SL IB Psychology

Your notes

Neurotransmitters & Their Effect on Behaviour

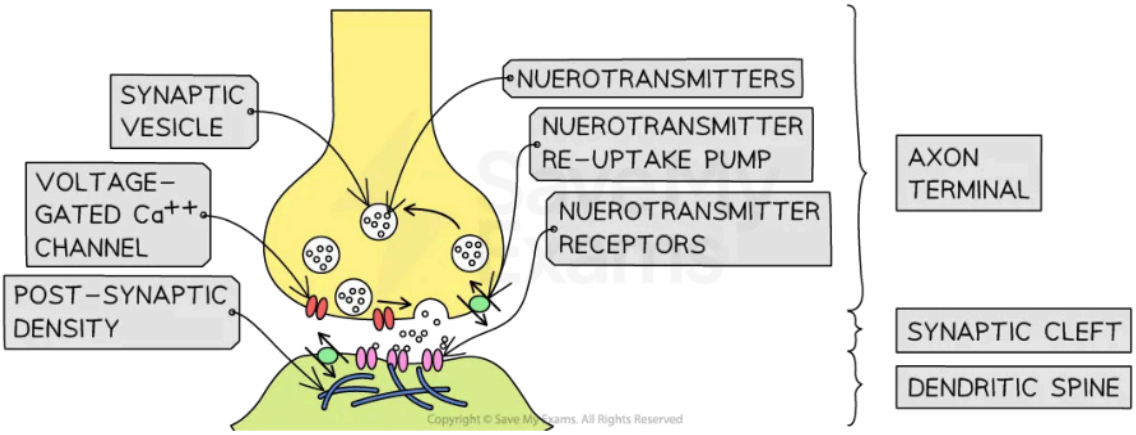
Contents

Your notes

Neurotransmitters & Their Effect on Behaviour

What are neurotransmitters?

- **Neurotransmitters** are chemicals that are transported via electrical impulses from the **presynaptic neuron** to the **post-synaptic neuron** across the **synaptic cleft**
- An **action potential** begins the process of **neurotransmission** at the **dendrites** of the neuron: this comes in the form of an **electrochemical** impulse which travels down the **axon** to the **terminal buttons** containing the **synaptic vesicles** holding the neurotransmitter which is then released into the synaptic cleft
- Synapses can be **excitatory** in the **presynaptic neuron:** this *increases* the probability of an action potential occurring in the **post-synaptic** neuron i.e. the neuron 'fires'
- Synapses can be **inhibitory** in the presynaptic neuron: this *decreases* the probability of an action potential in the post-synaptic neuron
- Molecules of the neurotransmitter which are not passed across the synaptic cleft are taken back up into the axon of the presynaptic neuron (known as **reuptake**)
- Examples of neurotransmitters include **dopamine** and **serotonin**

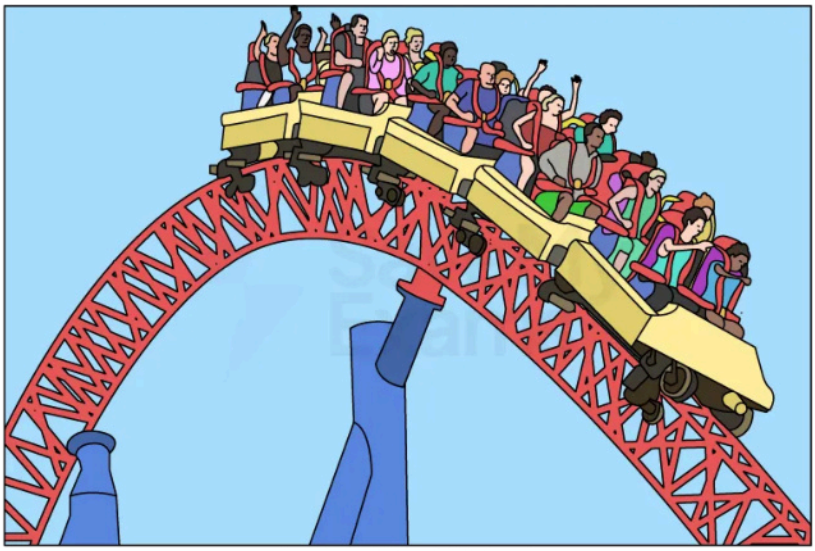


The process of neurotransmission

What is dopamine and what effect does it have on behaviour?

