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IL-1β pathway-dependent regulation of glutamate receptor activity by gut microbiota in bipolar depression

Key words: Bipolar disorder; Gut microbiota; Neuroinflammation; Glutamate receptor; Interleukin-1 (IL-1)

Research Summary

Our findings suggest that during depressive episodes in BD, alterations in intestinal flora may be associated with neuroinflammation and changes in the expression of glutamate receptor (NMDAR and AMPAR) genes in the prefrontal lobe, potentially involving an IL-1β-dependent pathway.

- Gut microbiota derived from BD patients can induce bipolar depression-like behaviors in mice.
- The increased level of IL-1β may be a key pathway of the brain-gut axis in BD.
- Altered IL-1β mediate the changes of transcription of glutamate receptor genes in BD.

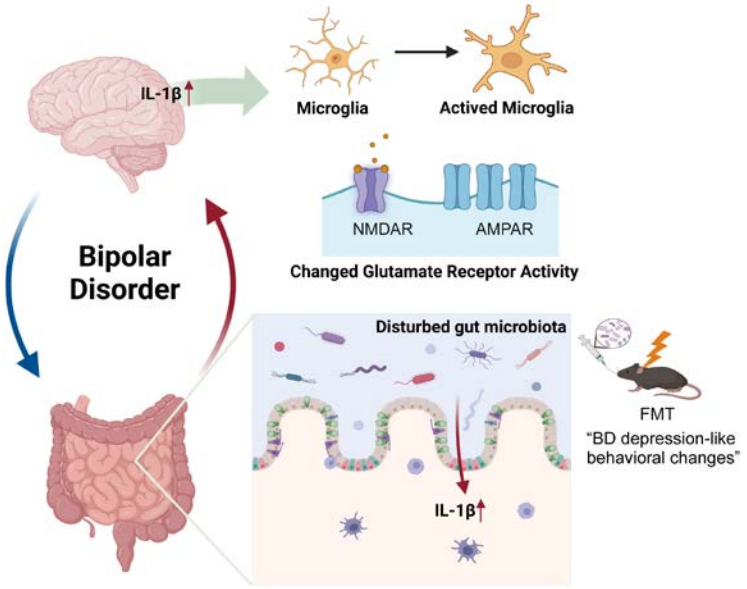
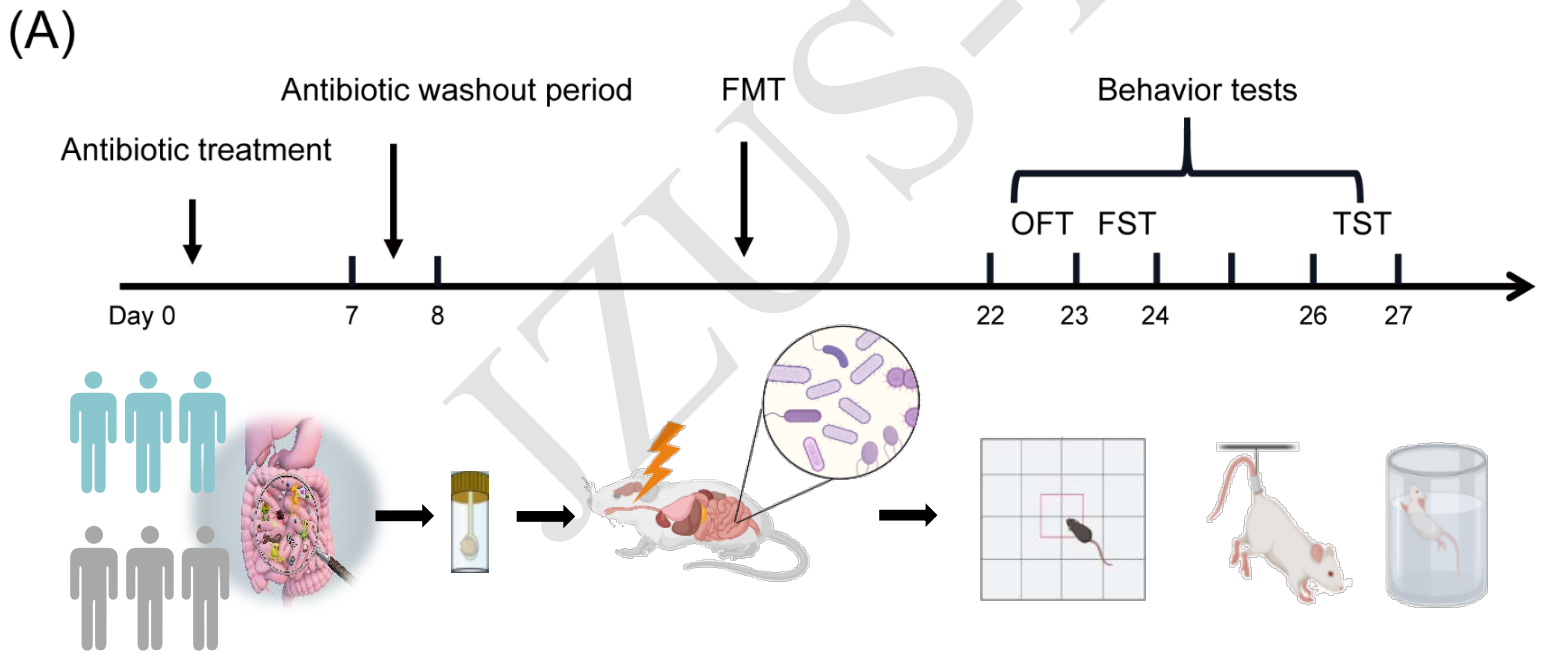


