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# PEER-REVIEW REPORT

**Name of journal:** World Journal of Psychiatry

**Manuscript NO:** 67401

**Title:** CPEB1, a novel risk gene in recent-onset schizophrenia, contributes to mitochondrial complex I defect caused by a defective provirus ERVW

**Reviewer's code:** 05115904
**Position:** Peer Reviewer
**Academic degree:** PhD
**Professional title:** Assistant Professor
**Reviewer's Country/Territory:** India
**Author's Country/Territory:** China

**Manuscript submission date:** 2021-04-25
**Reviewer chosen by:** AI Technique
**Reviewer accepted review:** 2021-06-21 03:19
**Reviewer performed review:** 2021-06-30 06:31
**Review time:** 9 Days and 3 Hours

| | |
|---|---|
| **Scientific quality** | [ ] Grade A: Excellent [ Y] Grade B: Very good [ ] Grade C: Good<br>[ ] Grade D: Fair [ ] Grade E: Do not publish |
| **Language quality** | [ ] Grade A: Priority publishing [ Y] Grade B: Minor language polishing<br>[ ] Grade C: A great deal of language polishing [ ] Grade D: Rejection |
| **Conclusion** | [ ] Accept (High priority) [ ] Accept (General priority)<br>[ Y] Minor revision [ ] Major revision [ ] Rejection |
| **Re-review** | [ Y] Yes [ ] No |
| **Peer-reviewer statements** | Peer-Review: [ Y] Anonymous [ ] Onymous<br>Conflicts-of-Interest: [ ] Yes [ Y] No |

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**SPECIFIC COMMENTS TO AUTHORS**

Summary: The authors have looked at CPEB1, NDUFV2 and NDUFVP1 as potential biomarkers for schizophrenia. The in vitro experiments are well designed that showed that ERVWe1 regulates CPEB1 proteins, which regulates NDUFV1 levels, which affects the mitochondrial complex, which causes abnormal mitochondrial functioning. This mechanism affects neurons and eventually leads to schizophrenia. I believe the manuscript may benefit from the suggestion given below. Major points: 1. Did the authors try the knockdown and over expression of CPEB1, ERVWE1 or NDUFVP1 in primary neurons, since authors have used SHSY5Y. Although SHSY5Y. is used routinely, the metabolism by mitochondria may not be close that in normal neuronal cells. 2. The potential biomarkers were looked in the blood samples of schizophrenia patients, can a confirmatory staining be done using some archived schizophrenia barin tissue samples, so that biomarkers are linked with the disease directly. 3. The discussion section of article, should ideally refraining from staining the results again. However, the current discussion section has extensively reiterated the results again. The result interpretation would be emphasized here. 4. Authors should discuss or speculate on the following points • Schizophrenia mostly sets in after the age of 20s, can the current mechanism explain this onset. • Which other factors regulate the expression of NDUFV2 other than ERVWE1 which might have impact on the current results • Does the abnormal mitochondrial activity proposed by the authors also affect the muscle cells of the schizophrenia patients • Can the schizophrenia patients have dysregulated NDUFVP1 independently of CPEB1? Minor points: Language editing: Although the manuscript is well written there are few places were correction would help. Several key sentences seem incomplete and need revision to make them clear for example: • To address novel potential risk factors and underlie the mechanisms of mitochondrial complex I

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deficiency caused by ERVWE1 in schizophrenia. • Recent studies have shown that human endogenous retroviruses (HERVs), making up about 8% of the human genome, is acted as a novel risk factor for schizophrenia