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Original Research Article

A Comprehensive Covid–19 Research Registry

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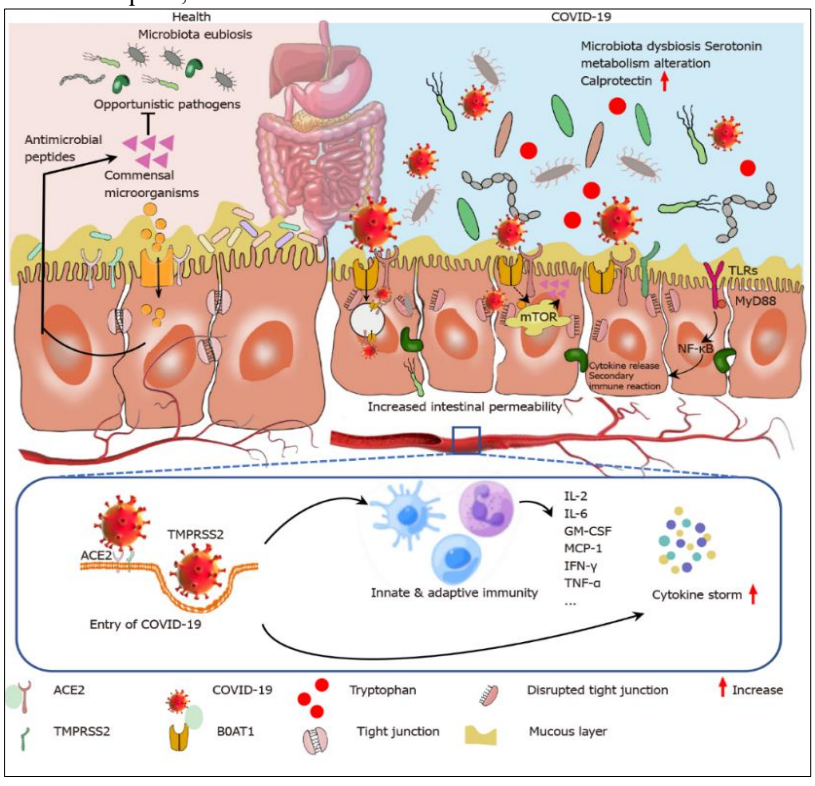
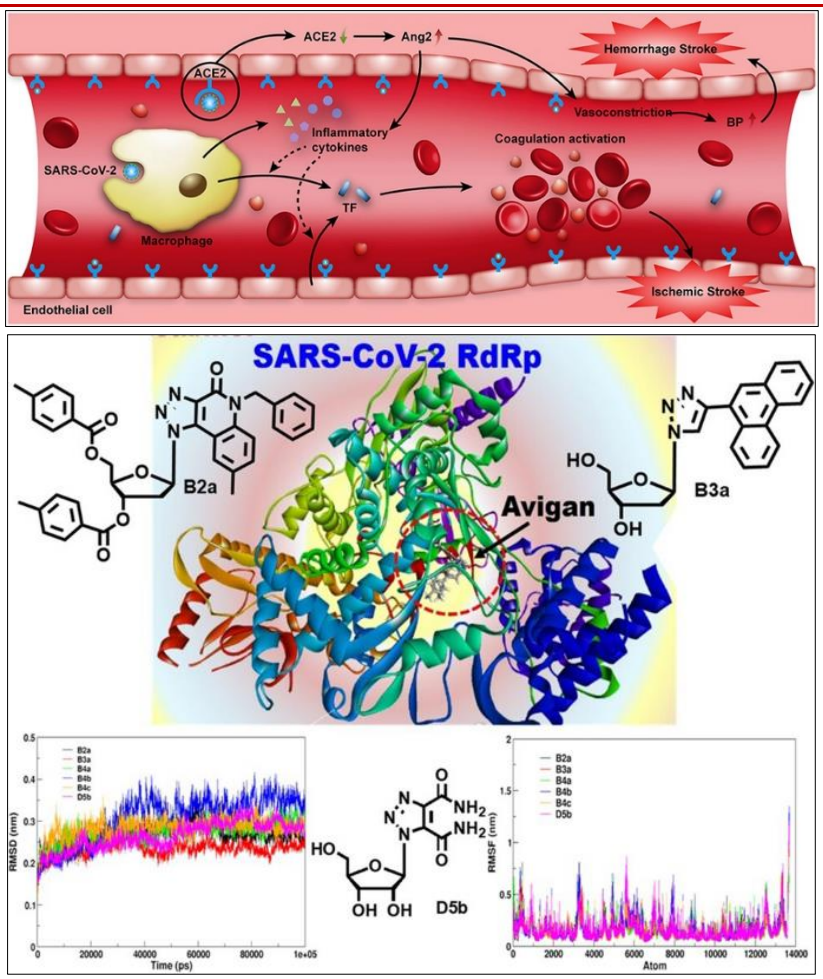
Abstract

In December 2019, an outbreak of a new type of acute respiratory disease (pneumonia) was reported in central China, and the number of people infected with it increased rapidly. Doctors named this disease COVID–19 and identified its origin as a virus called SARS–COV–2. So far, no effective drug has been produced that can be used to treat this disease with certainty, but some drugs have been identified and introduced that have shown a significant effect on the recovery of patients. The aim of this study is to evaluate and analyze the drugs that have been used to treat Covid–19 patients so that the drugs that have the greatest effect on the recovery of patients can be identified and introduced. The drugs lopinavir and ritonavir, in combination with complementary drugs such as interferon alpha, have been effective in reducing the load capacity of the Betacoronaviruses family. The drugs hydroxychloroquine and chloroquine have been effective in limiting the replication of COVID–19 in laboratory conditions. The antiviral drug amantadine reduces the replication capacity of the virus. Remdesivir can prevent lung damage caused by coronavirus infection in humans. In the case of favipiravir, studies have shown a recovery rate of 91.43%, indicating a very high effectiveness of this drug. Favipiravir and remdesivir have shown significant effectiveness. The drugs lopinavir and ritonavir, used in combination with interferon alpha, as well as hydroxychloroquine and chloroquine, have shown low effectiveness.





Key drugs target and discovery for SARS–CoV–2 DNA/RNA–dependent DNA/RNA polymerase replication by blocking interaction of virus polymerase with DNA/RNA.

Keywords: Coronavirus; COVID–19; Antiviral Drug; DNA/RNA Polymerase.

INTRODUCTION

In December 2019, a sudden increase in patients presenting with clinical symptoms of SARS–like pneumonia caused by a new unknown agent of the coronavirus family occurred in Wuhan (Hubei, China) and progressed rapidly [1]. The history and history of the patients indicated that they were exposed to the seafood market in Hubei, China, and therefore the probable origin of the virus was reported from seafood found in this market [2]. On the other hand, some studies suggested that pangolins or anteaters were the agents of transmission of the new coronavirus to humans. Subsequent studies suggested that the virus was transmitted from the Phyllopus bat to the pangolin and then evolved to humans. In other words, the bat is the primary host and the pangolin is the intermediate host of this virus [3].

Since then, the virus has spread rapidly to all provinces in China and 27 other countries around the world, with the number of infected people reaching 70,000 by February 17, 2020 (less than 2 months) [1]. According to the World Health Organization (WHO) in January 2020, every citizen living in Wuhan was suspected of having COVID–19 for 14 days before the onset of symptoms [2,7]. After some time, the causative agent of this disease was classified as SARS–CoV due to the severe acute respiratory syndrome, and finally, after isolation and definitive identification, the World Health Organization named this new virus COVID–19 on February 11, 2020. Other types of this family have emerged and spread in the past, including SARS and MERS, which have been considered serious health threats [1]. COVID–19 is a pathogen that attacks the human respiratory system and, as mentioned, causes severe acute respiratory syndrome [1]. For reasons that are not yet clear, this virus can cause a diverse range of symptoms in humans, from the common cold to more severe diseases such as MERS and SARS [4]. In other words, to date, most COVID–19 patients have shown mild symptoms such as dry cough, sore throat, loss of smell and taste, and fever. However, some patients have experienced various fatal complications, including organ failure, septic shock, pulmonary edema, severe pneumonia, and acute respiratory syndrome. To date, 54.3% of COVID–19 patients have been male, with a median age of 56 years. Most patients requiring intensive

