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Footshock stress induces freezing behavior and interleukin-1beta expression in the medial prefrontal cortex, amygdala, and hippocampus during situational reminder: a posttraumatic stress animal model test

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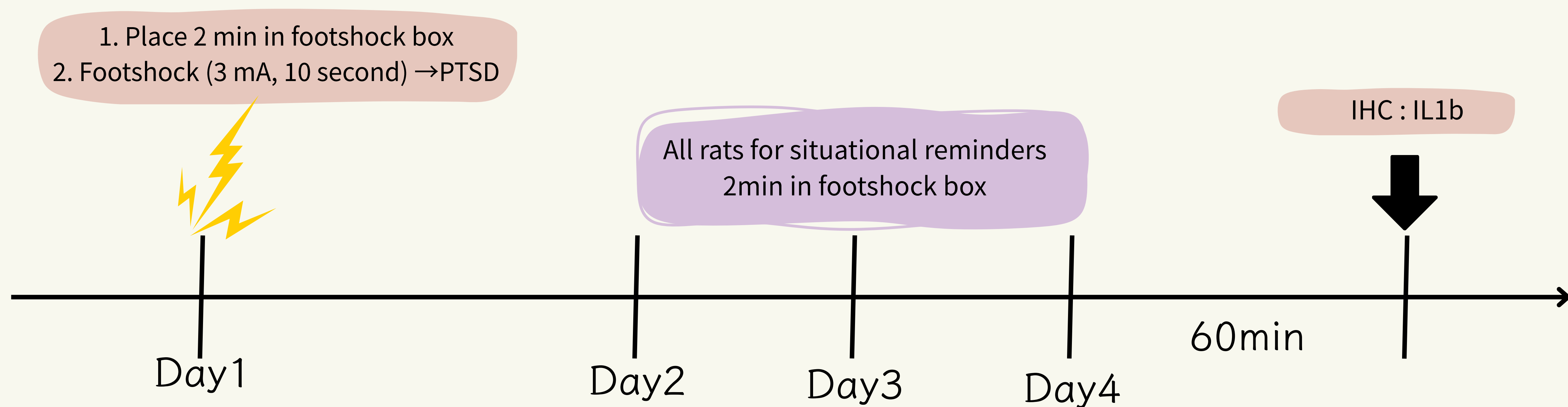
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ABSTRACT



A growing body of evidence showed that post-traumatic stress disorder (PTSD) symptoms might induce neuroinflammation responses in brain. However, no animals research examined this issue in IL-1b expression in the specific brain areas. The present study used the animal model that footshock (3m, 10 second) treatments were conditioned with the contextual stimulus. Later, all rats were subjected to those rats exposed in the footshock box for two minutes over three times once a day. The freezing behaviors were tested in each session. Finally, the neuroinflammation proteins, interleukin-1beta (IL-1b), was assessed with the immunohistochemical staining. The results showed that footshock treatments induced severe freezing responses from session 1 to session3. IL-1b expression was not significant differences in the sub-areas of the medial prefrontal cortex [e.g., cingulate cortex 1 (Cg1) and prelimbic cortex (PrL)], the amygdala [e.g., basolateral amygdala (BLA) and central amygdala (CeA)], and the hippocampus [e.g, CA1, CA3, and dentate gyrus (DG)]. These data might be inconsistent with our anticipatory results. The data need to be discussed in the further research.

EXPERIENCE DESIGN



RESULTS