Figure 6

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Innovation points

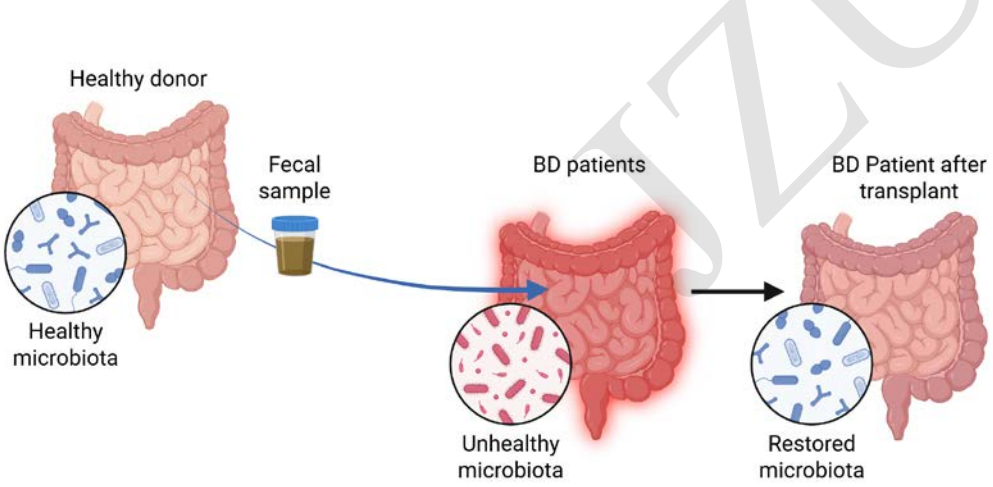
Model Innovation: An FMT-induced Humanized Mouse Model for Bipolar Depression

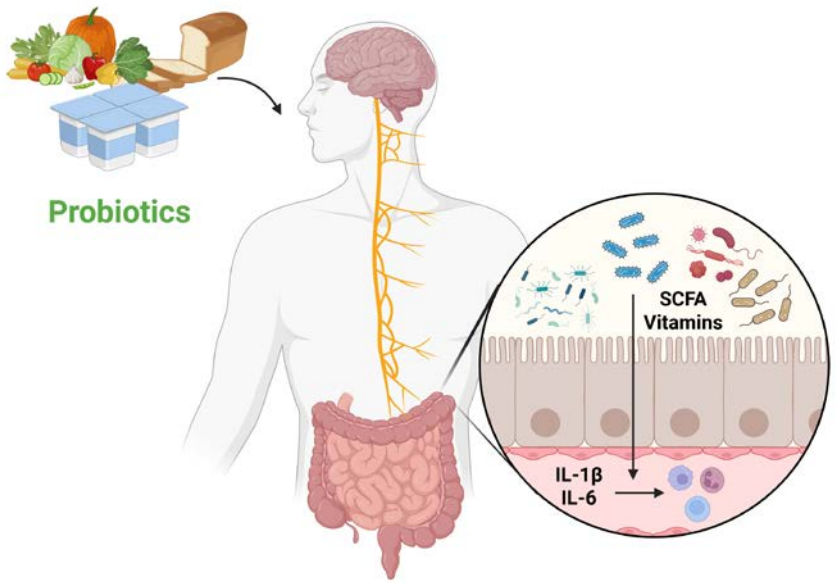


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Innovation points

- **Revealing a gut-brain axis mechanism specific to depressive episodes in Bipolar Disorder.**
- **Identifying neuroinflammation (IL-1β) as a central hub in this pathway.**
- **Providing evidence for the "Microbiota-Immune-Neural" interaction in BD.**
- **Offering a scientific rationale for novel immune-modulating or microbiota-targeted therapies for BD depression.**





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