care have been older and have underlying diseases such as cardiovascular, cerebrovascular, endocrine, gastrointestinal, and respiratory diseases. In addition to the common symptoms, these individuals have also reported shortness of breath, dizziness, abdominal pain, and anorexia [6,5]. Overall, COVID–19 is a self–limiting acute illness, but it can result in mortality of up to 2%. Deaths in this disease usually occur due to the dangerous complications mentioned above, especially extensive alveolar damage and progressive respiratory failure [3]. Studies indicate that the highest incidence of the disease is among the age group over 50 years and the lowest among people aged 0 to 9 years, and the mortality rate increases significantly in people over 60 years. As mentioned, the COVID–19 virus is in the human coronavirus group. In general, 7 different species of human coronavirus are divided into two groups: Alphacoronaviruses, including 229E and NL63, and Betacoronaviruses, including OC43, HKU1, SARS, MERS, and COVID–19 [7]. Coronaviruses are spherical in shape and contain single–stranded linear RNA of positive polarity and have the largest genome among RNA viruses (bp 30,000). It buddies from the membrane of the endoplasmic reticulum or Golgi bodies and does not grow easily in cell culture. The genomic organization of coronaviruses is pol–S–M–N and one of its receptors is aminopeptidase N. Two main groups of structural proteins are present in coronaviruses, including spike, nucleocapsid, matrix, envelope, and non–structural proteins such as proteases. An interesting point about COVID–19 is that, unlike other members, this virus does not use known coronavirus receptors such as aminopeptidase N and dipeptidyl peptidase 4 and requires angiotensin–converting enzyme receptor 2 for entry into the cell [8]. ACE2 is a type 1 membrane protein that is expressed in the lung, heart, kidney, and intestine, and its reduced expression is effective in causing cardiovascular diseases. COVID–19 binds to this receptor and enters through its spike protein. Reports suggest that in COVID–19, this binding is 10 times stronger than in SARS. On the other hand, cleavage of this protein by host cell cysteine proteases such as cathepsin L (CTSL) and cathepsin B (CTSB) is important for virus entry. Both of these enzymes are located in lysosomes and are key components of the lysosomal pathway [8].

Scientists around the world are searching for an effective drug or are designing a vaccine. Many countries affected by the pandemic have been forced to use hydroxychloroquine, an effective antimalarial drug, despite its side effects [9]. It appears to prevent the virus from entering the cell by inhibiting glycerolation in the host cell receptor, proteolytic processing, and endosomal acidification [10]. Lopinavir/ritonavir are among the other drugs used to treat COVID–19 patients. These drugs, which are widely used for HIV, act in COVID–19 by inhibiting the chymotrypsin–like protease 3–. Importantly, the timing of these drugs, which should be administered during the early phase of viral replication (the first 7 to 10 days), is very important [9]. Another drug used in the treatment of COVID–19 is amantadine. This drug can disrupt viral replication by reducing CTSL gene expression and disrupting the lysosomal pathway, reducing the amount of this virus in patients [8]. The aim of the present study is to investigate the effect of different drugs on the treatment of COVID–19.

RESULTS AND DISCUSSION

The use of different drugs in the treatment of COVID–19 has reported different results. For example, in a study conducted on a 50–year–old man hospitalized on January 21, 2020, the results indicated that the drugs used were ineffective in reducing the patient's symptoms. The patient did not visit the clinic with symptoms of fever, chills, cough, fatigue, and shortness of breath and was immediately admitted to the influenza ward and received oxygen. The drugs prescribed for this patient included interferon alfa–2b (5 million units, twice daily) and lopinavir plus ritonavir (500 mg twice daily, orally) as antivirals and moxifloxacin (0.4 g once daily, intravenously) to prevent secondary infection. Given the shortness of breath and hypoxemia, methylprednisolone (80 mg twice daily, intravenously) was used to reduce lung inflammation. After receiving the drug, his body temperature decreased from 39 to 36.4°C. However, there was no improvement in other symptoms, including cough, shortness of breath, and fatigue. On day 12 of the disease, chest X–ray showed progression in the liver and spread to both lungs. On day 13 of the disease, the patient's symptoms had not improved and oxygen saturation remained above 95%. In the afternoon of day 14 of the disease, hypoxemia and shortness of breath worsened, and despite receiving HFNC oxygen therapy (concentration 100%, flow rate 40 liters per minute), the oxygen saturation decreased to 60%. The patient went into sudden cardiac arrest and despite immediate invasive ventilation, chest compressions, and adrenaline injection, resuscitation was unsuccessful and he died [11]. In another study in which lopinavir/ritonavir was used, the efficacy of these two drugs was reported. The study was conducted by administering the drug to a 54–year–old Korean man living in Wuhan, China. He arrived in Korea on January 20, 2020, and the first symptoms of chills and muscle pain appeared on January 22. After contacting a public health center on January 25, he was admitted to a negative pressure room at California South University (CSU) Hospital, and tested positive for COVID–19 on January 26. The patient was prescribed 2 tablets (lopinavir 200 mg/ritonavir 50 mg) orally. Significantly, the β–coronavirus load decreased from the day after taking lopinavir/ritonavir, and no detectable coronavirus titers were observed thereafter. It is possible that the reduction in SARS–CoV–2 load was due to the administration of lopinavir/ritonavir, or both. Therefore, more data need to be collected to find out the direct effect of lopinavir/ritonavir on the treatment of COVID–19. Comparing these two studies, it can be concluded that further evidence and studies are needed to evaluate the effectiveness of these two drugs, and factors such as the

time of drug administration may have an impact on the results of these drugs (Figures [1–16-12].

In addition to these two review articles, another article was published in April 2020 that examined 199 patients with COVID–19, which rejected the effect of administering lopinavir and ritonavir on improving the disease in 99 patients taking these two drugs (compared to a control group of 100 patients without taking lopinavir and ritonavir) (Figures (1–16)) [15].

In another study, in addition to the two drugs lopinavir/ritonavir, galidesivir, a nucleoside RNA polymerase inhibitor and considered as potential candidates for treatment, was used. Repurposing these drugs, which are available for immediate use in the treatment of SARS–CoV infections, could improve the treatment situation (Figures (1–16)) [16].

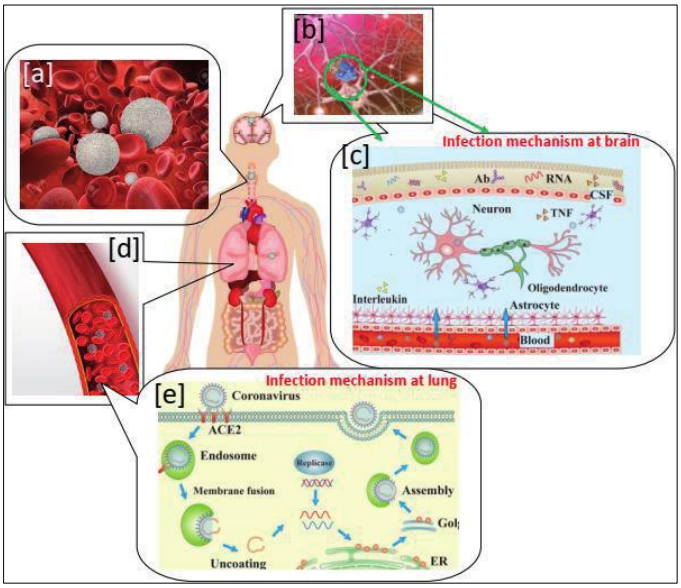


Figure 1: Schematic of amantadine blocks the M2 protein and lopinavir is a protease inhibitor, which inhibits the protease and prevents the progression of viral infection. Remdesivir and favipiravir also inhibit RdRp and are effective in treating coronavirus infection (13–15)

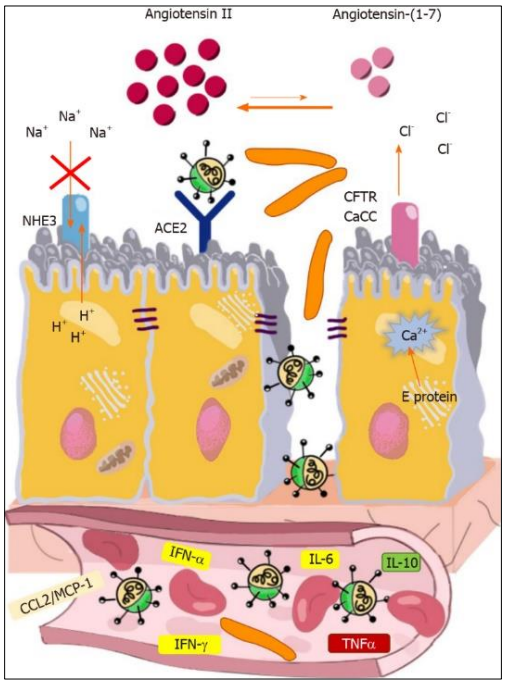


Figure 2: Schematic of detailed interaction study of Bag's unnatural nucleosides' drugs with the AA residues in detailed interaction study of Bag's designer Avigan analogues s with the AA residues in the binding pocket of SARS–CoV–2 RdRp

