the resources individually or in groups, in or out of the classroom. These packs help teachers:

- *Use research-based learning to engage whole classes, not just as a 'stretch' for the most able*
- *Support more students earlier in high academic achievement*
- *Improve all-school enrichment strategies by providing opportunities and resources*
- *Increase motivation and subject interest*

To do this, we encourage the 'supported use' approach. In other words, teachers provide some guidance and support to students in their independent use of the RBC packs.

### Target Pupils



The RBC packs bring inspired subject learning to all students. These packs specially engage those students who might need extra support and encouragement and could benefit from engaging in the subject in a new way. The aim is that they are delivered with some teacher guidance to build the confidence of students as they complete a pack.

These packs build students' prior attainment rather than being offered only to those already academically able and motivated.

See more about delivery options on the following pages.

# For Teachers

## Using RBC packs

### Delivery Options

To ensure all students can benefit from these materials, we recommend they are delivered with 'supported use.'

Supported Use means this resource is designed to be used partially with teacher introduction or instruction. While not marked, each chapter and the final reflection activity are set up so a teacher can help ease the students into the subject area or use the resource in class.

More ideas for using these packs in your school:

1. **Research Challenge**

The resources can ignite curiosity about new topics and encourage independent research. Schools could hold a research challenge across a class or year group to submit a work based on the resources. Pupils could submit individually or in small groups, with a final celebration event.

2. **"STEM", "Social Sciences" or "Arts & Humanities" Morning/ Day**

We know class time can be tight, so some schools 'launch' these packs and have students start them as part of a special subject day. This can be great for all-staff engagement too.

3. **After School Club**

The resources can be completed in small groups (4-8 pupils) across weekly lunch clubs or after-school clubs. Groups can reflect on their learning by presenting a talk or poster on the subject matter at the end of the course.

# For Teachers

## Using RBC packs

### Delivery Options [cont.]

**4. Classroom Debate/ Discussion if a written Final Reflection Activity isn't possible**

Resource packs can function as 'transition' projects over the summer, serving as an introduction to the next level of study between KS3 and KS4, or KS4 and KS5. Students could present their reflections on the experience in a journal.

### Model Answers

For each answer section, you have been provided with a 'model answer'. These are an example of the sort of answer a student might give to each question, although in many cases there may be multiple answers a student could give. These serve as a starting guide.

Each answer is linked to a question from the RBC.

### Origin and Evaluation

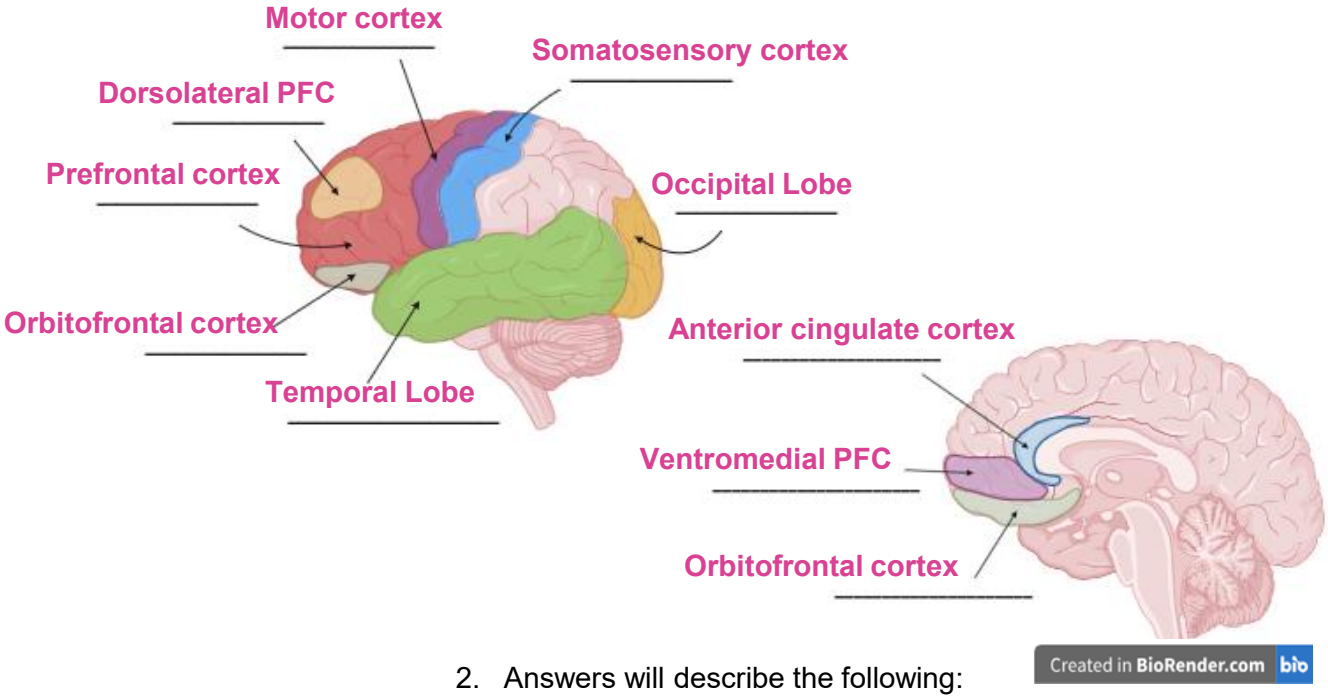
The RBC programme builds on the University Learning in Schools programme (ULiS), which was successfully delivered and evaluated through the London Schools Excellence Fund in 2015. The project was designed in a collaboration between Achievement for All and The Brilliant Club, the latter of which is the sister organisation of AccessEd. ULiS resulted in the design and dissemination of 15 schemes of work based on PhD research for teachers and pupils at Key Stage 4 and 5.