- Dopamine is one of a group of neurotransmitters known as **monoamines**
- Brain regions and structures associated with dopamine production include the **ventral tegmental area,** the **nucleus accumbens,** the **caudate nucleus** and the **basal ganglia**
- Dopamine (as is the case with all neurotransmitters) acts a chemical messenger, communicating between cells (from the brain to the rest of the body)
- Dopamine has been strongly associated with reward, **motivation**, intense pleasure and, by association, **addiction**

- Dopamine has strong associations with the function of movement and motor skills as seen in dopamine deficiency in **Parkinson's disease**
- Irregular levels of dopamine have also been linked to the symptoms of **schizophrenia** (known as the **dopamine hypothesis**)



Roller coasters provide a dopamine boost for some people!

What is serotonin and what effect does it have on behaviour?

- Serotonin is also a monoamine, specifically known as **5-HT** as it can also act as a **hormone** (but for the purposes of your IB studies only refer to it in its role as a neurotransmitter)
- Serotonin plays a crucial role in regulating functions such as sleep, mood, body temperature
- Low or irregular levels of serotonin have been linked to **affective disorders** such as **depression** and **anxiety**
- Some drugs prescribed for depression (**antidepressants**) are known as **SSRIs** (**Selective Serotonin Reuptake Inhibitors**) as they prevent the reuptake of serotonin, ensuring that it is passed onto the post-synaptic neuron
- Serotonin is made from an amino acid called **tryptophan** which can be found in foods such as turkey, eggs, cheese and tofu

Examiner Tip

Remember that when you answer a question on neurotransmitters, make sure that you really are answering the question! As pointed out above, some neurotransmitters also function as hormones so it is vital that you have selected the correct choice in order to answer the question. See our suggestion of which hormones to use in the exam in the Hormones and Pheromones section of these Revision Notes.

Your notes

Which studies focus on the effect of neurotransmitters on behaviour?

- **Fisher et al. (2005)** in her study of the link between romantic love and **dopaminergic** activity in the brain
- **Brunner et al. (1993)** in his case study of abnormal, anti-social behaviour in one family explained by a dysfunctional **MAOA** gene (linked to the 5-HT) gene

Fisher and Brunner's studies are available as separate Key Studies – just navigate the Brain and Behaviour section of this topic to find them.


Your notes

Worked example

To what extent could neurotransmitters be said to affect behaviour? [22]

The key to answering this question is to ensure that you are meeting the demands of the command term. 'To what extent' requires you to present an argument that X theory or study can only go so far in providing an explanation for Y behaviour. Take a look at this paragraph for an example of how to build this into your critical thinking:

The extent to which dopamine could be said to affect behaviour is limited in terms of Fisher et al.'s (2005) experiment. Using fMRI technology to determine a link between early-stage romantic love and dopaminergic activity is a reductionist approach to explaining a complex, multi-faceted behaviour which is unlikely to be exactly the same for each person experiencing it. To this extent the study can only provide evidence that the 17 participants in the study appeared to experience similar brain activity in the ventral tegmental area, caudate nucleus and other dopamine-associated regions. The results have limited generalisability due to the artificial task and environment of the study so general laws of behaviour cannot be established using this study alone.

Two Key Studies of Neurotransmitters: Fisher et al. (2003) & Brunner et al. (1993)


Your notes

Examiner Tip

You can use Fisher et al. (2005) in a question on Techniques Used to Study the Brain. Brunner et al. (1993) can also be used to answer questions on Genes& Behaviour and Genetic Similarity

Key Study 1: Fisher et al. (2005)

Your notes

Aim: To investigate a possible link between dopaminergic brain regions/systems and the early stages of romantic love

Participants: A **self-selected** sample of 10 female and 7 male students from New York State University, aged 18–26 years old (mean age = 20 years). All participants reported that they were 'in love' (time spent together from a range of 1–17 months with a mean of 7 months)

Procedure: Participants were placed in an **fMRI** scanner and shown a photograph of their romantic partner followed by a distraction task and then a 'neutral' photograph of an acquaintance with whom they had no emotional connection

Results: The fMRI showed that the areas of the brain most active when the photograph of the romantic partner was viewed were the right **ventral tegmental** areas in the midbrain and the right **caudate nucleus** also in the midbrain. These regions of the brain have been associated with **dopamine** production which in turn has been associated with **motivation** and **reward** i.e. someone has the desire to perform a behaviour because of the positive consequences of that behaviour

