mg/kg/day.

In a SCID mouse infection model inoculated with an influenza virus A (H3N2), therapeutic effect was observed by a 14-day oral administration of favipiravir with a dose of 30 mg/kg/day.

3. Mechanism of action[14, 20]

It is considered that favipiravir is metabolized in cells to a ribosyl triphosphate form (favipiravir RTP) and that favipiravir RTP selectively inhibits RNA polymerase involved in influenza viral replication. With regards to the activity against human DNA polymerases α, β and γ, favipiravir RTP (1000 μmol/L) showed no inhibitory effect on α, 9.1-13.5% inhibitory effect on β and 11.7-41.2% inhibitory effect on γ. Inhibitory concentration (IC_{50}) of favipiravir RTP on human RNA polymerase II was 905 μmol/L.

4. Resistance[14]

No change of susceptibility of type A influenza viruses to favipiravir was observed after 30 passages in the presence of favipiravir, and no resistant viruses have been selected. In clinical studies including the global phase III study, information about emergence of favipiravir-resistant influenza viruses has not been obtained.

PHYSICOCHEMISTRY

Nonproprietary name: Favipiravir
Chemical name: 6-Fluoro-3-hydroxypyrazine-2-carboxamide
Structure formula:

12. In-house document (*In vivo* kinetics/animal)
13. In-house document (Study of combination therapy with oseltamivir)
14. Takahashi K, et al. Jpn J Med Pharm Sci. 2011;66:429.
15. In-house document (Antiviral activity and cross resistance)
16. Ito Y, et al. Nature. 2009;460:1021.
17. Watanabe T, et al. Nature. 2013;501:551.
18. In-house document (Therapeutic effect/mice)
19. In-house document (Therapeutic effect/immune-deficient mice)
20. Furuta Y, et al. Antimicrob. Agents Chemother. 2005;49:981.

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