LKMCo evaluated the project. Overall, pupils made higher-than-expected progress and felt more engaged with the subject content.

# Resource One
# Model Answers

**Answers**

1. Please see the labelled diagrams below:




2. Answers will describe the following:

- **PFC:** Behavioural control and personality - Phineas Gage, Skinner evidence.
- **Orbitofrontal cortex:** impulsivity, aggression, eating behaviour.
- **Anterior cingulate cortex:** apathy, motivation, impulsivity, aggression, eating behaviour.
- **Dorsolateral and ventromedial PFC:** aggression, apathy, impulsivity, eating behaviour.
- **Temporal lobe:** auditory processing and memory.
- **Motor cortex:** voluntary action.
- **Somatosensory cortex:** touch and sensation.
- **Occipital lobe:** Visual processing.

# Resource One
# Model Answers

**Answers *(continued)***

3. Answers will cover how neuroimaging studies have linked the following regions to eating behaviour:

   Orbitofrontal cortex, anterior cingulate cortex, dorsolateral and ventromedial PFC.

4. This question asks students to think deeper about the real-life implications of assessing behaviour. Answers will select one assessment for each key behaviour from the table in Figure 3 and explore the real-life advantages and limitations of each to justify their choice.

   Answers should consider:

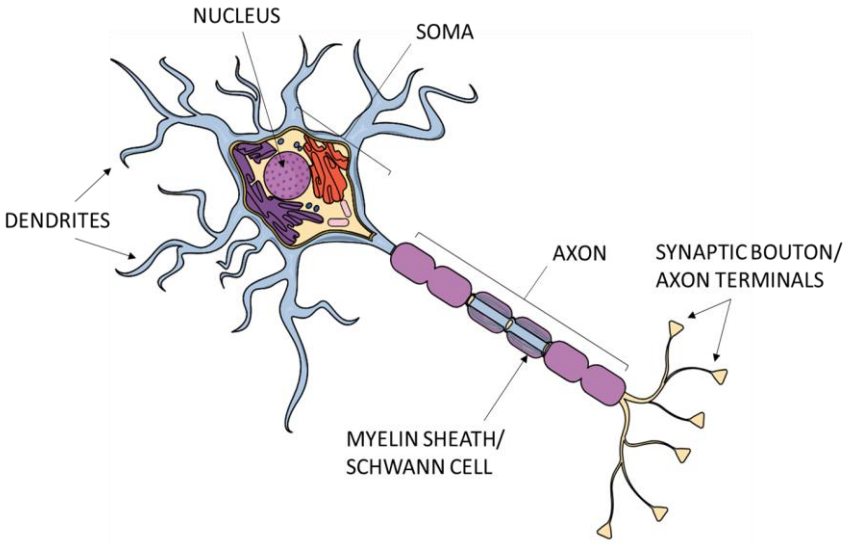
- Ideas of cost
- Time
- Feasibility/ aka resources needed for the measure
- Quality and detail of data collected

5. The poster will explore all aspects of the resource, including identifying the key regions of the PFC, discussion of the history of the PFC (e.g., Phineas Gage and Skinner research), discussion of neuroimaging studies in healthy people that have helped us understand the localisation of behaviour in the brain, and how we might assess that behaviour. Diagrams and spider diagrams are encouraged to condense the material into bite-sized chunks.