Conclusion: People who are in the early stages of romantic love may access brain regions associated with dopamine release when in the presence (physical or virtual) of their romantic partner. Therefore, it could be said that people become 'addicted to love'

Evaluation of Fisher et al. (2005)

Strengths

- The findings support previous research into the role of dopamine in **substance** addiction so it is interesting to see how romantic love may fall under the same framework of **craving** and **withdrawal**
- The use of fMRI clearly shows a link between dopaminergic areas of the brain only when the photograph of the romantic partner was shown which increases the **internal validity** of the study i.e. Fisher really was measuring the effect of romantic love on the brain

Limitations

- It could be argued that a sample with a mean age of 20 years are more likely to be socially active and involved in pleasure-focused activities than an older sample which would mean that the **'pleasure centre'** of their brains would be more receptive to dopamine
- There could be other explanations for the activation of the dopamine-rich areas of the brain being active during the fMRI e.g. excitement at taking part in a study; curiosity as to the outcome of the study, so Fisher cannot claim cause-and-effect from her findings

Key terms:

- **Dopamine**
- **Ventral tegmental area**
- **Caudate nucleus**

Key Study 2: Brunner et al. (1993)

Your notes

Aim: To investigate the violent, anti-social behaviour of specific male members of a large family in the Netherlands. The behaviour exhibited by the males in the family was borderline **mental retardation** (their average **IQ** was around 85), and violent behaviour.

Participants: 5 males from a family in the Netherlands, all of whom had the same **genetic** condition, transmitted via the **X chromosome** on the **MAOA gene**. The family lived in a remote rural region of the Netherlands. Two **carrier** females and one **non-carrier** female were used as a control and compared with 3 clinically affected males. (*Carrier means that some of the females carried the faulty gene in their genotype but it was not expressed in the phenotype i.e. their behaviour).*

All of the affected males acted aggressively when angry, fearful, or frustrated. Examples of their violent, anti-social behaviour included attempted rape of one of the female members of the family, arson, attacking a mental institute warden with a pitchfork, voyeurism (spying on the females in the family at night), exhibitionism (appearing naked in public). Only one of the males in the family with the faulty gene finished primary education.

Procedure: A case study (close study of a small group of individuals from one family) and quasi experiment. A quasi experiment is one in which the **IV** is naturally occurring i.e. it can't be manipulated by the researcher – in this case the individuals involved either had the faulty gene or they didn't have the faulty gene. Brunner conducted **DNA analysis**, obtained via urine samples. **Observations** of the males and **interviews** with the family provided **qualitative** data.

Results: None of the affected males had **dysmorphic** signs of the genetic mutation i.e. they didn't 'look abnormal' or different physically to the unaffected males. Unaffected males in this family attended normal schools, and most had steady jobs. All the females (including several carriers) also functioned normally.

A **base change** in the DNA structure was identified in all 5 affected males. This in turn resulted in flawed **monoamine metabolism**, which is linked with a deficit of the **enzyme monoamine oxidase A** (MAOA) – an enzyme which (among other functions) regulates the supply of **serotonin** levels to the brain. The reason only males are affected is because it is specifically the single X chromosome which is responsible for the production of MAOA.

Conclusion: The dysfunctional MAOA gene may be linked to irregular serotonin **metabolism** which could in turn be responsible for the mental retardation and aggressive behaviour of the affected males. MAOA deficiency may account for an individual's inability to regulate their aggression. This MAOA deficiency is now known as **'Brunner syndrome'**.

Evaluation of Brunner et al. (1993)

Strengths

- Serotonin deficiency has been linked to unbalanced mood in **previous research** so there may be some **validity** to 'Brunner Syndrome'
- By using one extended family the researchers were able to directly test their theory by using family members as **control** samples rather than an unrelated general population, thus validating the idea

that the males' behaviour was **genetic** rather than as a result of their **environment**

Limitations

- Brunner's research cannot conclusively support the idea that the affected males' anti-social behaviour was as a result of serotonin deficiency which means that the findings may lack **validity**
- The affected males may have encountered more adverse reactions from others e.g. hostility, aggression, confrontations due to their reduced IQ and lack of impulse-control which could have exacerbated their anti-social tendencies i.e. **nurture** may have influenced their behaviour as well as **nature**

Key terms:

- **X Chromosome**
- **MAOA gene**
- **Serotonin**

Worked example

Explain the effect of one neurotransmitter on human behaviour. [9]

See how the following paragraph adheres to the command term 'Explain':

Research into serotonin has found that it has a calming effect on behaviour as it has been linked to balancing mood in people with, for example, depression and OCD. The MAOA gene has been implicated in the production of serotonin (5 H-T) so if there is an imbalance in the production of this enzyme via faulty genetics then the resulting behaviour is likely to be aggressive, hostile and anti-social. This is what Brunner et al. (1993) found in their investigation of specific affected males in a large extended family in the Netherlands.

