Institute of Psychiatry, Psychology and Neuroscience
King's College London
Cover Sheet for Assessments

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You must complete all sections of this coversheet.

K-number	21223299
Programme name	PG Cert/PG Dip/MSC Summative Assessment
Module Title and code	Psychology & Neuroscience of Affective Disorders - 7PAYFAFF
Word Count [Word limit: 1000 +10% or 7-8 minutes +10%]	1048
Selected Paper for Lay summary assignment	Kotoula et. al. (2021)

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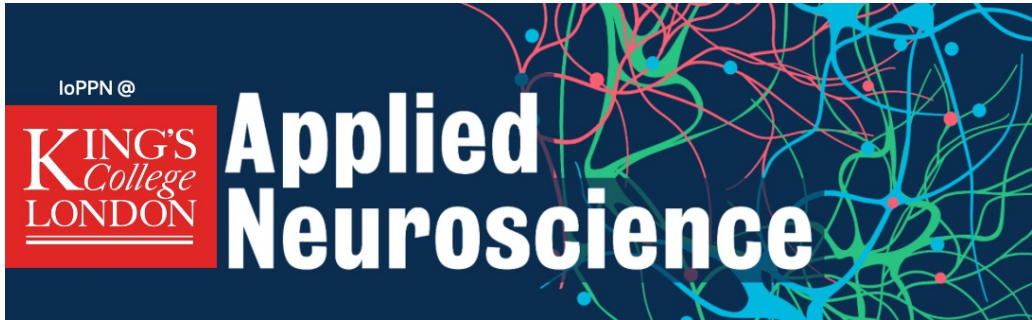
1.	Generative AI not used at all for this assessment	
2.	Generative AI used for checking spelling and grammar (but I confirm that the work submitted represents a genuine demonstration of my own skills and subject knowledge)	
3.	Generative AI used to support learning (you may select multiple options if relevant):	X
	(a) Generate ideas and/or structure suggestions	X
	(b) Assist with understanding concepts/summarising sources to aid understanding	X
	(c) Provide feedback on ideas to help improve learning/understanding	
	(d) Generate illustrative images (references should be provided for these within your main reference list)	
	(e) Generate some other aspect of the assessment which fits within appropriate use. *Please name AI tool and very brief details of how it was used here:*	

If you have an additional electronic coversheet that you need to attach, please include it after this coversheet.

Ketamine's Quick Effects on the Brain: A New Approach to Treating Depression



1. Introduction to the Research

- Key question: How does ketamine change the brain's "reward system" (the part that helps us feel happy) before people start feeling better?
- Scientists from King's College London and University College London did an interesting experiment:

> They looked at how a medicine called ketamine changes the brain very quickly;

> The study was published in 2021 in a journal that talks about brain pictures and mental health; and

> A large drug company called Johnson & Johnson paid for the study (This is important to know because it might affect how we think about the results)

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Why Is This Important?

- Depression is a big problem that affects over 264 million people worldwide.
- Most medicines for depression take a long time to work, sometimes 4-6 weeks.
- Ketamine can help people feel better in just hours, especially with enjoying things again (scientists call this "anhedonia" when people can't enjoy things).
- If we can understand how ketamine works so fast, we might be able to create even better treatments.

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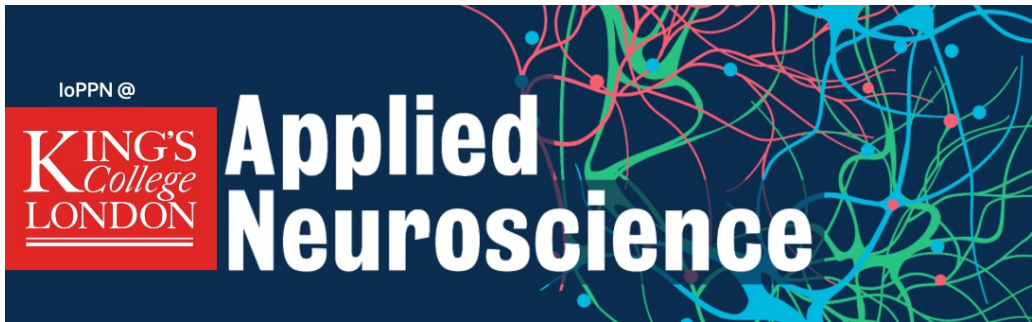
2. Summary of Key Findings



Ketamine quickly changes activity in the brain's reward areas

- These changes happen before people say they feel better
- Biggest effects in the nucleus accumbens and putamen (the brain's "pleasure centers")
- A chemical that ketamine turns into in the body, ɨor “Special K-Helper,” might be important (Kraus et al., 2023).

Why This is Beneficial:

It shows that ketamine directly affects how we process rewards:

- Might explain why ketamine works so fast to help depression; and
- Could lead to new, faster-working treatments.