# Resource Two
# Model Answers

**Answers**

1. See labelled diagram below:




2. Model answers will describe the step-by-step process of neurotransmission and propagation of an action potential, including the terms saltatory conduction and action potential.

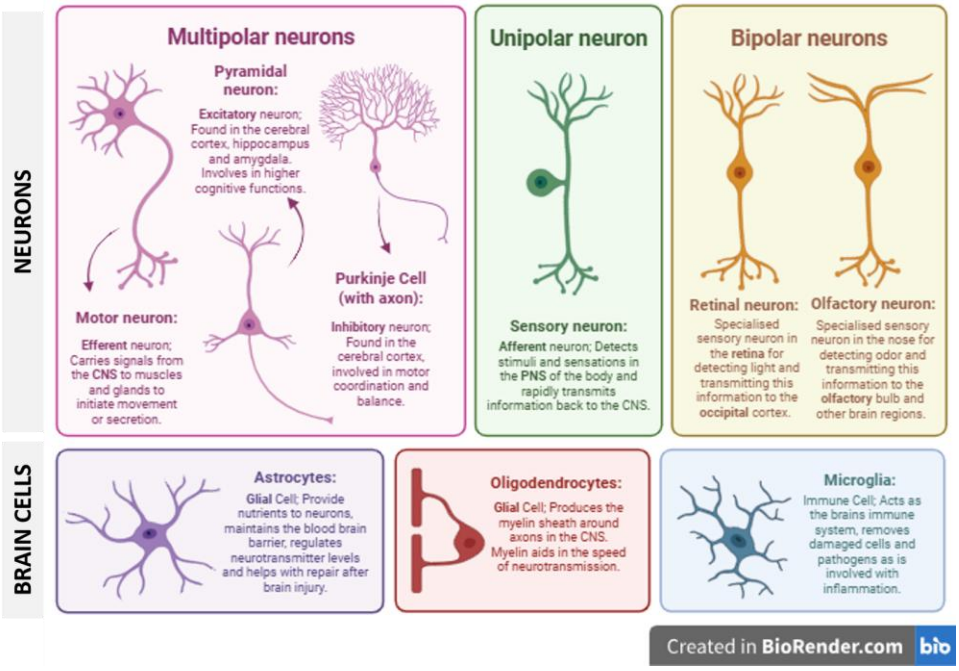
- **Resting potential maintained:** The neuron starts at a resting membrane potential (~ -70 mV), maintained by the sodium-potassium pump and ion channels.
- **Threshold reached:** A stimulus depolarises the membrane to a threshold (~ -55 mV), triggering the opening of voltage-gated sodium ($Na^+$) channels.
- **Depolarisation:** $Na^+$ rushes into the axon, causing the inside of the membrane to become more positive — this is the action potential.
- **Repolarisation:** Shortly after, voltage-gated potassium ($K^+$) channels open, allowing $K^+$ to flow out, restoring a more negative internal charge.

# Resource Two
# Model Answers

**Answers** ***(continued)***

- **Propagation of the action potential:** The change in voltage at one section of the axon triggers the next section to depolarise, allowing the signal to move rapidly along the axon. This continues until it reaches the axon terminal.
- **Saltatory conduction:** The rapid transmission of an action potential along a myelinated axon, where the electrical impulse jumps from one node of Ranvier to the next, speeding up signal propagation.

3. Answers will include comparisons between location, shape, function, and type of neuron (i.e., excitatory/inhibitory). Specifically referencing details in this diagram and any knowledge from further reading.



# Resource Two
# Model Answers

**Answers** ***(continued)***

4. See the passages from the resource, but in essence, model answers will include:

- Grey vs white matter organisation.
- Grey – neuron cell bodies, and unmyelinated axons, etc., - GM found in cerebral cortex.
- WM – myelinated axons, fatty myelin sheath – makes it look pale – found below the cerebrum cortex, organised into tracts.
- White matter tracts – bundles – for faster transmission and long-distance communication- network and connectivity– tractography on DTI.