Your notes

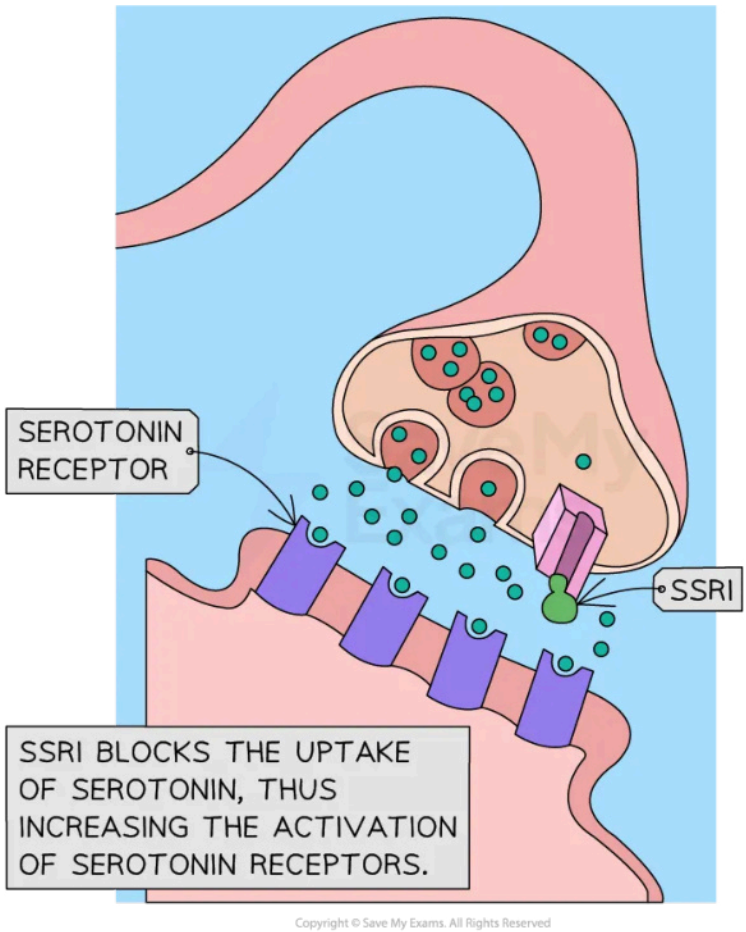
Neurotransmitters: Agonists & Antagonists

Your notes

Neurotransmitters – Agonists & Antagonists

What are agonists?

- An **agonist** is a molecule that binds to a **synaptic receptor** and activates it to promote a reaction e.g. with **neurotransmission** this reaction takes place within the **synapse**
- **Drugs** can act as agonists i.e. they affect the degree of a **neurotransmitter's** effect (as they are made outside of the body they are known as **exogenous** agonists)
- If a drug (e.g. an **anti-depressant)** increases the effect of a neurotransmitter (e.g. **serotonin)** it is known as an agonist
  - ***Selective Serotonin Reuptake Inhibitors (SSRIs)*** *are serotonin agonists as they prevent the re-absorption of serotonin back into the presynaptic neuron and thus increase the amount of serotonin available to travel around the brain*
  - *SSRIs are commonly used to treat* ***depression*** *but they can also be prescribed to treat* ***anxiety disorders*** *such as* ***OCD*** *and* ***PTSD***



The action of an SSRI is an example of an agonist in action.


Your notes

What are antagonists?

- An **antagonist** is a molecule that binds to a synaptic receptor but this time the effect is the opposite to that of an agonist: it decreases the effect of the neurotransmitter
- **Drugs** can act as antagonists i.e. they affect the degree of a **neurotransmitter's** effect (as they are made outside of the body they are known as **exogenous** antagonists)
- If a drug (e.g **ecicopam)** decreases the effect of a neurotransmitter e.g. **dopamine** it is known as an antagonist
- An antagonist reduces the action of what would normally happen when a substance binds with and effectively blocks the receptor
- Dopamine antagonists stop dopamine binding with receptors in the post-synaptic neuron which then prevents the usual feelings of reward, pleasure and motivation from occurring
- Dopamine antagonists are used to treat drug **addiction** but they can also be used to treat **schizophrenia, psychosis** and **bi-polar disorder**

Examiner Tip

Remember that agonists and antagonists do not change the effect of a neurotransmitter. An antagonist will not change an excitatory neurotransmitter into an inhibitory one; it will just lower the degree of the excitatory response.