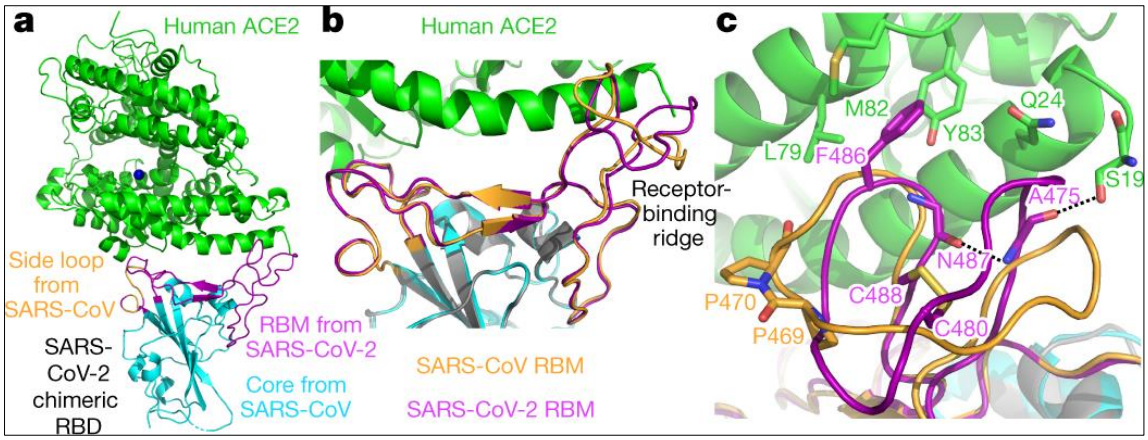


Figure 3: (a) Genome structure of COVID–19, (b) spike protein structure of COVID–19 constructed from C–I–TASSER and (c) human angiotensin converting enzyme 2 (ACE2) (yellow color) and spike protein trimmer (right side multicolor (magenta, cyan and blue))

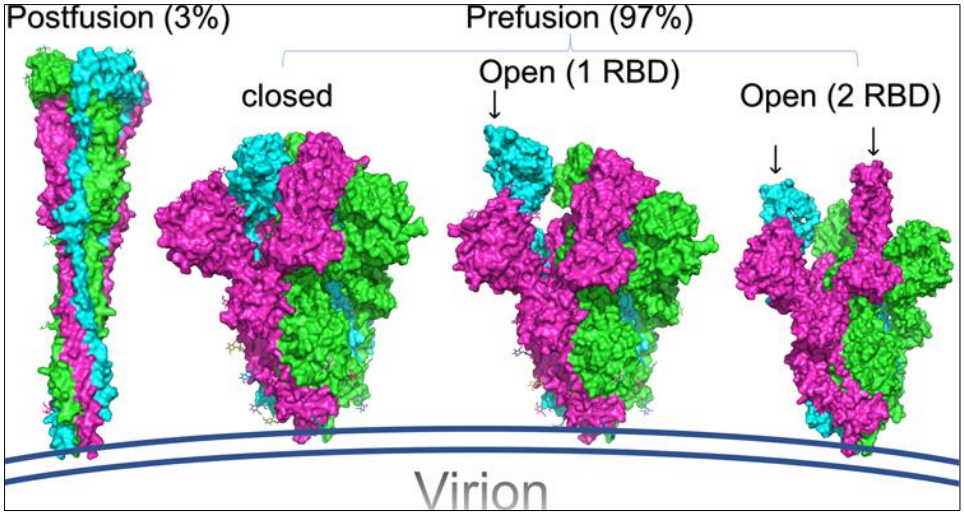


Figure 4: Superimposed structures of RdRp in complex.

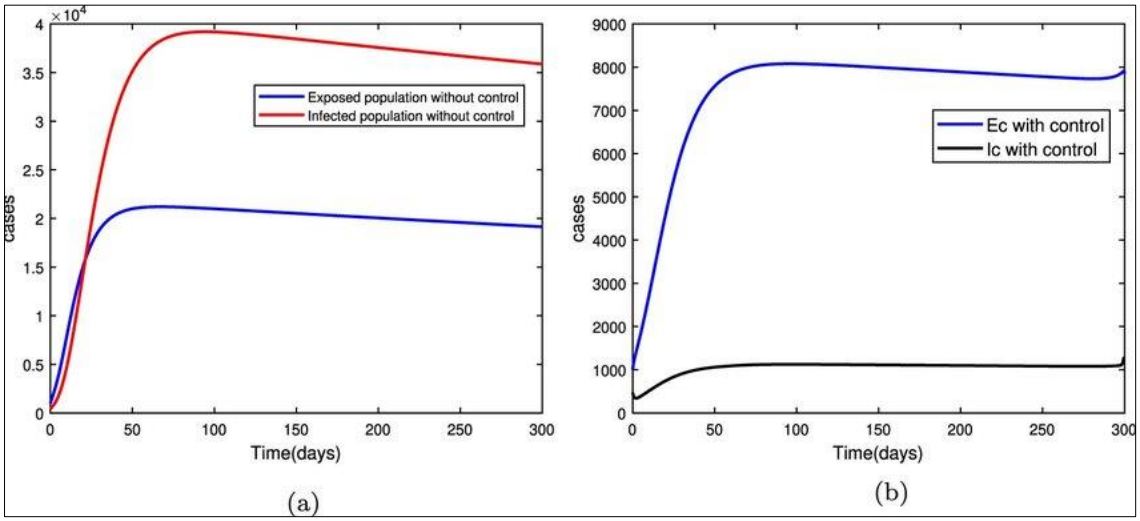


Figure 5: Simulation data of (a) exposed population without control and (b) infected population without control

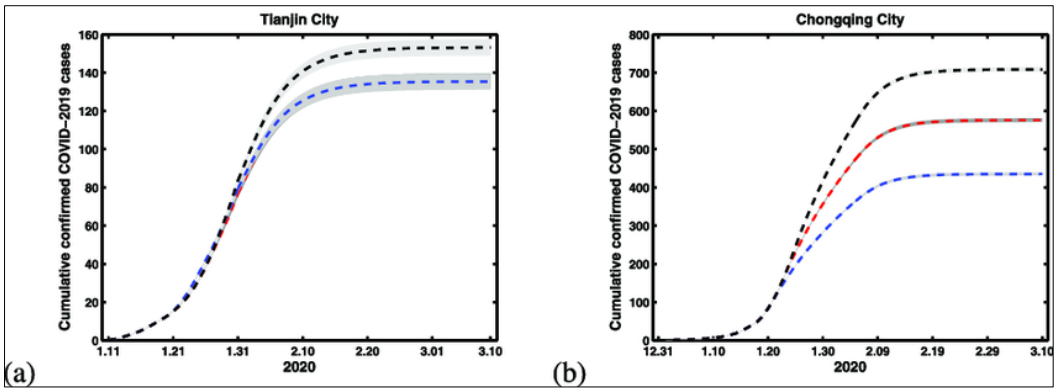


Figure 6: Simulation data of communicative confirmed COVID–19 cases in (a) Tianjin city and (b) Chongqing city

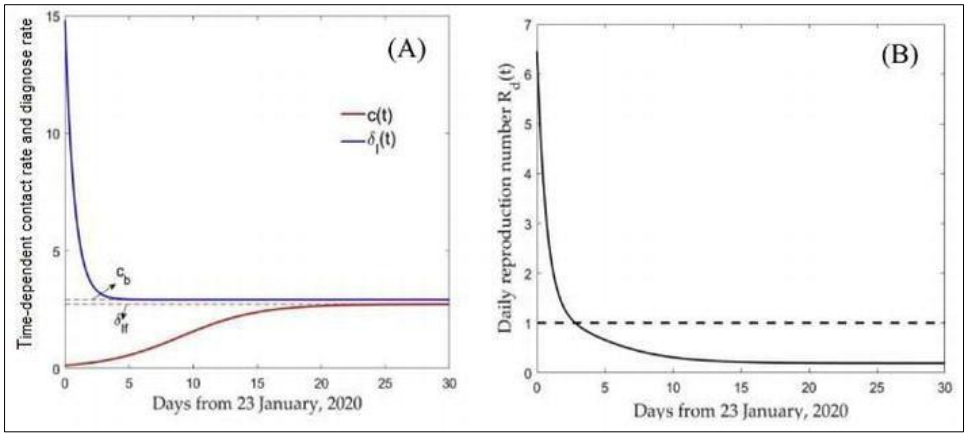


Figure 7: Simulation data of (a) contact rate C (t) and diagnosis rate and (b) effective daily reproduction ratio for the period of 30 days