[Visual: Before and after brain scans showing increased activity in reward areas]

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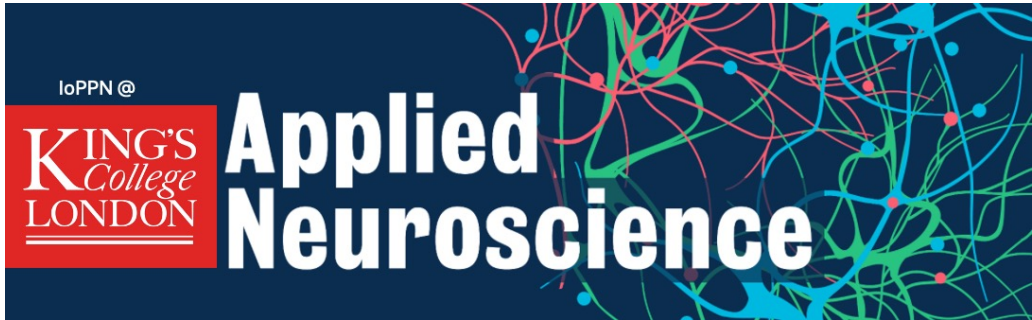
3. Methodology



Who they studied

- 37 people who used to have depression but were feeling okay now; and
- Who weren't taking any other depression medicines.

What They Did:

- Each person came for two visits, about a week apart.
- On one visit, they got ketamine through an IV (like a special drip).
- On the other visit, they got saltwater (which does nothing).
- Neither the people nor the scientists knew which was which until later.
- 2 hours after the IV, people did a special task in a brain scanner called an MRI.

The Reward Task:

- Like a computer game where you try to win money.
- Press a button really fast when you see a target.
- The scanner takes pictures of your brain while you play.

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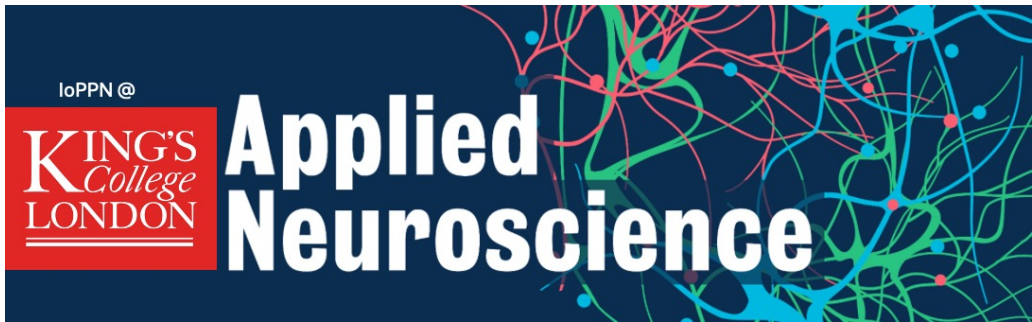
4. Results & Implications



What They Found

- Ketamine increased brain activity during the money game
- This happened when people were waiting to win and when they found out if they won
- The biggest changes were in the striatum (a part of the brain important for rewards)
- All this happened before people said they felt any better

What This Might Mean:

- Ketamine directly affects how we process rewards
- It's not just a side effect of feeling better
- This could explain why ketamine helps anhedonia (not enjoying things) so quickly
- It might "turn up the volume" on the brain's ability to experience pleasure

[Visual: Brain diagram highlighting areas with increased activity]

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5. Quality Checklist Assessment

Key points from the checklist:

- Randomization and blinding: Adequate (double-blind, crossover design)
- Participant inclusion: Comprehensive (all 37 participants included in analysis)
- Methodological rigor: High (well-controlled study with advanced brain imaging)
- Adherence to standards: Clear reporting of methods and results
- Transparency in reporting: High (all measures and analyses reported)
- Ethical considerations: Addressed (study approved by ethics committee)

Strengths:

- Well-designed study with careful controls
- Used advanced brain imaging techniques
- Looked at brain changes before mood improved

Limitations:

- Participants weren't currently depressed
- Only examined one dose, so long-term effects unknown
- Small sample size (37 participants)
- Can't definitively prove brain changes cause symptom improvement
- Reward task may not fully capture real-life reward experiences

controlled clinical trial, looks a lot like a true experiment, but people or samples are not randomly assigned to the groups so there are a lot more problems with bias creeping in.

Quality Guide:

✓ YES ✗ NO ? UNSURE **n/a** NOT APPLICABLE *delete as appropriate*

1. Did the experiment answer a clear and focused question about 'cause' and 'effect'?	✓ ✗ ? **Yes**
Comments: Kotoula's study investigated the effect of ketamine on neural correlates of reward processing in unmedicated patients in remission from depression, focusing on the causal relationship between ketamine administration and changes in brain activity.	
2. Did the authors publicly share a detailed plan or 'protocol' of how they would go about doing the study before they completed it? If they did, did they stick closely to the plan in the final study publication? **If so, please add relevant weblink for the trial protocol here:**	✓ ✗ ? **No**
Comments: No. There is no mention of a publicly shared detailed plan or protocol for the study in Kotoula's paper.	
3. Were samples and people randomly put into the different groups, e.g., the tested condition of interest versus at least the other control group/s or sample/s? And was this randomisation process explained clearly and openly in enough detail? **Comments:** The participants in Kotoula's study were randomly assigned to receive either ketamine or a placebo, and the randomisation process was clearly explained.	✓ ✗ ? **Yes**
4. Once randomly placed into the different conditions, was enough detail provided by authors to explain how the research team were prevented from knowing about the samples or people's group membership to give confidence that they were truly unaware? And did they remain unaware ideally until the end of the experiment?	✓ ✗ ? **Yes**