Which studies focus on agonists & antagonists?

- **Crockett et al. (2010)** in a **lab experiment** which investigated the role of SSRIs in **prosocial** behaviour
- **Romach et al. (1999)** used a **natural experiment** to investigate the effectiveness of a dopamine antagonist on participants with cocaine addiction

Crockett and Romach's studies are available as separate Key Studies – just navigate the Brain and Behaviour section of this topic to find them.

Examiner Tip

It is hugely important that you don't confuse agonists with antagonists in the exam as the two are NOT the same! Try to find your own way of remembering which is which e.g. 'My aunt is an antagonist as she decreases my fun' = antagonists decrease the effect of a neurotransmitter.

Drug treatment may be necessary to break addiction to other drugs such as cocaine.

Worked example

Explain the role of one agonist in human behaviour. [9]

Take a look at this paragraph for an example of how to present the role of the SSRI and in an exam answer:

Selective Serotonin Reuptake Inhibitors (SSRIs) are agonists (antidepressant drugs) for serotonin as they block the brain's re-absorption of serotonin. What this means is that during the process of synaptic transmission some molecules of serotonin do not cross the synaptic gap but instead are absorbed back into the presynaptic neuron (the neuron that fired the electric action potential to start the process of neurotransmission). Some SSRIs are agonists as they block the receptor responsible for the re-uptake of serotonin back into the presynaptic neuron (leaving it in the synapse to bind with the receptor again) and therefore they are used to treat affective disorders such as depression and anxiety as these illnesses have been linked to irregular levels of serotonin in the brain.

Your notes

Two Key Studies of Agonists & Antagonists: Crockett et al. (2010) & Romach et al. (1999)

Your notes

Key Study 1 (Agonist): Crockett et al. (2010)

Aim: To investigate the role of a **serotonin agonist** (**SSRIs** specifically) in prosocial behaviour (e.g. deciding that harmful behaviours towards others are unacceptable).

Participants: 24 males from Cambridge in the UK with a mean age of 25.6 years. The participants were screened for **psychiatric** and **neurological** disorders before the study began.

Procedure: The participants were given either an SSRI (Citalopram), a drug used to treat **ADHD** or a placebo. The experiment used a double-blind design.

The first part of the procedure involved participants being asked to make moral judgements about a series of **hypothetical** scenarios, for example:

1. *Would you push someone in front of a train if it meant saving five other people?* This was the **emotionally salient** 'personal harm' condition i.e. the idea involves actually physically pushing someone in front of a train
2. *Would you flick a switch so that a train hits one person instead of five?* This was the less emotionally salient 'impersonal harms' condition i.e. the idea involves harming someone at a distance and is thus less personal

The responses were measured according to how many times each participant judged that an action (personal or impersonal harm) was 'acceptable'.

Results: The emotionally salient personal harm condition (i.e. the idea of actually pushing someone in front of a train) produced the lowest number of 'acceptable' responses in both conditions i.e. participants were reluctant to imagine themselves physically harming another person even to save five other lives.

Participants in the SSRI condition (who had been taking Citalopram) made more prosocial responses i.e. by condemning harmful actions, compared to the ADHD drug group and the placebo group. The moral judgements made by the ADHD drug group and the placebo group were similar, showing no significant differences in prosocial responses.

Conclusion: Some SSRIs, such as Citalopram, may function as serotonin agonists, enhancing the effect of the serotonin in the brain which in turn may promote prosocial behaviour.

Evaluation of Crockett et al. (2010)

Strengths

- The use of a double-blind procedure increases the **internal validity** of the findings
- Screening the participants for psychiatric and neurological disorders before the study also increases the validity of the findings as it helps to factor out any possible **confounding variables** linked to mental illness

Limitations

- A sample of 24 participants is very small and reduces both the **reliability** (due to a lack of **statistical power**) and **generalisability** (again, due to the sample size) of the findings
- The study used an **independent measures** design so the differences in prosocial behaviour could simply be due to **participant variables** e.g. participants in the SSRI group may simply all have been naturally more prosocial than those in the other two groups


Your notes

Key terms:

- **SSRIs**
- **Serotonin**
- **Double-blind**

Key Study 2 (Antagonist): Romach et al. (1999)

Your notes

Aim: To investigate the role of a **dopamine antagonist** (e**copipam**), in the treatment of cocaine **addiction**.

