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Favipiravir: An Effective Rna Polymerase Modulating Anti-Influenza Drug

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Combination of all the aspects are covered in this review from anti-influenza drug.Several articles neglected to include a point of view. The majority of review papers emphasiseon epidemiology. In order to lessen the financial burden of disease and its negative effects, it is important to raise awareness about disease and how to manage it.The medicine favipiravir,additionally called T-705 (5-fluro-2-oxo-1H-pyrazine-3-carboxamide), by selection and powerfully inhibits the polymer-dependent polymer accelerator (RdRp) of polymer viruses. Favipiravir was discovred by the company Toyama Chemical Co., Ltd.By searching a chemical library for the respiratory illness virus's antiviral properties. Favipiravir-RTP (favipiravir ribofuranosyl-5'-triphosphate), the active version of the drug, is recognised by RdRpas a substrate and restrains the polymer accelerator's activityFavipiravir is efficient against a broad kind of subtypes and variations of respiratory disorder viruses.

Keywords: Antiviral Activity; Favipiravir; Polymer Dependent Polymer Acecelerator(RdRp); Respiratory disorder viruses.

The influenza virus causes one of the most common illnesses, influenza, which has an annual epidemic worldwide. The A(H5N1) extremely dangerous avian influenza virus was identified in 1997, in people, in Hong Kong and continues to cause outbreaks[1]. In 2013, China experienced avian influenza A(H7N9)[2] and the 2009 H1N1 influenza pandemic, which killed 17,700 people in that year[3] A substantial rate of resistance to amantadine, an M2 protein inhibitor, was observed following treatment. Due to the lack of diversity in the MOA of the currently existing anti-influenza medicines, itsnecessitate to find outa anti-influenza medicine withnew mechanism action. The world is currently being invaded by viruses spread by arthropods or wild animals. 2014's Western African Ebola and Lassa virus outbreaks sparked public anxiety about prevention and treatment in the event that they do[4,5] These viruses are extremely dangerous and can result in deadly illnesses[6] A pyrazine analogue that was initially tested for anti-influenza viral action in cells led to the discovery of favipiravir by chemical modification. The RNA polymerase of the influenza virus is effectively and specifically inhibited by the drug favipiravir[7] as well as efficient against each subtype which includes strain influenza

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viruses, such as those responsive to or resistant to the commercially available M2 inhibitors and neuraminidase. In tests with different RNA viruses, favipiravir showed antiviral properties[8]