5. Model answers will include the step-by-step process of synaptic transmission (below). They will include applied knowledge from further reading/watching the clip on why this is useful for brain function and plasticity, with particular reference to Hebb's theory of fire and wire, Long-term potentiation, and Long-term depression. They do not need to describe the processes of LTP and LTD, but must be able to use the idea that neurons that work together form stronger connections, which improve the efficiency of brain function.

- An action potential arrives at the axon terminal (synaptic bouton) on the **presynaptic neuron**.
- Depolarisation at the synaptic bouton opens voltage-gated calcium ion (Ca2+) channels, and $Ca^{2+}$ ions enter the axon terminal.
- **Vesicles** containing **neurotransmitters** then fuse to the presynaptic membrane and are released into the synaptic cleft via **exocytosis**.

# Resource Two
# Model Answers

**Answers** ***(continued)***

- Neurotransmitters **diffuse** across the synaptic cleft and bind to specific **receptors** on the **postsynaptic** membrane or the postsynaptic neuron.
- This causes ion channels to open in the postsynaptic neuron and cause either a depolarisation or hyperpolarisation of the postsynaptic neuron, depending on the type of neurotransmitter and receptor.
    - A depolarisation will elicit an **excitatory** postsynaptic potential (EPSP).
    - A hyperpolarisation will elicit an **inhibitory** postsynaptic potential (IPSP).

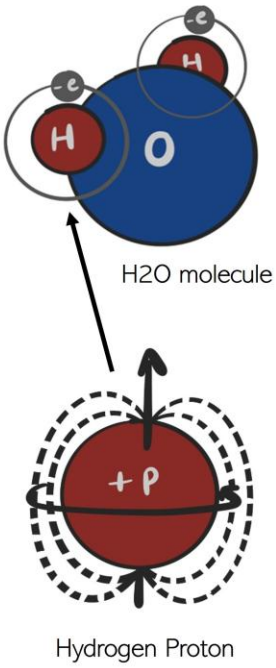
  If the signal is strong enough, it may trigger a new action potential in the postsynaptic neuron.
- The synapse is reset:
    - **Reuptake:** Neurotransmitters are reabsorbed into the presynaptic neuron.
    - **Degradation:** Enzymes break down the neurotransmitters.
    - **Diffusion:** some neurotransmitters diffuse out of the synaptic cleft.

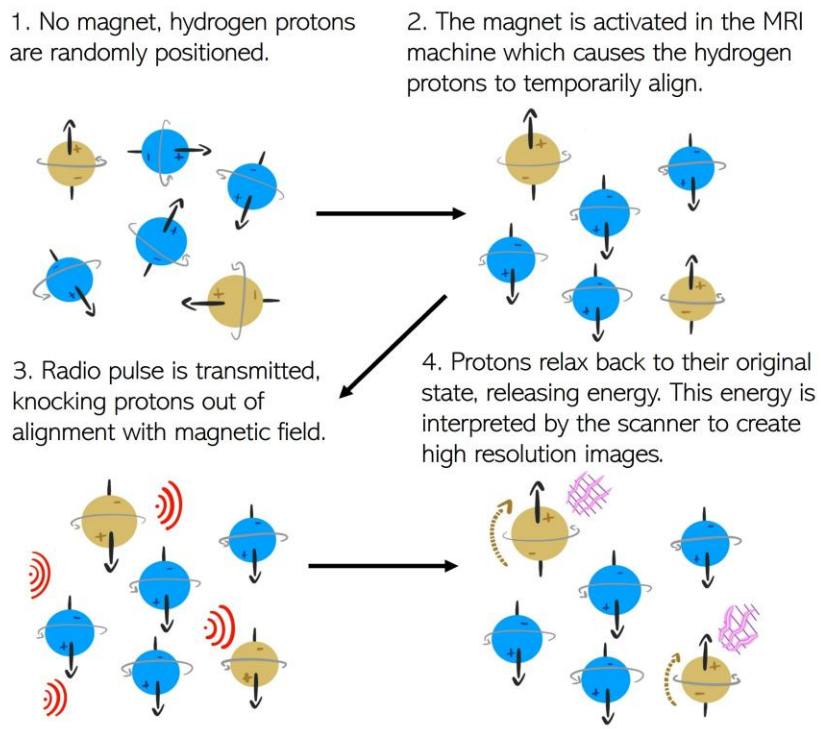
Final: Synaptic transmission – important for brain plasticity – allows neurons to communicate and modify the strength/efficiency of connections in response to activity and experience – Neurons that fire together wire together – Hebb. Activity-dependent changes—such as long-term potentiation (LTP) and long-term depression (LTD)—underlie the brain's ability to adapt to new information.