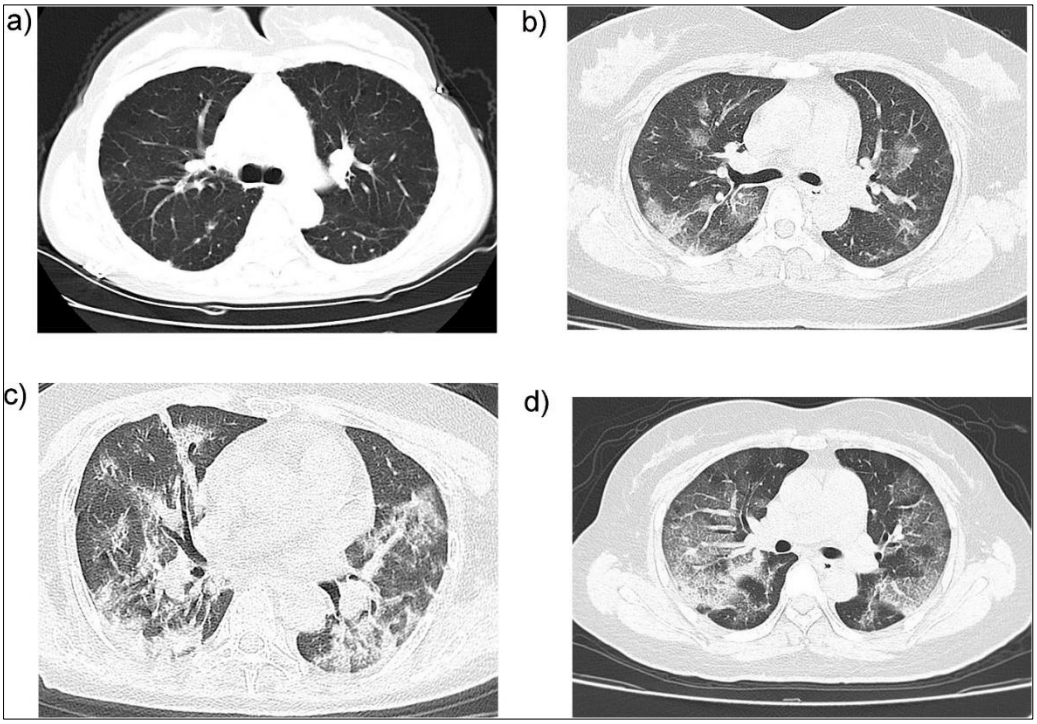


Figure 8: Possible targets of COVID–19 (lungs, heart, kidneys, intestines, brain and testicles) (a) COVID–19 distribution and ACE2 receptor in human, (b) COVID–19 transmission to brain through upper nasal transtibial path, (c) inset image shows binding mechanism of spike protein at the site of neuron and (d) showing COVID–19 distribution through blood circulation at lungs

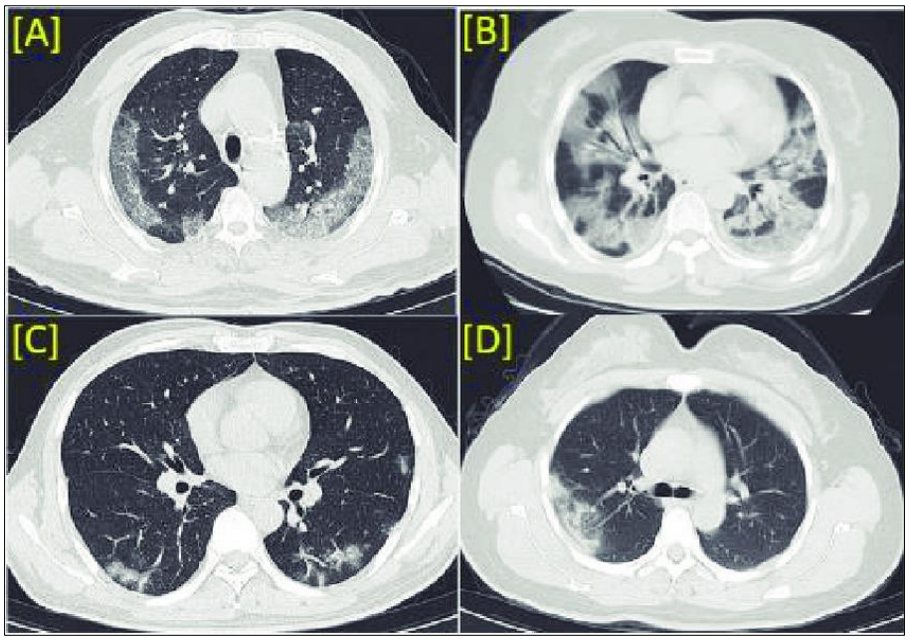


Figure 9: Prolonged COVID–19 pneumonia in a 75–year–old female with follicular lymphoma who had last received rituximab 180 days before COVID–19 diagnosis. Chest CTs of the patient taken (a) 60 days, (b) 90 days, (c) 120 days and (d) 150 days

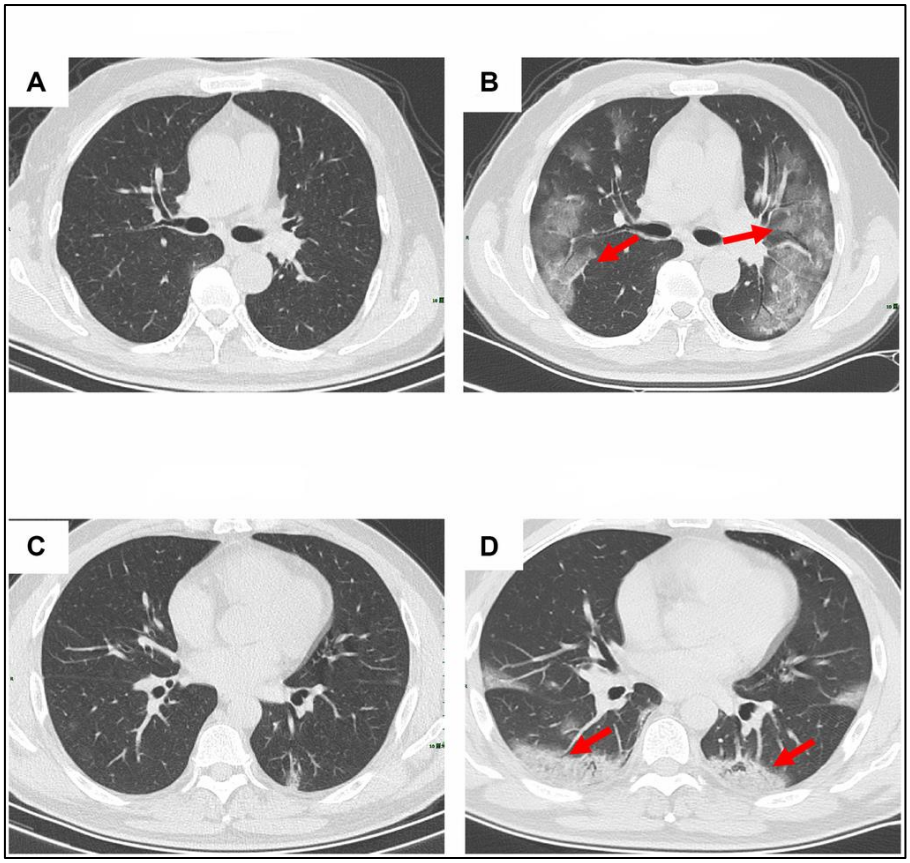


Figure 10: (a) Conjugation of spike protein on to the surface of graphene via 1–pyrenebutyric acid N–hydroxysuccinimide ester, (b) model showing spike protein on the surface (covered with graphene) of field effect transistor, (c) FET sensor sensitivity in presence of SARS–COV–2 antibody and in absence of SARS–COV–2 antibody and (d) FET sensor sensitivity in MERS–COV and SARS–COV–2