Mechanism Action Of Favipiravir

Favipiravir's antiviral activity was diminished in presence of purine bases or nucleosides, demonstrating that pyrimidine nucleosides are not favipiravir's main competitors. It is possible to test the influenza virus *invitro* using MADIN DARBY CANINE KIDNEY (MDCK) cells. Favipiravir was administered to MDCK cells, and HPLC was used to examine cellular metabolites. There were three different favipiravir compounds found: favipiravir ribofuranosyl-52 -monophosphate (FAVIPIRAVIR-RMP), favipiravir ribofuranosyl-52 -triphosphate (FAVIPIRAVIR-RTP) and favipiravir ribofuranose (FAVIPIRAVIR-R), These findings imply that after being absorbed into cells, favipiravir is activated. Chemically produced FAVIPIRAVIR-RTP was examined to see if it could stop the influenza virus's RNA polymerase activity by incorporating P-GTP. From nanomolar to the micromolar concentrations of favipiravir-RTP blocked functioning of viral RNA polymerase[7] FAVIPIRAVIR-RTP interacts with the RdRp molecule, however the exact mechanism of this connection is yet unclear. Nucleotides required for viral RNA replication and transcription are expected to be hindered by favipiravir by either misincorporating while a viral RNA is forming or by attaching to conserved polymerase domains.In *invitro* large quantity of infection at high (10 PFU/cell) or low (0.0001 PFU/cell), favipiravir caused nfluenza virus infection-related fatal mutagenesis and decreased viral titer. Sequence examination of several nucleoprotein (NP) clones showed a rise in number of identifiable C–T and G–A as well as C–U transition mutations, and it should beenhance in alteration rate and transform in a NP gene's nucleotide profiles concurrently studied from different clones [9,10] A number of RNA viruses may participate in mutagenesis as a result of favipiravir's virucidal activities, according to earlier investigations.As a source for RdRp and a template for viral RNAs in a primer extension test, we used influenza H1N1 extracts. Cap-snatching and transcription were induced by adding 52 Cap1 RNA to the test mixture. FAVIPIRAVIR-RTP, the particular moleculehas been integrated in the developing RNA in presence of the compound, and the strand extension was stopped.[11] A guanosine analogue with antiviral properties, ribavirin is useful against multiple RNA viruses. In order to create ribavirin monophosphate and its triphosphate form, ribavirin goes through phosphorylation in cells. Despite the fact that ribavirin's reported numerous modes of action[12] IMPDH (INOSINE MONOPHOSPHATE DEHYDROGENASE) inhibits by ribavirin-MP, which results decrease in guanosine triphosphate (GTP) levels, which is the most well-known[13] Favipiravir-RMP and ribavirin-MP were tested for their ability to inhibit IMPDH; their respective 50% inhibitory concentrations (IC50) were 601 and 3.9 mol/L [7]When MDCK cells are treated with ribavirin-MP instead of favipiravir-RMP, the amount of GTP was substantially lower [14] For humans and viruses to survive, nucleic acid synthesis is necessary. Humans lack RdRp, unlike RNA viruses, but do having a DNA-dependentRNApolymerease (DdRp) and DNA-dependentDNA polymerase (DdDp). The ability of Favi-RTP to suppress certain polymerase activities was examined[15] It is possible to use favipiravir in conjunction with other antivirals, like as oseltamivir, because it targets the influenza RdRP. Animal studies have demonstrated that the two medications work in synergy.[16] These results are consistent with the conclusion that favipiravir did not stop DNA and RNA synthesis in MDCK cells at 636 mol/l.[7] Mechanism of action as shown in the figures.

Effects on influenza virus

Favipiravir has shown antiviral activity against all influenza virus strain subtypes, such as type A, B, and C, in tests utilising influenza virus strains from laboratories with 50% effective concentrations (EC_{50}) varying from 0.014 to 0.55 g/m[17] influenza viruses A like A(H1N1)pdm09, A(H1N1), A(H3N2) and B seasonal strains of influenza, as well as the extremely contagious AVIAN INFLUENZA VIRUS A(H5N1) is recovered in humans, were tested *invitro* to see if avipiravir could stop the spread of these viruses. There are some of these strains that are resistant to both NA inhibitors as well as strains that are resistant to oseltamivir or zanamivir. It should be highlighted that all of the tested strains were resistant to favipiravir's antiviral effects[18]

Favipiravir has a good antiviral index and did not cause cytotoxicity in MDCK cells at 50% cytotoxic concentrations (CC50) of >1000 g/mL[17]

Efficacy of Favipiravir In Other Viruses

An elevated case fatality rate is caused by hemorrhagic fever and/or encephalitis caused by the RNA viruses arena, bunya, flavi, and filoviruses. These viral illnesses do not have vaccinations or licenced anti-viral treatments, highlighting the critical need for efficient broad spectrum anti-virals. For arenaviral hemorrhagic fever, ribavirin is a only medication that works and is used outside of its intended usage. On a comparison with prior research, its therapeutic effects are determined.[19]

Arenaviridae

Numerous arenaviruses are known to lead to serious illnesses in humans[20] Ribavirin is a only medication that has been licenced for treatment of the arenavirus infections despite toxicity concern. Favipiravir and ribavirin were examined for their In *invitro* effectiveness against pathogenic arenaviruses.The EC_{50} values for Junin virus, (PICV) and Tacaribe virus in a cytopathic effect (CPE) experiment employing Vero cells ranged from 0.75 to 0.90 g/mL.. The (EC_{90}) valuefinds next tothe Lassa virus (LASV) in a virus yield reduction experiment day 3 and 5 in post-infection with Vero cells were 1.7and 11.1 g/mL, respectively[21] Favipiravir was administered orally to PICV-infected hamsters for seven days, starting four hours after infection. This treatment prevented fatalities, decreased in blood and tissue viral loads, and prevented liver damage[22] Favipiravir 98.5 mg/kg/day b.i.d. used to seven days was administered to hamsters with the PICV infection from 4 to 6 days after viral infection notably enhanced the endurance rate (P 0.001)[23] The use of oral favipiravir in guinea pig models of PICV infection shown therapeutic effectiveness., even after symptoms appeared[24] The effectiveness of oral favipiravir in LASV infection models in mice and guinea pigs was recently demonstrated[21,25] In addition, favipiravir has been shown higher selectivity for tested viruses than ribavirin

