Dear Dr Timothy Shipley, Executive Editor of BMC Gastroenterology,

Thank you very much for the comments on our manuscript “Resolvin D1 reverses chronic pancreatitis-induced mechanical allodynia, phosphorylation of NMDA receptors, and cytokines expression in the thoracic spinal dorsal horn” (MS: 1448786458725205). According to the reviewer’s comments, we performed double immunostaining of NMDA receptor with neuronal and glial markers. We observed that NR1 and NR2B are exclusively expressed on spinal neurons but not on glia. The changes in the manuscript were indicated with red color.

We hope all these changes could work and we hope our manuscript can be accepted by your journal.

Best wishes,
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Point-by-point response to the concerns
Reviewer1:
1) Pain continues to be an enigma not only in CP but in numerous neuropathic pain disorders, and understanding its exact mechanism of generation bears major importance. In the presence of several studies reporting on the beneficial impact of several therapeutic agents on CP-related pain, it remains important to
demonstrate the cells upon which these agents act. Therefore, in my view, showing this cell type by e.g. double immunolabeling as suggested before would confer the study a very high quality. In particular, the authors should make use 1) of a neuronal marker, 2) of a microglial marker, and 3) of an astrocyte marker to show the site of RvD1 action.

Response: Thanks for your good suggestion. We performed double immunostaining of NMDA receptor with neuronal and glial markers. The data were present as Fig. 5. We observed that NR1 and NR2B are exclusively expressed on spinal neurons but not on glia. These data suggest that RvD1 could modulate neuronal NMDA receptor activities, via direct or indirect actions.