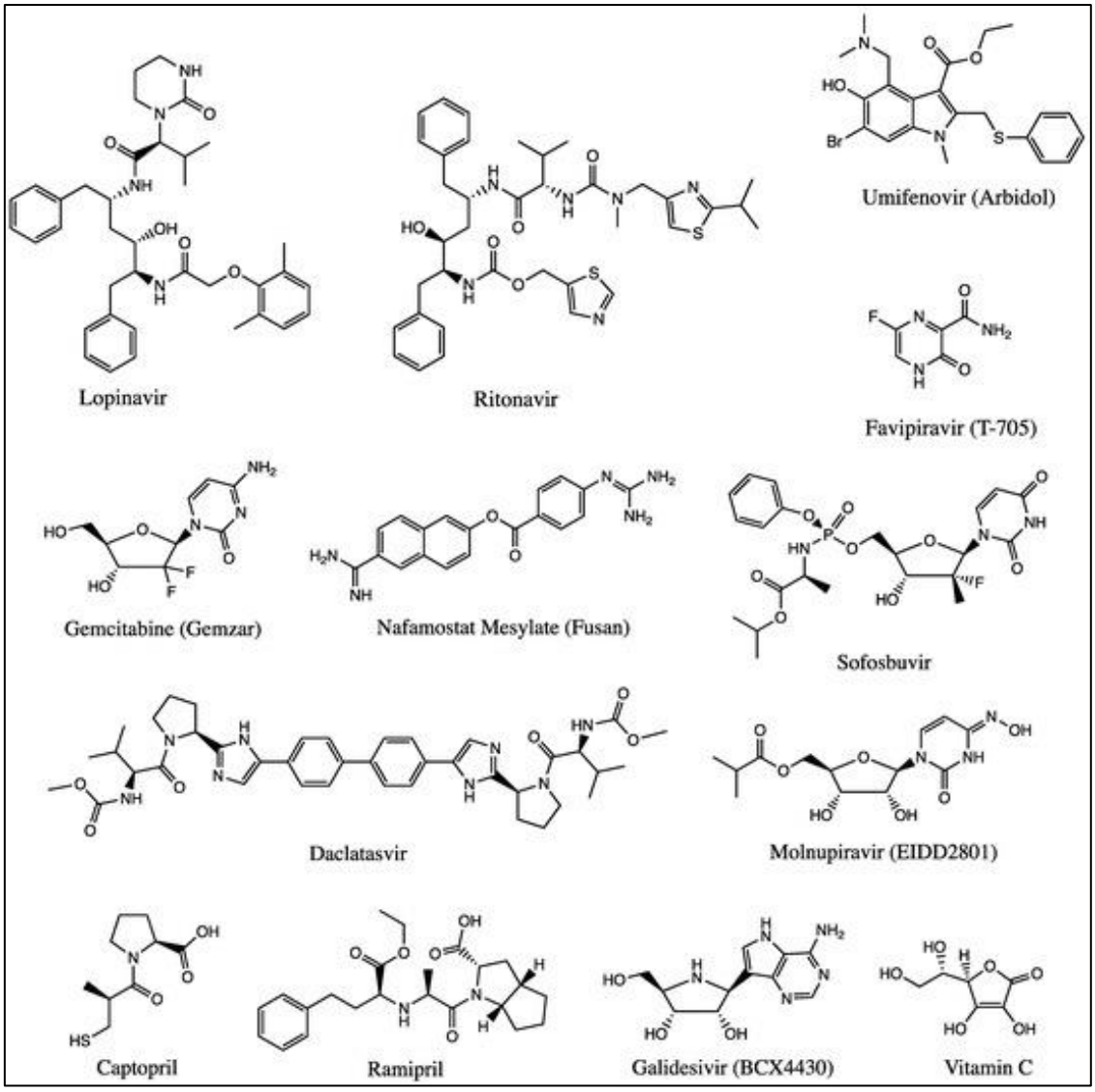


Figure 11: The molecular structures of 13 commercial drugs that are used or under clinical trial to fight against COVID–19

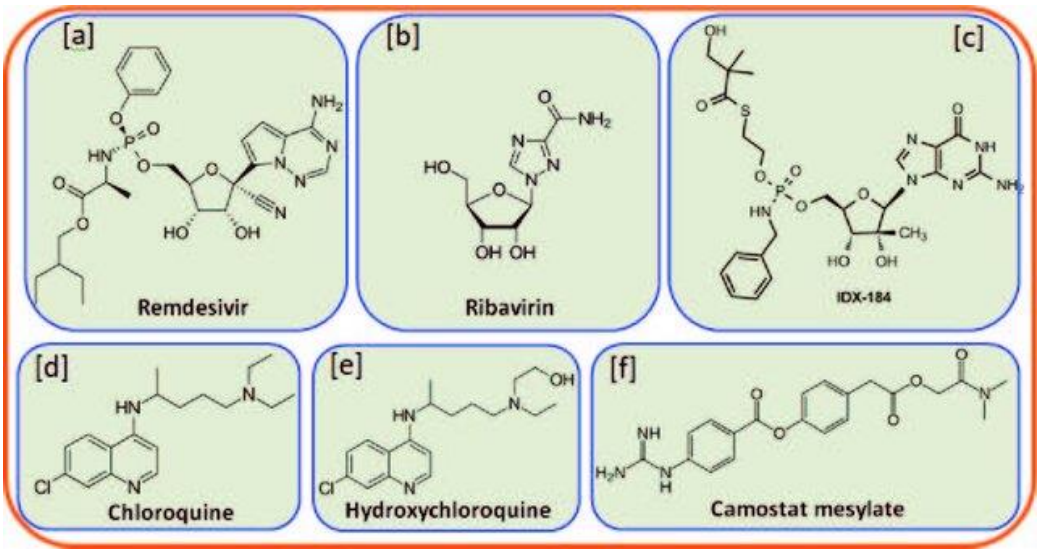


Figure 12: Molecular structures of viral entry inhibitor (a) remdesivir, (b) ribavirin, (c) IDX–184, (d) chloroquine, (e) hydroxychloroquine and (f) camostat mesylate

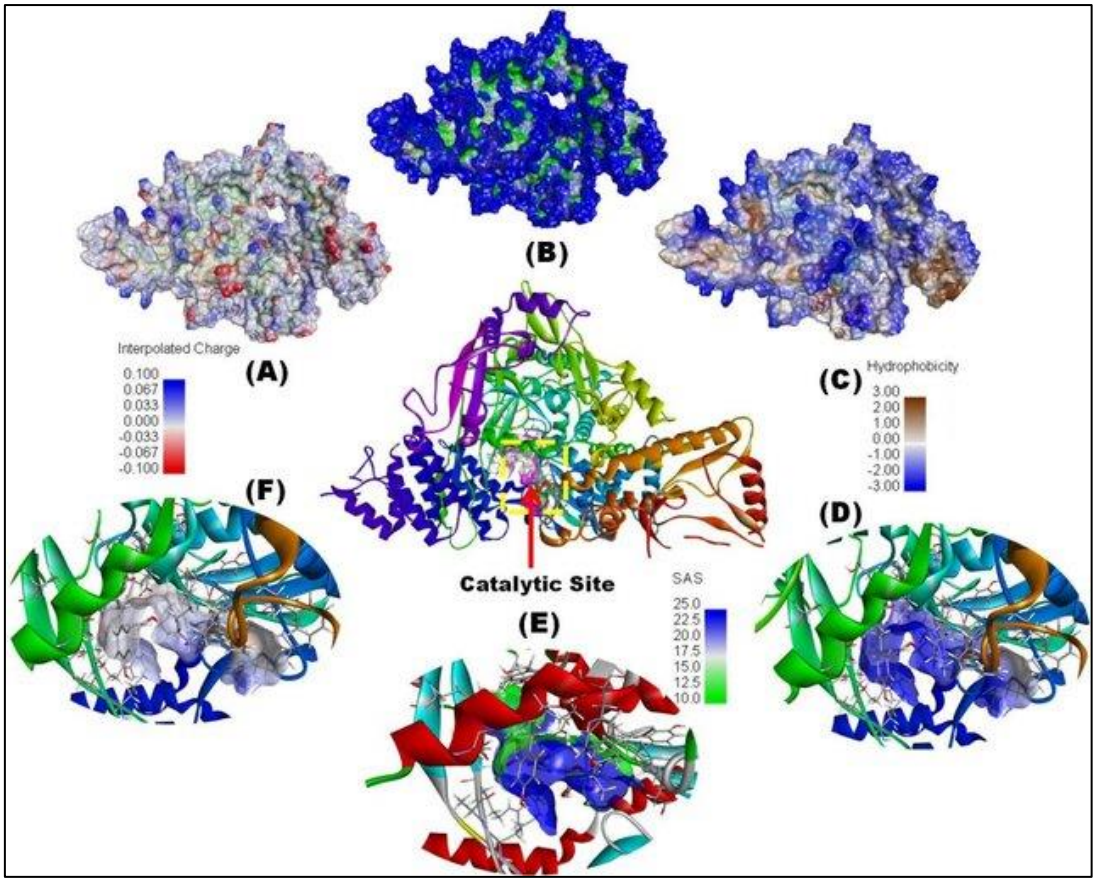


Figure 13: The telescopic view of the active site and their physical properties such as hydrophobicity, solvent accessibility and surface charge density of the AA present in the binding pocket