Bunyaviridae

The Bunyaviridae family of viruses includes the hantavirus, La Crosse virus, Rift Valley fever virus, Crimean-Congo hemorrhagic fever virus, Severe fever thrombocytopenia syndrome virus, and Rift Valley hemorrhagic fever virus. The disorders caused by these viruses include hemorrhagic fever, fever with thrombocytopenia, fever with renal or pulmonary syndromes, and others., and other dangerous conditions., and *Invitro* research, this drug outperformed ribavirin in terms of the strength and specificity of its antiviral activity against these type of viruses in Vero cells[26,27,28] Favipiravir, taken orally twice daily, prevented death, decreased in blood and tissue viral loads, and controlled hepatic lesion in PTV infectivity models in a mice and hamsters [22,26] When the dosage started two days after infection, survival rates of mice with CCHFV infection were improved by favipiravir b.i.d[29] RVFV-infected hamsters were treated with oral favipiravir twice daily, which prevented fatalities and reduced virus titers in serum and tissues[30] SFTSV, a newly discovered virus found in China, Korea, and Japan, causes the developing viral disease known as severe fever thrombocytopenia syndrome (SFTS)[31,32] SFTSV has a year-round seasonality, however spring to fall is when it occurs more frequently. FFU reduction experiments employing Vero cells revealed that favipiravir prevented SFTSV replication with EC_{50} values of the 0.71–1.3 g/mL[28] IN MICE LACKING INTERFERON ALPHA RECEPTORS (IFNAR/), the therapeutic effect of favipiravir were shown in an infection model with SFTSV. as opposed to starting Compared to the placebo group, there were significantly increased endurance rates between 4 and 5 days after infection (P 0.03 and P 0.05, respectively).Favipiravir oral therapy at 300 mg/kg one time a day kept all mice alive (P 0.001) for the 5 days starting 2-3 days of after the infection. Clinical research trials on the SFTS has started intoJapan as a result of the findings of this pre-clinical investigation[33]

Flaviviridae

The WEST NILE VIRUS (WNV) and the YELLOW FEVER VIRUS (YFV) were just two of the harmful flaviviruses that favipiravir stopped from reproducing[34,35] to achieve flavivirus effectiveness comparable to influenza virus, higher favipiravir concentrations were required. In yield-reduction test utilisingthe Vero cells, favipiravir's EC_{90} for YFV was 51.9 g/ml[34] When Treatment started four hours before to infection, oral administration of favipiravir at range of doses 210-410 mg/kg/day intended to eight days dramatically decreases the mortality rate in hamsters with YFV

infection[34] Initiating oral favipiravir therapy at dose of 400 mg/kg/day within a two days of the infection resulted in complete protection. The antiviral effectiveness of favipiravir against WNV was shown in both*in vitro* and *in vivo*[35] With EC_{50} at 53 g/mL in a Vero cells, favipiravir prevented WNV virus from proliferating. When given orally to WNV-infected mice, favipiravir 398.5mg/kg/day b.i.d., starts for four hours post-subcutaneous infection, prevented 9/10 from dying (p 0.01) andreduces six days after infection the viral proteins and RNA synthesis in brain areas.. In the second species, same efficacy was shown. 400 mg/kg/day administered orally to hamsters starting 4 hours after the subcutaneous infection.As Similar to mice, drug favipiravir drastically increased survival rate (p 0.01).That result should be noted on day 7 after infection, no evidence of WNV's covering protein was found in brain of hamsters given favipiravir.The zika virus (ZIKV) is newly discovered flaviviridae arbovirus that is primarily spread by mosquito bites. As of now, the evidence points to a connection between prenatal infection and microcephaly. The ZIKV infection may be treated with favipiravir, which suppressed ZIKV replication in a Vero cells ranges of EC_{50} of 3.4-3.7 g/ml[34]