Participants: A **self-selecting** sample of 15 participants (3 women, 12 men, aged 26–44 years with a mean age = 34 years) who were cocaine addicts.

Procedure: The study took place over two weeks during which the participants were hospitalised to ensure that **experimental controls** could be guaranteed. The procedure used a **randomised** double-blind design with 4 conditions: participants took either a placebo,10mg, 25mg, or 100mg of ecopipam orally on 4 separate occasions.

Two hours after the placebo or ecopipam had been taken each participant was injected with 30mg of cocaine. The participants were assessed for the **subjective** (individual) effects of having taken cocaine e.g. **biological measures** such as blood pressure and heart rate and psychological measures, e.g. feelings of being 'high' or feelings of anxiety, confusion, feeling the effect of the drug etc. All of the participants were asked about their desire to take cocaine which was an important measure as this would indicate how effective ecopipam was in reducing **cravings** for cocaine.

Results: Participants in the dopamine antagonist group (who had been taking ecopipam) reported reduced feelings of **euphoria** and of being 'high' (which is the opposite of what would normally follow a cocaine hit). The ecopipam also appeared to reduce the anxiety associated with taking cocaine e.g. **withdrawal**, and worry about how to 'score' the next hit. Participants who took ecopipam expressed less desire to use cocaine than those in the placebo group. The most effective dosage of ecopipam was found to be between 25mg and 100mg.

Conclusion: Some drugs such as ecopipam may function as dopamine antagonists and may be effective in reducing the craving for cocaine as well as the effects of taking cocaine.

Evaluation of Romach et al. (1999)

Strengths

- This study has useful **application** as the findings could be used to inform further treatment of cocaine addicts, and possibly those addicted to other substances such as alcohol
- The use of several different measures e.g. heart-rate, **self-reported** anxiety means that the study used **triangulation** which increases the **validity** of the findings

Limitations

- Self-reported data is prone to **social desirability bias** e.g. the participants may have reported reduced feelings of craving to please the researchers or to project a more positive image of themselves and their recovery from drug addiction
- The study does not tell us what happened after the addicts left the safe, controlled environment of the hospital – it could be that some of the ecopipam group **relapsed** quickly which would mean that the drug had no real effect long-term

Key terms:

- **Ecopipam**

- **Dopamine**
- **Cravings**


Your notes

Examiner Tip

Remember that you can only be asked an agonist/antagonist question on Section A of Paper 1: it will NOT appear as an ERQ 22-mark question on Section B. However, evaluation of each study is included here as you might wish to use either study in a Section B question (ERQ, 22 marks) on the effect of one neurotransmitter on behaviour. If you choose serotonin for example, you could use Brunner et al. (1993) as the effect of serotonin on behaviour (anti-social, violent behaviour linked to irregular serotonin levels) and then use Crockett et al. (2010) as further evidence that balanced serotonin levels (achieved via SSRIs as serotonin agonists) are linked to prosocial i.e. non-violent behaviour.