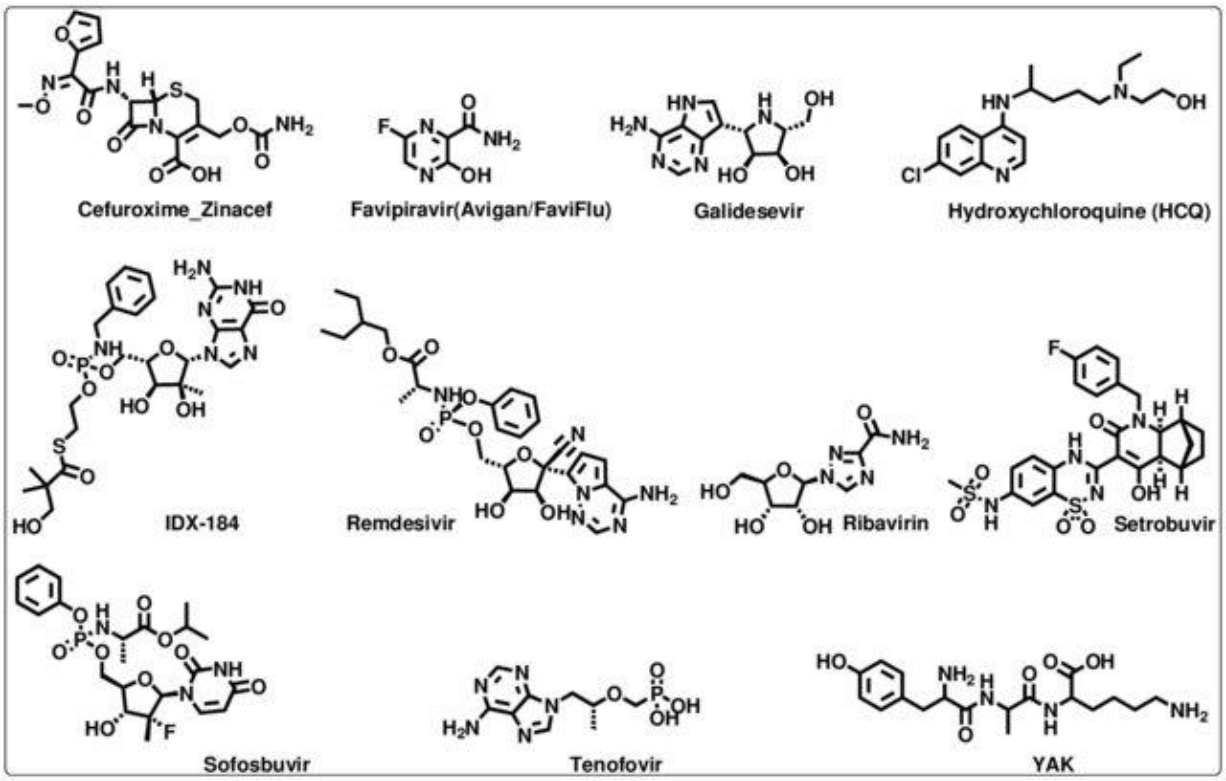


Figure 14: The molecular structures of 11 commercial drugs that are used or under clinical trial to fight against COVID–19

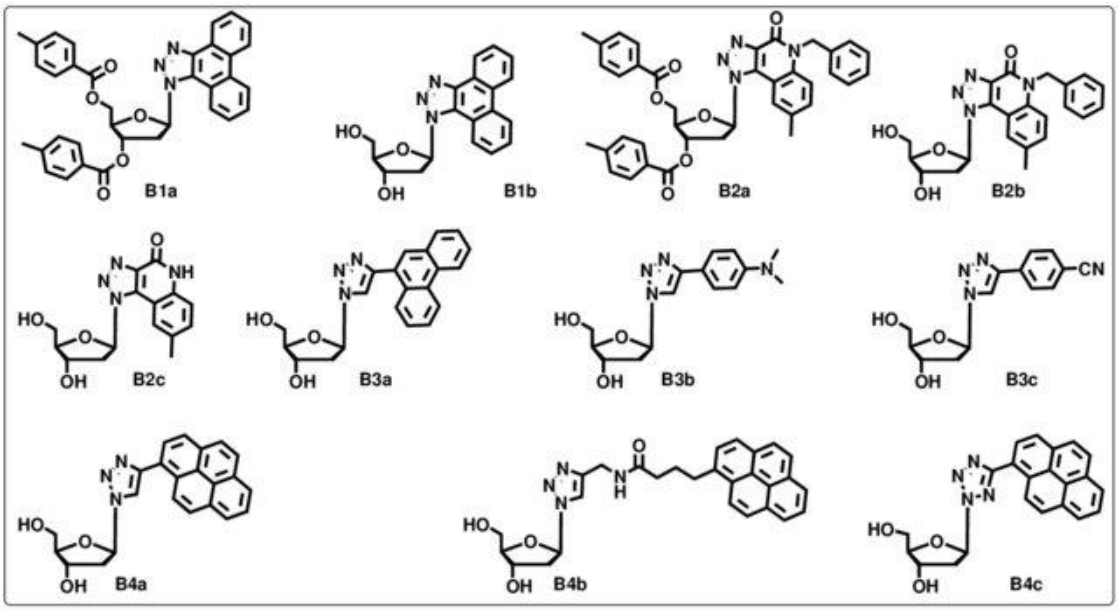


Figure 15: The molecular structures of our reported unnatural nucleosides as possible inhibitors of SARS–CoV–2 RdRp

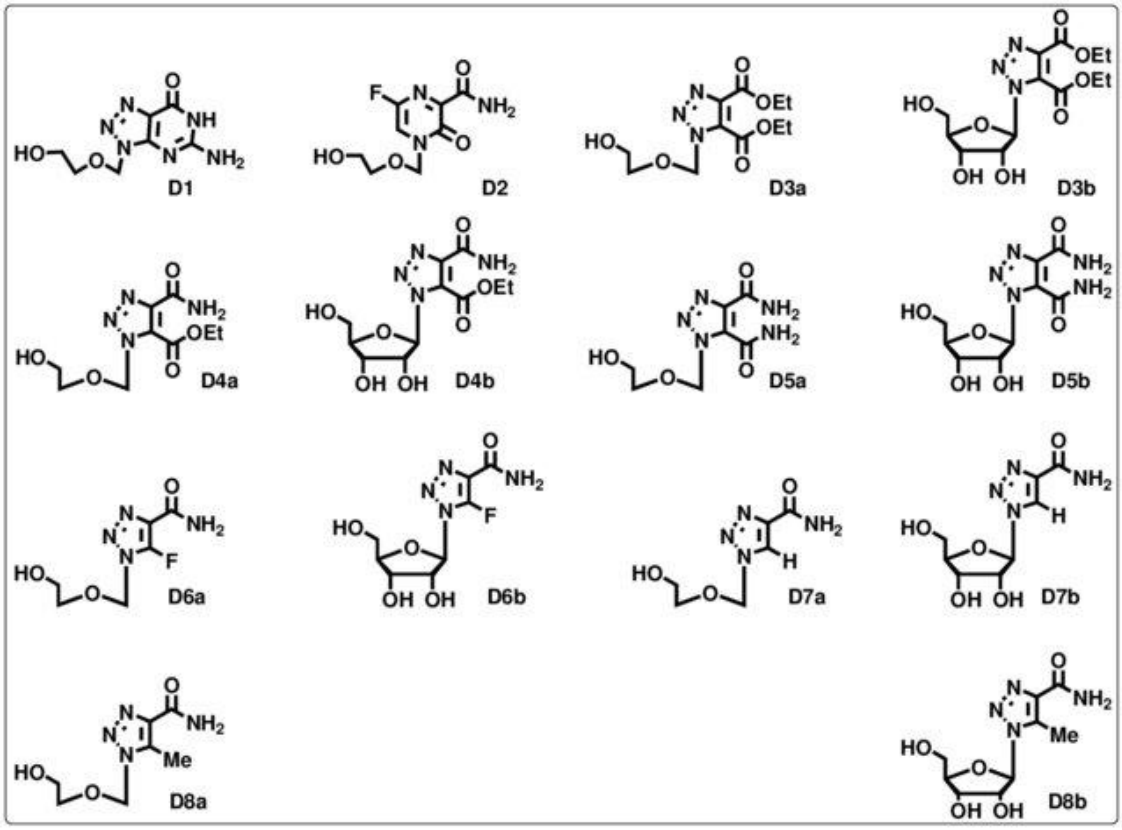


Figure 16: The molecular structures of our designed unnatural nucleosides as possible inhibitors of SARS–CoV–2 RdRp

Regarding the use of chloroquine, the results of the reports were more consistent. In a review of six articles, chloroquine appeared to be effective in limiting the replication of COVID–19 in vitro (16). However, a controversial point was raised in another study that examined the effects of chloroquine and hydroxychloroquine in diabetic and non–diabetic patients and concluded that in diabetic patients (as a group of people with underlying disease), the effectiveness of hydroxychloroquine on blood sugar, cardiovascular function and viral load in patients with diabetes requires further investigation (Tables (1–3)) [17–37].