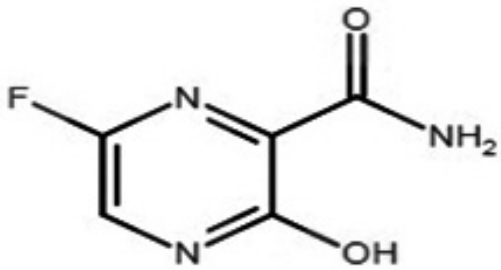

Fig. 1. Structure Of Favipiravir

Togaviridae

Favipiravir demonstrated antiviral efficacy against theWESTERN EQUINE ENCEPHALITIS VIRUS (WEEV) grown into Vero cells with EC_{90} at 47 g/ml[36]Favipiravir was administered orally to WEEV-infected mice for seven days starting

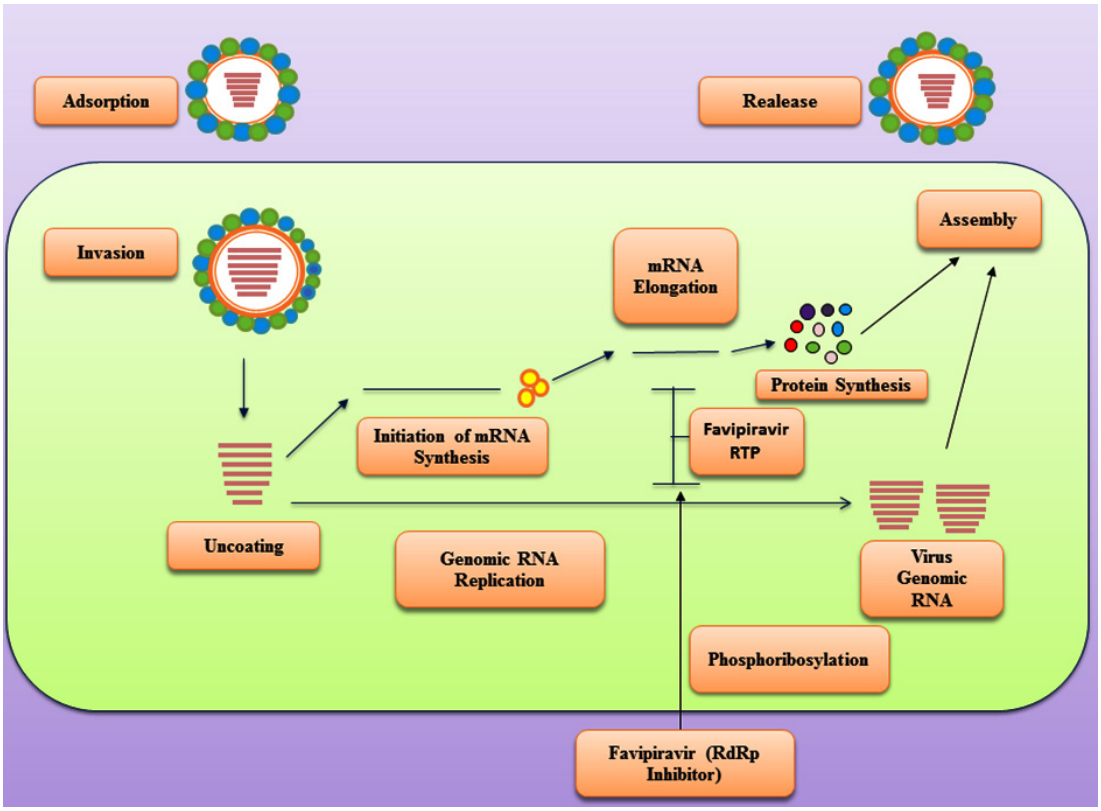

Fig. 2. Mechanism Action Of Favipiravir

four hours before infection. This treatment significantly higher rates of survival (p 0.01) as well as lengthened duration of thefatality. At Day 4 post-infection, the infection level in the brain reduced to one tenth, but there was no statistically significant difference. Favipiravir did not eradicate the disease's mildly manifested symptoms.