# Resource Three
# Model Answers

**Answers**

1. Physics image of MRI and how it works:




2. Structural MRI works by harnessing the **magnetic field** of **hydrogen atoms** from $H_2O$ molecules in different brain cells and tissues. In simple terms, your body and brain contain a lot of water, which is H2O molecules that include hydrogen atoms. Each hydrogen atom has a **proton** at its centre, which acts like a tiny magnet. An MRI machine is made up of a very strong magnet, and when a person lies in the machine, the magnet temporarily aligns the hydrogen protons with the strong magnetic field or against it. The MRI machine then transmits a radio wave pulse through the brain, knocking protons out of alignment with the field.

# Resource Three
# Model Answers

**Answers** ***(continued)***

When this radio frequency stops, the protons relax back to their original state, which releases energy. The MRI scanner measures this energy, and, through a complex process, a computer can convert these measured signals into high-resolution images. Different types of tissue produce different signals, allowing us to visualise tissue differences on a structural MRI.

3. See the table identifying the advantages and disadvantages of each type of scan:

| Scan type | Advantages | Disadvantages |
|---|---|---|
| **Magnetic Resonance Imaging (MRI)** | • Used to produce a 3D image<br>• More cost-effective (student mentions the difference in cost between 1.5T vs 7T)<br>• Can be used for clinical practice or research<br>• Depending on use, the scan can take 5-10 minutes<br>• Can collect structural and functional data within the same scan timeframe | • Unable to show underlying pathology<br>• Research scans can be expensive<br>• Lying still for a long time can be challenging for some participants |
| **Positron Emission Tomography (PET)** | • Indicates underlying pathology not visible on an MRI scan (so can show changes before atrophy occurs)<br>• Helpful for identifying disease-specific changes | • Injection of radioactive ligand - some people may not want this<br>• Due to radiation: higher risks<br>• Much higher cost<br>• Fewer PET scanners and sites that make certain tracers<br>• Lower resolution, can see general changes in larger regions<br>• Some tracers are only validated for research and not clinical practise |

# Resource Three
# Model Answers

**Answers** ***(continued)***

4. Students will create an aesthetic poster which describes how each neuroimaging technique works and includes the advantages and disadvantages of each technique. See resource 3 for explanations of each neuroimaging technique and the advantages and disadvantages laid out in (3).

5. Students should be directed to do some self-led research before the group discussions and debate. Students should debate the utility and feasibility of these imaging techniques in practice for research or clinical purposes. This will encourage students to use their critical and logical thinking skills. Some prompts for discussion should include:

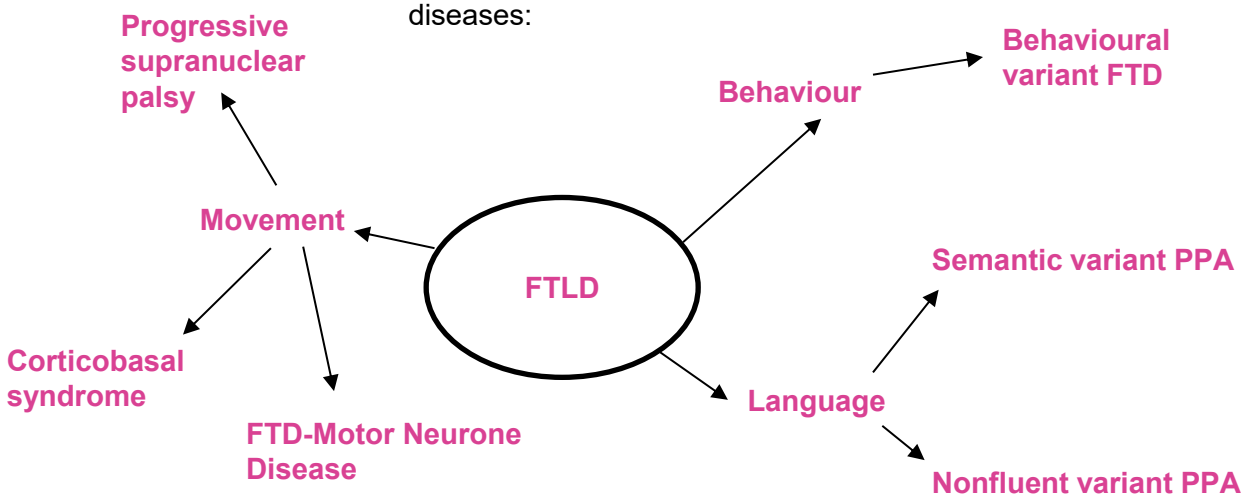
- Cost of scans – Direct the students to research the cost of these scans online
- Time
- Data acquired – what kind of data do you get, and why is that helpful in a research or clinical context
- How much of that type of data will be needed to get a good understanding? I.e., you may only need one structural MRI scan for a diagnosis, but 20 MRI scans for a group-comparison research study.