Another drug evaluated in the current study is amantadine. Fewer studies have been conducted on the effectiveness of amantadine compared to other drugs, but it is important to note that this drug has been used since

the beginning of the outbreak and has been effective in patients, including young people aged 16 to 23 years (Tables (1–3)) [38–66].

In a study of 1,062 patients randomized to remdesivir (541 to remdesivir and 521 to placebo), remdesivir had a median time to recovery of 10 days, while placebo had a median time to recovery of 15 days. Remdesivir accelerated recovery by 5 days and also limited the spread of the virus to the patient's lungs (Tables (1–3)) [67–84].

In fact, in vitro studies have shown that remdesivir can inhibit diseases caused by coronaviruses such as SARS–CoV and MERS–CoV. In an in vitro test using primary human respiratory epithelial cells, remdesivir was effective against Bat–CoVs, pre–outbreak Bat–CoVs, and human–CoV in human lung cells. This study showed that Remdesivir was superior to lopinavir, ritonavir, and interferon beta in vitro and in a mouse model of MERS–CoV (Tables (1–3)) [85–110].

In relation to another drug called favipiravir, a study examined the effects of favipiravir (FPV) versus lopinavir (LPV) and ritonavir (RTV) for the treatment of COVID–19. Patients with laboratory–acquired COVID–19 receiving oral FPV (day 1 at a dose of 1600 mg twice daily; days 2–14 at a dose of 600 mg twice daily) plus aerosolized interferon (IFN)–α (5 million international units twice daily) were enrolled in the FPV arm of the study, while patients treated with lopinavir and ritonavir (days 1–14 at a dose of 400 mg/100 mg twice daily) plus aerosolized IFN–α (5 million international units twice daily) were enrolled in the control arm [25]. Chest computed tomography (CT) changes, viral clearance, and drug safety were compared between the two groups. For the 35 patients enrolled in the FPV arm and 45 patients in the control arm, all characteristics were comparable between the two arms. The FPV arm showed significant improvement in chest CT compared with the control arm, with a recovery rate of 91.43% versus 62.22%, respectively. After adjusting for confounders, the FPV arm also showed significant improvement in chest CT. FPV is independently associated with faster viral clearance. Furthermore, fewer adverse events were observed in the FPV arm than in the control arm. In this pre–trial–controlled study, FPV demonstrated better treatment responses in COVID–19 in terms of disease progression and viral clearance. These preliminary clinical results provide useful information about the treatment of SARS–CoV–2 infection (Tables (1–3)) [111–145].

In this study, several articles have been reviewed on the efficacy of drugs with different mechanisms and effects on the COVID–19 virus. Reports indicate that the drug lopinavir/ritonavir, which was used to treat a large number of patients, did not completely and definitively cure the patients. It was also observed that the administration of other drugs such as moxifloxacin (to prevent secondary infection) and methylprednisolone did not affect the efficacy of the drug. In another study, it was shown that the use of lopinavir/ritonavir reduces the viral load of the β–coronavirus, but more evidence is needed to determine the direct effect of lopinavir/ritonavir on the treatment of COVID–19 (Tables (1–3)) [146–150].

The efficacy of the drug hydroxychloroquine in diabetic patients is still questionable. Binding to the receptor is essential for the virus to enter the host cell, and this binding is considered the first step in pathogenesis. Therefore, strategies that can prevent this binding would be very effective in treating this disease. The results of the studies showed that drugs such as Remdesivir and Favipiravir were more effective than other drugs such as lopinavir and ritonavir [30–32]. Recently, a group of scientists in different parts of the world led by the California South University (CSU), headed by Prof. Dr. Alireza Heidari, conducted more or less successful research on the drug hrsACE2, which was published in the scientific journal Cell. This drug effectively prevents the coronavirus from attaching to the body of cells. As mentioned, one of the main receptors of this virus that distinguishes it from SARS is a key protein called ACE2 on the surface of the cell membrane, which plays an important role in the process of attaching the virus to the body's cells. Therefore, the development of a drug that can prevent the virus from attaching to this receptor is a major advance in the field of treatment of this disease (Tables (1–3)) [151–159].

Another drug that can be investigated in the treatment of coronavirus disease is the drug amantadine. Because, as mentioned, this drug reduces the capacity of virus replication due to the disruption it causes in the lysosomal pathway. Since a specific drug that targets the ACE2 receptor has not yet been produced, amantadine can be used as a replication inhibitor [35]. Amantadine is used to treat Parkinson's disease. In addition, this drug is also used to prevent and treat respiratory tract infections caused by influenza A strains. This drug prevents influenza infection by inhibiting the uncoating process of the virus and the release of its nucleic acid into respiratory epithelial cells. Therefore, it can also be effective in the treatment of co–infection with COVID and influenza. Amantadine is well absorbed from the gastrointestinal tract, distributed into saliva and nasal secretions, and can reach various areas where the virus is colonized. Therefore, it seems that its use can be effective in COVID19 patients. Therefore, if the drug fails to inhibit the binding of the virus to the receptor and its entry into the cell, it can interfere with the next stage of pathogenesis, which is viral replication. The results of various studies indicate that the effectiveness of the drug Remdesivir and interferon beta is greater than that of the drugs lopinavir, ritonavir, and interferon beta in vitro and in the MERS–CoV mouse model (Tables (1–3)) [160–170].

Table 1: Current drugs in clinical trials against COVID–19

Drug	Dosage	Mode of administration	Trial phase	Result/outcome	References
Remdesivir or placebo	200 mg on day 1 followed by 100 mg daily for 9 more days	Intravenous	3	Shortened recovery time No associated mortality	Beigel et al., 2020
Remdesivir	200 mg on day 1 followed by 100 mg daily for 9 more days	Intravenous	3	Improved breathing and clinical conditions	Grein et al., 2020
Remdesivir	100 mg at every 24 h for 9 days	Intravenous	3	Improved breathing Became stable at room air	Hillaker et al., 2020
Hydroxychloroquine (HCQ) sulfate + Azithromycin (AZ)	200 mg HCQ thrice daily, with AZ (500 mg daily on day 1, 250 mg on days 2–5), 10 days	Oral	3	Reduce viral carriage Effect reinforced by addition of AZ	Gautret et al., 2020
HCQ + AZ	200 mg HCQ for 10 days with 5 days of AZ (500 mg daily on day 1, 250 mg on day 2–5), 10 days	Oral	3	Low proportion of adverse events of patients with mild symptoms	Million et al., 2020
Chloroquine (CQ) phosphate + Lopinavir/ritonavir	500 mg of CQ, 400 mg/100 mg/capsule of lopinavir/ritonavir, twice daily for 10 days	Oral	4	Quick discharge from hospital Few adverse events	Dong L. et al., 2020
CQ + Lopinavir/ritonavir	500 mg CQ with 400/100 mg of lopinavir/ ritonavir, twice daily, 10 days	Oral	4	Achieved lung clearance Became SARS-CoV-2 negative after 2 days	Huang M. et al., 2020
Favipiravir	1,600 mg twice in day 1 followed by 600 mg twice daily for days 2–10	Oral	2	Relieved pyrexia and cough Raised uric acid in serum	Chen C. et al., 2020
Favipiravir	1,600 mg twice in day 1 followed by 600 mg twice daily for days 2–14	Oral	2	Shortened viral clearance duration	Cai et al., 2020
IFN-α	5 million units (U) + 2 ml sterile water for injection, twice daily, 10 days	Vapor inhalation	N/A	Not mentioned	Dong L. et al., 2020
Ribavirin + IFN-β1b or Lopinavir/ritonavir	400 mg ribavirin at every 12 h, 8 million IU of IFN-β1b on alternate days, or 400 mg lopinavir and 100 mg ritonavir at every 12 h, 14 days	Subcutaneous injection, via nasogastric tube	2	Better virological and clinical condition No serious adverse events	Hung et al., 2020
Arbidol	200 mg, thrice daily, 10 days	Oral	4	Not mentioned	Dong L. et al., 2020
Arbidol (Umifenovir)	200 mg, thrice daily, 10 days	Oral	4	Relieved pyrexia and cough	Chen C. et al., 2020
Tocilizumab	400 mg diluted with 100 ml 0.9% normal saline, twice daily, 10 days	Intravenous	3	Body temperature returned to normal Relieved clinical symptoms	Xu X. et al., 2020
Nafamostat	200 mg; 24 h continuously with acetaminophen	–	-	CRP level decreased SARS-CoV-2 negative	Jang and Rhee, 2020