With EC_{50} of 0.3-9.4 g/mL, favipiravir showed antiviral efficacy against the CHIKUNGUNYA VIRUS (CHIKV) in aVero cells.When given orally to CHIKV-infected mice, favipiravir 299 mg/kg/day b.i.d. starts 1 dayearlier than or four hours after the infectivity increased survival rates[37]

Picornaviridae

Favipiravir suppressed FOOT AND MOUTH DISEASE VIRUS (FMD), replication In *invitro* at EC_{50} of 12 g/ml[38]Favipiravir inhibited poliovirus replication in the Vero cells and rhinovirus replication with EC_{50} of 4.7 and 24 g/mL and selected indices is 28 and >42correspondingly[17] Favipiravir had an EC_{50} of 23 g/mL and prevented enterovirus replication[39]

Caliciviridae

Utilizing RAW 264.7 murine leukaemia macrophage cells for the CPE assay, favipiravir proved effective against a murine norovirus at EC_{50} with 39 g/mL. Favipiravir had an EC_{50} of 19 g/mL, according to real-time PCR, which showed that it prevented the synthesis of viral RNA [40] Oral administration of the favipiravir at the dose of 500 mg/kg/day b.i.d. intended for eight weeks and it starts four weeks after the viralinfectivity effectively reduced the relative amount of norovirus antigen positive animals and the viral titer in the faeces in mouse model of persistent norovirus infection[41] TheActivity of RNA polymerase for the Human Norovirus was shown to be reduced by favipiravir-RTP[42]

Filoviridae

In Vero E6 cells, favipiravir demonstrated antiviral effectiveness against the Zaire Ebola virus (Mayinga 1976 strain) with EC_{50} of 10.7 g/mL. IFNAR/C57BL/6 mice which are infected with the Mayinga virus strain had a higher survival rate than the placebo group, oral administration of this drug prevented every fatalities as well as reduced viral titers from the blood for 8 days starting six days after infection [43] Similar to this, oral administration of favipiravir to IFNAR/A129 mice infected with the E718 strain for 14 days, starting an hour after infection, completely prevented infected mice from dying[44] Western Africa saw an EVD outbreak in 2014. Guinea's government and French Institute of

Table 1. *Invitro* impact of Favipiravir anad Ribavirin on Arenavirus inhibition[68]

Virus	Strain	Favipiravir			Ribavirin		
		CC_{50}	EC_{50}	SI	CC_{50}	EC_{50}	SI
JUNV	Candid1	185	0.75	234	48	2.5	16
PICV	An 4763	170	0.90	180	35	3.0	10
TCRV	TRVL11573	210	0.90	225	65	2.2	25

Table 2. *Invitro* impact of Favipiravir anad Ribavirin on Bunyaviruses inhibition[69,70]

Virus	Strain	Favipiravir			Ribavirin		
		CC_{50}	EC_{50}	SI	CC_{50}	EC_{50}	SI
LACV	-	>1000	4.8	>195	870	15	50
PTV	Adames	>1000	28	>32	897	40	20
RVFV	MP-12	>975	4.8	>195	>902	12	>68
SFNV	Naples	>1000	15	>54	>728	20	>32
DOBV	Sotkamo	755	12	50	295	17	16
MPRLV	HV9021050	748	10	72	255	10	20
PHV	MP40	599	10	57	245	5.5	42
SFTSV	SPL010	>156	0.8	>165	>155	7.6	>19