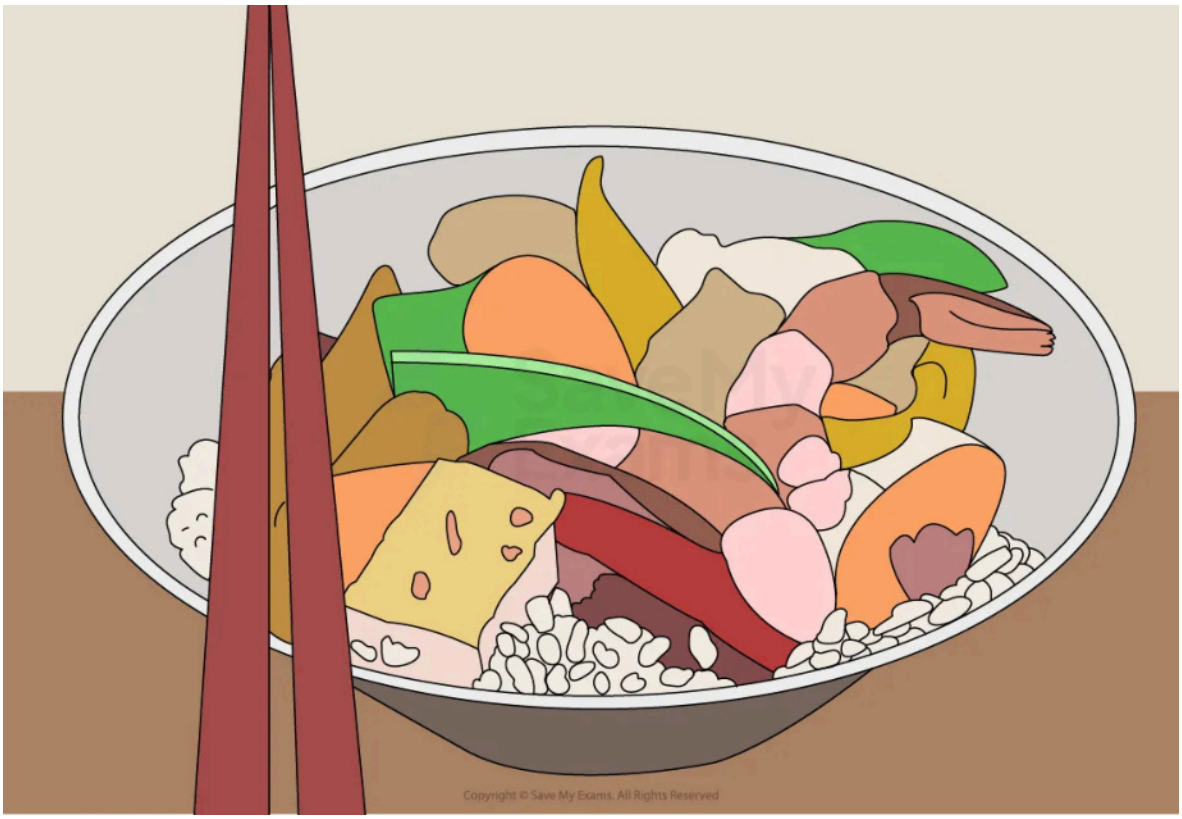
Excitatory & Inhibitory Neurotransmitters

Your notes

Neurotransmitters – Excitatory & Inhibitory Synapses

What are excitatory synapses?

- During the process of **neurotransmission** small sacs called **vesicles** containing the **neurotransmitter** are released into the **synapse** to the **post-synaptic neuron** where **cells** containing **receptors** may trigger a change in the cells
- The change which occurs in the post-synaptic neuron will be either **excitatory** or **inhibitory** depending on the type of neurotransmitter involved
- If the neurotransmitter causes an electrical signal to be transmitted down the cell they are known as excitatory neurotransmitters
- Excitatory neurotransmitters increase the likelihood that the neuron will fire an **action potential**
- **Glutamate** is a key excitatory neurotransmitter which has numerous functions including gut and digestive health, memory, learning and strengthening the immune system
- Too much glutamate in the brain can lead to some **neurodegenerative** diseases such as **Alzheimer's** and **Huntington's** disease
- Artificial food additives such as **monosodium glutamate** (MSG) may contribute to specific **psychiatric** illnesses such as **psychosis, anxiety** and **depression**



Looks tasty…but watch those MSG levels!

Your notes

What are inhibitory synapses?

- During the process of **neurotransmission** small sacs called **vesicles** containing the **neurotransmitter** are released into the **synapse** to the **post-synaptic neuron** where **cells** containing **receptors** may trigger a change in the cells
- The change which occurs in the post-synaptic neuron will be either **excitatory** or **inhibitory** depending on the type of neurotransmitter involved
- If the neurotransmitter blocks an electrical signal, preventing it from being transmitted down the cell, it is known as an inhibitory neurotransmitter
- **Inhibitory neurotransmitters** decrease the likelihood that the neuron will fire an action potential
- **Gamma-aminobutyric acid (GABA)** is a key inhibitory neurotransmitter which has numerous functions including relieving anxiety, aiding concentration, improving sleep
- Practising **yoga**, meditation and relaxation exercises have been linked to increased levels of GABA

Examiner Tip

Remember that you do not need to know BOTH excitatory and inhibitory neurotransmission for the exam – the Paper 1 Section A question will be worded as

'Outline/Describe/Explain EITHER excitatory OR inhibitory neurotransmission' so you don't need to revise both.

Which studies focus on agonists & antagonists?