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QUALITY ASSESSMENTS FOR TYPES
OF STUDIES IN LAY LANGUAGE

Comments: The study employed a double-blind design where both the participants and the researchers administering the treatment and assessing the outcomes were unaware of the group assignments.	
5. Were all the samples and people who took part in the study still included in the calculations for the results and thought about in final conclusion (i.e., those who may have withdrawn, those substances that show different reaction)? And did the authors share how many of these samples or people there were and their key characteristics?	✓ ✗ ? **Yes**
Comments: Kotoula's study included all participants who completed the study in the final analysis and provided detailed information on the sample size and characteristics.	
6. If relevant, were all the people who took part in the study unaware or 'blind' to condition they were given? Related to this: **• Were the researchers 'blind' to the to the condition they were giving to the sample or people who took part in the study?** **• Were the people who do all the calculations on the study data also 'blinded' (i.e., the statisticians)?**	✓ ✗ ? **Yes**
Comments: Both participants and researchers were blinded to the treatment conditions, including those conducting data analysis.	
7. Were the samples or groups similar (e.g., for people in terms of age, gender and how severe their condition is at the start of the study?)	✓ ✗ ? **Yes**
Comments: The study groups were matched for demographic and clinical characteristics to ensure comparability at baseline.	
8. Apart from the tested conditions or manipulations being tested, were all samples or groups treated equally in every other respect?	✓ ✗ ? **Yes**
Comments: All participants were treated equally in all other respects apart from the administration of ketamine or placebo.	
9. Were the results or 'effects' of the experiment talked about in detail by the authors? Authors should say at the start what their main (or 'primary') measures and less important	✓ ✗ ? **Yes**

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('secondary') measures of interest are. Did they report on all of these measures openly and clearly?		
Comments: Kotoula's paper detailed both primary and secondary measures and discussed the results comprehensively.		
10. Were all the necessary calculations done to see if the tested condition was different when compared to the other group/s or sample/s?	√ X ?	**Yes**
Comments: The study employed appropriate statistical analyses to compare the effects of ketamine versus placebo.		
11. Can the findings be used in the real world?	√ X ?	**Yes**
Comments: Yes. The findings have potential clinical applications, particularly in the context of treating anhedonia and other depressive symptoms with ketamine.		
12. Were there any other issues or problems with the experiment in terms of bias or limitations we should know about? (please state below)	√ X ?	**No**
Comments: The study acknowledged its limitations, including the small sample size and the need for further research to confirm the findings. The study received ethical approval, and informed consent was obtained from all participants. The study did not report any conflicts of interest, and the results were reported clearly, accurately, and comprehensively, considering all relevant outcomes.		

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6. Research in Context

How Kotoula's Study Fits with Recent Research:

- A 2023 meta-analysis by Kraus et al. [2] confirms ketamine's rapid antidepressant effects and its impact on reward processing, supporting Kotoula's findings
- Abdallah et al. (2022) [3] showed ketamine also changes how different brain areas connect, complementing Kotoula's work on specific brain regions
- Study found ketamine improved anhedonia within 40 minutes, aligning with Kotoula's focus on rapid reward system changes (Nugent et al., 2019) [4]

Why This is Important:

- Highlights the crucial role of the reward system in depression
- Suggests new directions for antidepressant development
- Shows the value of studying drug effects before symptom changes
- Supports a new understanding of depression as a reward processing disorder

[Visual: Timeline comparing traditional antidepressants vs. ketamine, with recent study findings]

7. Conclusion and Future Directions

The Big Takeaway:

- Ketamine quickly changes how the brain processes rewards
- This might be why it helps depression so fast, especially with anhedonia

What's Next:

- Test in people who are currently depressed
- Look at what happens with multiple ketamine doses over time
- Connect brain changes to improvements in symptoms
- Study "Special K-Helper" ((2R,6R)-HNK) as a potential treatment on its own
- Use advanced brain imaging to see how ketamine changes brain connections

Why This Matters for the Future:

- Could lead to new depression treatments that work faster
- Might help develop medicines with fewer side effects
- Shows the importance of studying the brain's reward system in mental health.

Add reference list here

References:

1. Kotoula, V., et al. (2021). Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 7(3), 285-292.

2. Kraus, C., et al. (2023). Nature Reviews Neuroscience, 24(3), 145-164.

3. Abdallah, C. G., et al. (2022). Neuropsychopharmacology, 47(5), 1032-1041.

4. Nugent, A. C., et al. (2019). Translational Psychiatry, 9(1), 1-10.