Table 3. Antiviral Activity of Favipiravir on other Viruses

Group	Family	Virus	EC_{50} (µg/mL)	References
RNA (-) Strand	Orthomyxoviridae	Influenza A (seasonal)	0.01–0.92	Shiraki, Kimiyasu et.al.2020[17]
		Influenza A (H5N1)	0.2–1.8	£agocka, Ryta et.al. 2021[57]
		Influenza A (H1N1)pdm09	0.12–3.51	Goldhill, Daniel H et.al.2021[18]
		Influenza A (H7N9)	1.3	Hayden, Frederick et.al. 2019[58]
		Influenza B	0.04–0.9	Shiraki, Kimiyasu et.al.2020[17]
	Bunyaviridae	Influenza CLa Crosse	0.03 -0.055	Shiraki, Kimiyasu et.al.2020[17]Komeno, Takashi, et al. 2022[22]
		Crimean-Congo hemorrhagic fever	1.2	Mirza, Muhammad Usman et.al. 2019[29]
		Severe fever thrombocytopenia syndrome	0.70–1.2	Li, Hao, et. al.2021[28]
		Rift Valley fever	4.1–5.0	Komeno, Takashi, et al. 2022[22]
	Arenaviridae	Lassa	1.5–11.2 (EC_{90})	Hansen, Frederick et.al. 2021[21]
		Tacaribe	0.8–4.3	Komeno, Takashi, et al. 2022[22]
	Filoviridae	Ebola	10.5	Jain, Mayur et.al. 2021[43]
	Rhabdoviridae	Rabies	5.1–7.1	Kimitsuki, Kazunori, et. al.2023[48]
	Paramyxoviridae	Human metapneumovirus	1.4–6.4 (EC_{90})	Dawes, Brian et.al. 2018[59]
RNA (+) strand	Flaviviridae	West Nile	52	Pérez-Pérez, María-Jesuìs, et. al.2021[35]
		Yellow fever	41	Gawriljuk, Victor et.al.2021[34]
		Zika virus	3.6–3.8	Eyer, Ludìk, et al. 2019 [60]
	Togaviridae	Western equine encephalitis	1.1, 48 (EC_{90})	Kovacikova, Kristina et.al. 2021[37]
		Eastern equine encephalitis	2.8	Kovacikova, Kristina et.al. 2021[37]
		Chikungunya	0.21–9.5	Kovacikova, Kristina et.al. 2021[37]
	Picornaviridae	Polio	4.7	Shiraki, Kimiyasu et.al.2020[17]
		Rhino	23	Shiraki, Kimiyasu et.al.2020[17]
		Enterovirus 71	23	Lanko, Kristina et.al. 2021[39]
	Caliciviridae	Noro	20–40	Ibrahim, N et.al. 2021[40]

Health and Medical Research (INSERM) conducts a medical trial using favipiravir (JIKI study)[45] With regard to low viral loads (cycle threshold 20), The toleration of favipiravir was good. and showed a tendency to reduce patient mortality[46] In EVD patients in Sierra Leone, favipiravir significantly (p 0.05) increased survival rates and decreased viral loads, according to a recent Chinese study[47]

Rhabdoviridae

Recently, it was discovered that favipiravir has action against the rabies virus in a murine neuroblastoma Neuro-2a cells, at EC_{50} values of 5.0-7.0 g/ml[48]When oral administration of the favipiravir was started 1 hour after RABV infection and continued for 7 days at dose of 300 mg/kg/day b.i.d., it dramatically reduced the morbidity and death ofthe virus-infected mice. When given after symptoms developed, favipiravir had no impact.These findings imply that favipiravir has a broad antiviral spectrum among RNA viruses and that it is effective in treating viral infections that are particularly resistant to treatment. The large anti-viral range of interferon and ribavirin makes them potentially useful medications, but their usage is constrained by their crippling adverse effects. Favipiravir, in contrast to these medications, was well tolerated in clinical tests.

Anti-viral Activity of Favipiravir

Antiviral activity of favipraviron various viruses are shown in the table

The Broad-spectrum Anti-rna Viral Action Of Favipiravir

This drug is useful against these infections in animal models and demonstrates a range of action against RNA viruses, including deadly RNA virusess. outlines the effectiveness of favipiravir in infections modelled to those found in humans. It has been utilised to treat human diseases including Ebola virus infection based on its success in animal models [46,47,49]Lassa fever [50] norovirus [51] and rabies [52] Notably, favipiravir has been proposed for new SFTS indications in Japan as a alarge range of anti-RNA viral drugs which is based on clinical investigations [53]Review of favipiravir's broad range of Animal models with anti-RNA virus action *in vivo* and in*in vitro*, as well as other anti-RNA virus mechanisms, has been done [8,38,54,55]