- **Kraal et al. (2020)** - a review article which explored research into a link between low-MSG diet and improved pain symptoms and better mental health
- **Streeter et al. (2010)** investigated whether changes in mood, anxiety, and GABA levels are linked to yoga practice

Kraal and Streeter's studies are available as separate Key Studies – just navigate the Brain and Behaviour section of this topic to find them.

Examiner Tip

Remember that you will only be asked about excitatory/inhibitory neurotransmission/synapses in Paper 1 Section A (SAQ, 9 marks) so you won't need to provide evaluation.

Yoga – good for the body, good for the mind too….

Your notes

Two Key Studies of Excitatory & Inhibitory Synapses: Streeter et al. (2010) & Kraal et al. (2020)


Your notes

Key Study 1 (Excitatory neurotransmitter): Kraal et al. (2020)

Aim: To investigate the role of **dietary glutamate** (in the form of **monosodium glutamate** (**MSG**)) in **chronic pain** and **psychiatric disorders** such as **anxiety** and **schizophrenia**.

Procedure: The researchers reviewed previously published research on the topic as part of their **review article**. Glutamate is a key **excitatory neurotransmitter** which, as a dietary source, can be found in the **food flavouring additive** MSG (which features particularly in cooking which uses a lot of soy sauce but also in parmesan cheese, meat, and many processed foods such as canned soup).

Results: The research reviewed suggests that an excess of dietary glutamate can be harmful to both physical and mental health. Too much dietary glutamate appears to be linked to ongoing chronic pain (pain which is continuous) and to a range of psychiatric disorders such as anxiety, **PTSD, OCD**, schizophrenia and **depression**.

Conclusion: Adopting a diet which is low in dietary glutamate such as MSG may lead to a decrease in chronic pain and better mental health.

Evaluation of Kraal et al. (2020)

Strengths

- The findings of this review have good **application** as they can be used to inform **preventative/intervention strategies** for patients with chronic pain and/or mental illness
- Conducting a review article means that the researcher is able to amass a lot of evidence to assess rather than relying on the results of one study alone

Limitations

- A review article uses secondary data which means that the researcher has not been able to exert any **control** over the conditions in which it was obtained which reduces the **reliability** of the findings
- It would be very difficult for policymakers such as governments or health committees to insist that people adopt a low-MSG diet which does limit the **usefulness** of the findings to some extent

Key terms:

- **Excitatory neurotransmitter**
- **Dietary glutamate**
- **Monosodium glutamate**

Key Study 2 (Inhibitory neurotransmitter): Streeter et al. (2010)


Your notes

Aim: To investigate whether yoga is linked to improved mood, decreased anxiety and GABA (an inhibitory neurotransmitter) levels.

Participants: 34 healthy adults, 19 of whom were **randomly allocated** to the yoga group and 15 to the walking group.

Procedure: The participants were instructed to either practice yoga or take walking exercise for 60 minutes at a time, three times a week across the course of 12 weeks. Mood and anxiety **scales** were taken at weeks 0, 4, 8, 12. The participants also underwent **magnetic resonance spectroscopy (MRS)** scans at each of these time intervals as well (MRS uses the same machinery as **MRI** scanning but it measures **metabolic** changes instead of structure).

Results: The participants who had engaged in yoga practice reported increased mood and a decrease in anxiety than the walking exercise group. There were **positive correlations** between improved mood and decreased anxiety and GABA levels in the **thalamus** (a structure in the brain involved in movement, sensory information, and alertness). The yoga group had positive correlations between changes in mood and changes in GABA levels i.e. as GABA levels increased so did mood.

Conclusion: Yoga may be linked to an increase in GABA which in turn may be linked to increased mood and decreased anxiety which could make it a good alternative to medication for patients with anxiety disorders.

Evaluation of Streeter et al. (2010)

Strengths

- The procedure took place over 12 weeks, which means that any variation or changes in GABA levels and mood were easy to measure and compare
- Using **objective measures** such as MRS increases the reliability of the findings

Limitations

- It is unclear as to whether the benefits experienced by the yoga group were short-term or long-term as no follow-up study was conducted
- **Individual differences** could account for the improved mood of the yoga group: perhaps this group were naturally more positive and upbeat than the walking exercise group

Key terms:

- **Inhibitory neurotransmitter**
- **GABA**
- **MRS**

Examiner Tip

There are quite a few sub-topics within the Neurotransmitters topic so make sure you have a good system for understanding which study goes with which topic. We have provided a Key Studies Summary Table (part of the Brain and Behaviour revision notes) to help you.