Table 2: Current vaccines in clinical trials against COVID–19

Candidate vaccine (NCT ID)	Composition	Mode of action	Dose	Clinical trial (volunteers)	Country and company/institute/ organization
mRNA-1273 (NCT04283461, NCT04405076)	SARS-CoV-2 S protein encoded mRNA in lipid nanoparticle	Immune response against Cov-2 S protein	50 μg	Phase I (105) Phase II (600)	Moderna, NIAID, Biomedical Advanced Research and Development Authority
SCB-2019 (NCT04405908)	CoV-2 S proteins trimer produced by mammalian cell culture	Antibodies against CoV-2 to prevent binding and infection	3 and 30 μg at days 1 and 22, respectively	Phase I (150)	Clover Biopharmaceuticals
NVX-CoV2373 (NCT04368988, EudraCT2020-004123-16)	Insect cells infections to express CoV-2 S protein.	Antigen presentation in the local lymph nodes	25 μg at days 1 and 22	Phase I (131)	Novavax
CoronaVac (NCT04352608, NCT04383574)	Inactivated SARS-CoV-2	Diverse immune response against numerous viral antigens	300 SU/ml antigen at days 1 and 29	Phase I (216) Phase II (950)	Sinovac Biotech Co.
Ad5-nCoV (NCT04313127, NCT04341389, NCT04398147, ChiCTR2000031781, ChiCTR2000030906)	Replication inactive adenovirus	Antibodies production against CoV-2 S protein.	1 ml injection in the deltoid muscle at day 1 (1×10^{11} vp)	Phase I (108) Phase I/II (696) Phase II (508)	CanSino Biologics, Institute of Biotechnology, Academy of Military Medical Sciences, China
ChAdOx1 nCoV-19 (NCT04324606, NCT04400838, EudraCT 2020-001072-15, EudraCT 2020-001228-32)	Attenuated adenovirus	Endogenous antibodies protection against SARS-CoV-2	A single dose of 5×10^{10} vp	Phase I/II (1,090) Phase II/III (10,260)	Consortium of the Jenner Institute, Oxford Biomedical Research Center, University of Oxford
Bacille Calmette-Guérin (NCT04387409 and another 13)	Live attenuated *Mycobacterium bovis*	Immune responses against *M. tuberculosis* infection	$2–8 \times 10^5$ CFU injection in 0.1 ml suspension	Phase III (18,798) Phase IV (2,800)	University Medical Center Utrecht, Radboud University and other organizations
Measles, mumps, and rubella (MMR) (NCT04357028)	Live-attenuated measles, mumps, and rubella virus	Cross reaction with SARS-CoV-2	0.5 ml	Phase III (200)	Cairo University Hospital Cairo, Egypt
INO-4800 (NCT04336410)	DNA plasmid that encodes S protein antigens of CoV-2	T cells, B cells, and encoded proteins production	1.0 mg ID injection at day 0 and week 4	Phase I (40)	Inovio Pharmaceuticals
AV-COVID-19 (NCT04386252)	DC and GM-CSF from blood monocytes	Non-mentioned	1× antigen with/without 500 μg GM-CSF	Phase I/II (180)	Aivita Biomedical, Inc.
Covid-19/aAPC (NCT04299724)	Lentivirus modified DC, immune modulatory genes, and CoV-2 minigenes	Priming T lymphocytes against CoV-2	Three subcutaneous injections 5×10^6 cells	Phase I (100)	Shenzhen Geno-immune Medical Institute Shenzhen, Guangdong, China
LV-SMENP-DC (NCT04276896)	DC modification with lentivirus vectors to express SMENP	Priming T lymphocytes against CoV-2	5×10^6 cells (subcutaneous) and antigen specific 1×10^8 CTLs (IV infusion)	Phase II (100)	Shenzhen Geno-immune Medical Institute Shenzhen, Guangdong, China

S, Spike; SU, subunit; vp, vaccine particle; ID, intradermal; DC, dendritic cell; GM-CSF, granulocyte-macrophage colony-stimulating factor; SMENP, Shenzhen Minigene Engineered-NP; IV, intravenous; CTLs, cytotoxic T lymphocyte.

Table 3: Suggested *in silico* medicines against COVID–19

No	Title of the work	Targets	Outcome	Biological screening	Ref
Vaccines					
1	A thermostable mRNA vaccine against COVID-19	RBD of SARS-CoV-2	ARCoV vaccine candidate	It has shown protection in animal models	Zhang et al. (2020b)
2	SARS-CoV-2 mRNA vaccine development enabled by prototype pathogen preparedness	Spike protein	mRNA-1273 vaccine	It has reduced the viral load 100 fold at the concentration of 0.1 μg	Corbett et al. (2020)
3	Design of a multiepitope-based peptide vaccine against the E Protein of human COVID-19: An immunoinformatics approach	E-protein	YVYSRVKNL, SLVKPSFYV, and LAILTALRL	---	Abdelmageed et al. (2020)
Inhibitors					
4	Peptide antidotes to SARS-CoV-2 (COVID-19)	Spike protein	SARS-BLOCK™ - a synthetic peptide scaffolds	Single-micromolar concentration	Watson et al. (2020)
5	Computational design of ACE2-based peptide inhibitors of SARS-CoV-2	RBD	Inhibitors-2, inhibitor-3, and inhibitor-4	---	Han and Král, (2020)
6	Design of potent membrane fusion inhibitors against SARS-CoV-2, an emerging coronavirus with high fusogenic activity	Spike protein	Lipopeptide (IPB02)	Dual split-protein based fusion cell-cell assay (0.025 μM)	Zhu et al. (2020b)
7	Peptide-like and small-molecule inhibitors against COVID-19	M^{pro}	Cobicistat, ritonavir, lopinavir, and darunavir	---	Pant et al. (2020)
Antibodies					
8	A human monoclonal antibody blocking SARS-CoV-2 infection	Spike protein	47D11 antibody	IC_{50} value: 0.57 μg/ml	Wang et al. (2020d)
9	Potent binding of 2019 novel coronavirus spike protein by a SARS coronavirus specific human monoclonal antibody	Spike protein	Monoclonal antibody (CR3022)	KD value: 6.3 nM	Tian et al. (2020)
Immunity enhancers or modulators					
10	The potential of antimicrobial peptides as an antiviral therapy against COVID-19	---	Lactoferrin	---	Elnagdy and Alkhazindar, (2020)
11	Type 1 interferons as a potential treatment against COVID-19	---	Type 1 interferons	---	Sallard et al. (2020)
Miscellaneous					
12	SARS-CoV and SARS-CoV-2 main protease residue interaction networks change when bound to inhibitor N3	M^{pro}	Identified the conformational changes in one cluster and four residues (131, 175, 182, and 185)	---	Griffin (2020)
13	*In silico* discovery of candidate drugs against covid-19	---	Identified 36-drugs candidates as effective agents against COVID-19	---	Cava et al. (2020)
14	Structural basis of SARS-CoV-2 $3CL^{pro}$ and anti-COVID-19 drug discovery from medicinal plants	Chymotrypsin-like cysteine protease ($3CL^{pro}$)	Identified 9-hit molecules for the management of COVID-19	---	Tahir ul Qamar et al. (2020)
15	Computational screening of antagonists against the SARS-CoV-2 (COVID-19) coronavirus by molecular docking	Main protease	Luteolin has been suggested as a hit molecule for the specific binding with SARS-CoV-2 main protease	---	Yu et al. (2020b)

CONCLUSIONS

According to the studies reviewed in this article, some different drugs, including remdesivir and favipiravir, have very high efficacy, and lopinavir and ritonavir have very low efficacy in the treatment of COVID–19, which requires further and more detailed studies. Also, the drug amantadine, which is an M2 protein inhibitor, can be somewhat effective in the treatment of COVID–19, but more studies are needed to determine the level of effectiveness. On the other hand, the drugs hydroxychloroquine and chloroquine have also shown fewer effective effects and are not recommended due to insufficient information about the effect of this disease in diabetics. Other drugs that are used as supplements to treat this disease, including interferon alpha and moxifloxacin, have performed well. Also, the anti–inflammatory drug methylprednisolone has an effective effect that can be used as a supplement to reduce inflammation caused by this disease.

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