# Resource Four
# Model Answers

**Answers**

1. Neurodegeneration is the pathological progressive loss of structure or function of neurons in the CNS that occurs in diseases such as Alzheimer's disease (AD). Neurodegeneration does not describe the clinical symptoms of a dementia but instead characterises the physical deterioration of brain tissue of the underlying disease.

2. See the spider diagram of the neurodegenerative FTLD diseases:




3. FTLD-TAU, FTLD-TAR DNA-binding protein 43 (FTLD-TDP-43).

4. Students will select one of the following:

- **Normal function:** TAU stabilises microtubules and axons, and TDP-43 regulates RNA.
- **Accumulation:** TAU: neurons and glia, TDP-43: forms cytoplasmic inclusions.

# Resource Four
# Model Answers

**Answers *(continued)***

Students may also include a key difference from their own research, but this must be justified.

5. Students will set out this question like a short-form essay. They should answer in the following format:

- First, explain what tau is and how tau accumulates in neurons (proteinopathy) (resource four):

  *FTLD-Tau is a microtubule-associated protein found in the microstructure of a neuron. In FTLD, abnormal tau phosphorylation can cause tau to misfold and accumulate toxically within neurons, forming tangles that can lead to cell death.*

- Explain how this disrupts neural function and synaptic communication (resources two and four):

  *The idea that TAU buildup affects brain cell structure and function may affect neurotransmission and plasticity. If neurons do not fire together, they will not wire together and therefore will be used less and may die off.*

- Think about where this might be happening in the brain if someone has behavioural changes and cognitive symptoms (resource one)

  *This section asks the student to consider what they were taught in resource one regarding the localisation of behavioural control and to link it to ideas about where in the brain these pathogenic processes might occur if someone has behavioural problems associated with FTD. Students should therefore describe how pathogenic TAU protein aggregations might be disrupting brain cells in the frontal and temporal lobes, particularly in the PFC.*

# Resource Five
# Model Answers

**Answers**

1. Dementia is defined as a clinical **syndrome**, whereby a person's cognitive abilities have declined, affecting their ability to look after themselves and function normally in everyday life. Dementia can affect several cognitive processes, including memory, language, problem-solving and **behaviour**. Dementia is not technically a disease, but instead is a clinical label that doctors give to a group of symptoms which are thought to be caused by a **neurodegenerative disease**.
2. Students should write a paragraph discussing the following symptoms:

- Personality and behavioural changes
- Increased impulsivity and poorer judgement
- Increased executive dysfunction
- Emotional blunting/apathy
- Lack of insight

3. In the structural MRI image, the frontal and temporal lobes have increased atrophy. Students should relate their understanding of brain localisation to the orbitofrontal cortex, anterior cingulate cortex, and atrophy of the dorsolateral and ventromedial PFC to the symptoms described in (2).
4. MAPT, C9orf72, GRN.
5. In presymptomatic healthy volunteers, Malpetti et al (2021) showed reduced synaptic density in the thalamus of C9orf72 mutation carriers, adding to the finding that brain atrophy occurs years before symptom onset. This is a vital finding for the understanding of disease development and progression.