Favipiravir Dosage For Treating Ebola and Influenza Infections is as Follows

Favipiravir's antiviral action that is (EC_{50}) beside the Ebola viruses as well as influenza differs from range 0.014-0.55 g/mL and 10 g/mL, respectively, in medium lacking adenosine and guanosine[17,43] When the nucleoside analogues like adenine, adenosine, guanine, guanosine, and inosine are introduced toanalyze at 10 times the EC_{50} (63.7 M), favipiravir's anti-influenza virus activity is rendered ineffective[7] ATP is present intracellularly in a range of tissues between 1 and 9 mM[56] indicating that favipiravir might require a larger concentration in the body.Pharmacokinetic values for favipiravir are 60 g/mL, 440–550 g hr/mL, 1 h, and 4.7–5.6 h. for theMaximum drug concentration (Cmax), area under the curve (AUC), maximum drug concentration time (Tmax), and half-life (t1/2), in that order (Avigan Tablets 200 mg package insert).By the recommendation of these drug in Japan for 1600mg two times daily are recommended for influenzaand 600 mg two times daily for 4 days, whereas5000 mg on day 1 and 2300 mg daily for 2 days of an Ebola infection 1 through 9. In case of the Ebola virus disease, target tissues, including hepatocytes vascular endothelial cells and hematopoietic cell get direct blood perfusion. In influenza, favipiravir's antiviral potency is attained in the lung's epithelium by the dispersion from the blood.The target cells and antiviral concentrations, which are two parameters, do not appear to have a significant impact on the dosage.It is need to improve favipiravir oral administration, intravenous formulations are being developed.

DISCUSSION

Previous research suggested that favipiravir-resistant influenza viruses were unlikely to develop, and numerous passaging trials yielded no resistant infections[61,62] According to Pauly and Lauring, there is only a small window of time in which resistance to nucleoside analogues can develop. In addition, they contend that using too much of such a medicine will cause deadly mutagenesis and population extinction.[63]. According to our hypothesis, the viral population in our study was kept at an appropriate level while selection pressure was maintained by ongoing exposure to favipiravir. Importantly, we discovered that two influenza A virus mutations combined For effective resistance, a viral RNA polymerase was

required. The main alteration was K229R in the PB1 subunit, which prohibited polymerases from the H1N1, H3N2, and H7N9 influenza A strains from incorporating favipiravir into nascent viral RNA viruses although at a cost to RNA polymerase activty in a cell culture assays. The PA subunit's P653L mutation helped to offset this expense. The mutagenetic effect of favipiravir in the setting of infectious viruses was reduced by PB1 K229R at the expense of growth, and this effect may be mitigated by PA P653L. The two mutations combined to create a virus that was 30 times less sensitive to favipiravir than the wild-type virus was while maintaining normal replication kinetics. Despite the fact that many nucleoside analogues have pleiotropic effects on influenza and other viruses, most mutations that have been documented to provide resistance to ribavirin or other nucleoside analogues have increased the virus's fidelity[64,65].We found that neither the polymerase fidelity nor the ribavirin resistance was increased by the K229R mutation. This further implies that favipiravir acts differently from ribavirin, which may assist to explain the synergistic impact of favipiravir and ribavirin when they are used in combination.[66,67]

CONCLUSION

At the Toyama Chemical Co., Ltd. Research Laboratories, phenotypic screening against the influenza virus led to the discovery of favipiravir. We conducted additional research to better understand the actions and antiviral effects of diverse viruses. The active form of favipiravir, favipiravir-RTP, is phosphoribosylated in cells where it is then recognised by RdRp as a purine nucleotide and it can beinhibits the activity of the RdRp enzyme.DNA-dependent RNA or DNA polymerases are unaffected by favipiravir-RTP. These individuals expound on how favipiravir prefers RNA viruses to DNA viruses and mammalian cells. No matter how susceptible or resistant to current anti-influenza medications a virus may be, this drug is efficient against a variety of influenza viruses. It is noteworthy that both *invitro* and *invivo,* favipiravir is helpfulagainst a broad spectrum of different type of RNA viruses. Studies conducted In *invitro* show that favipiravir is not becoming resistant to use. In Japan and the US, favipiravir has been given approval to treat influenza infection. Favipiravir is now well tolerated in humans, according to data from clinical studies. We pursued clinical research for fatal viral illnesses like Ebola and SFTS because of the favipiravir's broad antiviral spectrum. With these distinct features, we are certain that favipiavir will soon be a potent therapeutic treatment for RNA virus infections that have not yet been treated.

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Conflict of Interest

There are no conflict of interest

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