“Reduced Duration MMN in adolescents with Psychotic Symptoms: further evidence for Mismatch Negativity as a possible biomarker for vulnerability to psychosis” is an interesting study reporting on the reduction of MMN amplitude in a group of adolescents who while experiencing psychotic symptoms, did not seek clinical help and were from a community sample. This is an interesting finding suggesting early sensor deficits in a young non-clinical group.

The issues that need to be addressed are the following:

1. In the Introduction, the authors discuss MMN as being elicited independent of attention and then go on to say that its generators form a network subserving change detection and attentional switching – at the very least this is inconsistent. Furthermore, it actually is not true that MMN is free of attentional influences – thus, the statement that its generators belong to attention switching network is correct, but the idea that it is free of attentional influences is not – please rephrase that.

2. Please explain what PLE acronym refers to.

3. It is rather unfortunate that no task other than looking at a fixation point was used – in a long, passive paradigm like MMN people’s minds wonder and it is a good idea to have them do some kind of uniform task that will not engage their all attentional resources, or the MMN will be drastically reduced.

4. When one defines the latency measurement, it is a good idea to say something like: latency was defined as the most negative data point within the x-y msec latency.

5. In the statistical section, I would like to see a list of electrodes that went into each factor; from what I read, and I may be wrong – which is why I would like to have the electrodes listed as a function of a factor – some electrodes were entered twice into analysis which is incorrect. I actually quite believe that there was a real reduction in the MMN amplitude but it would be better to arrive at that conclusion with correct stats.

6. The authors found an impaired MMN and an unimpaired P3a. They conclude that this is evidence for disconnection hypothesis – it is not. The fact that one component is impaired and the other is not does not suggest disconnection – it suggests that one brain mechanism – that related to early sensory analyses and passive comparisons between a memory template and sensory evidence is
abnormal, while the other mechanism seems to be intact. The evidence that the authors cite for disconnection hypothesis from other authors indeed suggests that disconnection is one of the possible pathological changes in schizophrenia, but it has very little to do with the findings of this paper – please either omit or substantially change that argument.

7. A minor point – page 12: .. the ability to redirect attention to the novel stimulus .. is not due to the presence of a normal P3a – it is evidenced, or indexed by the a normal P3a.

8. Finally, as authors point out, it is not clear what relevance these findings have to the future development, or not, of a clinical psychosis, and future studies with larger samples that are longitudinally followed will need to address this issue.

**Level of interest:** An article whose findings are important to those with closely related research interests

**Quality of written English:** Acceptable

**Statistical review:** Yes, and I have assessed the statistics in my report.

**Declaration of competing interests:**

I have no competing interests