# Resource Six
# Model Answers

**Answers**

1. Students will describe FTD prevalence in the following context and statistics:

   *57 million people worldwide were diagnosed with dementia in 2021 (WHO), over 60% in LMICs. FTD accounts for approximately 10-20% of all dementia cases; however, it accounts for 3-25% of cases under 65, and 10-15% of those cases are caused by a genetic mutation in one of three genes. These figures are likely higher in LMICs, but the data have not yet been collated.*

2. Students will describe the different diagnostic tools to diagnose dementia:

- *What is a biomarker? – a biological indicator of what is happening inside the body and can be used to monitor disease progression.*
- *Neurologist listens to symptoms described by the patients or carers, and may look at performance on neuropsychiatric and cognitive tests. For someone with bvFTD, patients may describe impulsive or socially inappropriate behaviours and are likely to perform badly at complex tasks involving executive function, but may perform well on memory tasks.*
- *Structural MRI scan - see brain atrophy in areas affected in FTD, such as the frontal or temporal lobes, and more specifically in areas such as the orbitofrontal cortex and anterior cingulate cortex (see resource one).*
- *If the MRI scan is inconclusive, the patient may request a functional PET scan, such as an FDG-PET scan, to assess glucose metabolism (see resource three). This can be useful for seeing whether certain areas of the brain, such as the frontal and temporal lobes, are not working as they should.*

# Resource Six
# Model Answers

**Answers *(continued)***

- *Blood tests and possibly even a **lumbar puncture**, if they need to rule out any other diagnosis. **Cerebrospinal fluid** (CSF) taken from a lumbar puncture can be used to identify if any Alzheimer's disease-type biomarkers are present. If so, this might rule out a diagnosis of FTD. Genetic analyses of blood samples can indicate autosomal dominant genetic mutations known to cause FTD (see resource 5). Using all of these diagnostic biomarker tools and techniques can aid in diagnosing FTD.*

3. Students might think and explore reasons why we don't yet have a cure for dementia:

- Not enough funding for research.
- Rare dementias are hard to diagnose and recruit for research.

And then go on to say that, as such, we currently have some treatments that can help manage some of the symptoms:

- SSRIs – may be prescribed to help with impulsivity/disinhibition, irritability and agitation in FTLD.
- How these SSRIs work – with particular reference to synapses and neurotransmitters.
- How are these different to SARIs as described in the resource?
- But that SARIs might also help agitation.
- Speech and language therapists for language difficulties in FTD.
- Occupational therapists for daily living adaptations.
- Environmental modifications – dementia cafes and support groups.

# Resource Six
# Model Answers

**Answers** ***(continued)***

4. Encourage open group discussion to think about caregiver burden, societal burden, individual difficulties, the widespread problem and the growing incidence rates of dementia in people living longer. Encourage them to list these reasons in a spider diagram or table form for the final task.
5. Students should logically format the presentation, covering the following key factors:

- **What**: What are the current challenges in dementia care and research?
- **Where/Who**: Prevalence and incidence rates refer to worldwide statistics. Include mention of the patient affected, caregivers, and society.
- **How**: What do we already know about a rare dementia like FTD? How does this knowledge help us to ask the right questions?
- **Why**: Why is this important? Using everything from this final resource, why is it so important that we investigate dementia?

# Final Reflection Activity

## Further Guidance

Reflecting on everything in this coursebook, students are encouraged to integrate their knowledge from each resource and explore further areas of interest to answer this question. The following structure is encouraged:

- **Introduction:** Outline what you will cover.
- **Dementia:** Briefly define dementia and neurodegeneration, including prevalence, prognosis and key symptoms (Hint: to make this easier for yourself, pick bvFTD).
- **Brain regions:** Outline the key brain regions affected in this type of dementia and how this relates to the core symptomology of the syndrome.
- E.g., bvFTD – Frontal and temporal lobes, orbitofrontal cortex, anterior cingulate and ventromedial and dorsolateral PFC – regions linked to behaviour in aggressive behaviour, apathy, impulsivity, and eating behaviour.
- **Methods for diagnosis and investigation:** Discuss the different methods for diagnosing and researching dementia. Include brain imaging techniques and other diagnostic tools briefly outlined in the resource.
- **bvFTD diagnosis:** clinical assessment, cognitive tests, blood test, MRI, FDG PET, CSF to rule out AD
- **Hypotheses of dementia:** Outline how pathology and synaptic degeneration might underlie neurodegeneration.
- bvFTD – protein build up from TDP-43 or TAU might cause cell death, but synapse loss is thought to precede cell death – Malpetti et al 2023 and 2021 research
- **Current treatment approaches and finding a cure:** Discuss the current options for treatment and how scientists are working to develop a cure.
- Environmental modifications, possible SSRIs, speech and language therapists, occupational therapists, and dementia support groups.
- **Conclusion:** Briefly explain and discuss why this matters, relating to prevalence and burden of the disease and how this is important on an individual *and societal level*

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