Of the vast number of pathogens of infectious diseases, a special place belongs to viruses. It is no coincidence that the World Health Organization has declared the 21st century the century of viruses. Over the past 20 years, the world has faced epidemics of coronavirus infection: severe acute respiratory syndrome (SARS), Middle East respiratory syndrome (MERS), and COVID-19; outbreaks of avian and swine flu; the spread of Ebola and Zika fever, which have become serious challenges to all of humanity. Slowly progressing, persistent, and latent viral infections, the mortality rate from which exceeds the mortality rate from acute infections, pose no less danger. Some pathogens (for example, HIV) are characterized by long latency periods, high antigenic variability, and the ability to affect the immune system up to its complete destruction. Other viruses (for example, human herpesvirus 7 (HHV-7) and cytomegalovirus) may not explicitly manifest themselves but become activated due to stress or other factors and, by suppressing the immune system, "open the gate" for more dangerous viruses. In this regard, research and development of new effective antiviral drugs is a priority task of medicinal chemistry.

Benzazines are heterocycles of great potential which, due to their interaction with various molecular targets, can serve as the most important scaffold for the development of effective therapeutic drugs, including those for antiviral therapy. This is evidenced by regularly published reviews on the biological activity of quinolines, quinoxalines, and quinazolines, which, as a rule, are devoted to a specific class of compounds and the discussion of various types of biological activity. At the same time, no separate review articles on the antiviral activity of benzazines can be found.

Promising directions for the use of quinoline derivatives are outlined in a review by Goncharuk et al.° published in 2018. The discussion of antiviral activity is limited by data on compounds effective against Japanese encephalitis virus and HIV.

Quinoxaline derivatives are the subject of increased interest from researchers due to the wide spectrum of their biological activity. A 2015 review by Pereira et al.° noted the ability of quinoxalines to inhibit the replication of herpes simplex viruses of types 1 and 2 (HSV-1 and HSV-2), cytomegalovirus, varicella zoster and shingles viruses. Among the quinoxalines, potent inhibitors of HIV-1 reverse transcriptase activity and HIV-1 replication in tissue cultures have been identified. Quinoxalines, the target of which is the NS1 protein of influenza virus which plays a central role in suppressing the host cell interferon response, facilitation of the replication, and spread of the virus, are especially represented. Tariq et al.° provided data on the antiHIV activity of quinoxaline derivatives as non-nucleoside reverse transcriptase inhibitors (NNRTIs) and HIV integrase inhibitors as part of a review of the antiviral properties of quinoxalines published in 2018.

In the review by Khan et al.° from 2015° devoted to derivatives of quinazolines and quinazolinones, there is no separate section on antiviral agents; only 3-substituted 2-phenylquinazolines with antiviral activity are discussed.
nevirapine).

Figure 1. The structures of quinoline (1), quinoxaline (2), quinazoline (3).

The review published in 2018 by Alagarsamy et al. contains a section on the antiviral activity of quinazolines which supplies data on derivatives active against HIV-1 and HIV-2, cytomegalovirus, adenovirus type 2, HSV-1, tobacco mosaic virus, vaccinia virus; only a few compounds with moderate activity against influenza virus were reported.

The purpose of this minireview is to update the data on the antiviral activity of benzazine derivatives of quinoline (1), quinoxaline (2), and quinazoline (3) (Fig. 1), consider potential molecular targets of benzazine antiviral agents, and analyze the structure–activity relationship according to data published in 2015–2020.

Quinolines exhibiting antiviral activity

Compounds that are active against HIV-1 have been identified among quinoline derivatives. The main targets of potential antiHIV drugs are viral proteins involved in the intracellular multiplication of HIV (reverse transcriptase, integrase, and protease), as well as viral and cellular proteins involved in the attachment of viral particles to the cell. Thus, bromine- and chlorine-substituted chalcones 4a–e (Fig. 2) exhibited a high degree of inhibition of HIV reverse transcriptase.

Testing of 8-(naphthalen-1-yl)-substituted quinolines 5–8 (Fig. 3) revealed promising inhibitors of HIV-1 ribo-
nuclease H (RNase H) (Table 1). In vitro studies showed that 7-isopropoxy-8-(naphthalen-1-yl)quinoline (5) acts in the early stages of viral replication prior to viral assembly and budding. Compound 5 inhibits the activity of RNase H and binds directly to HIV-1 reverse transcriptase. In addition, additive inhibitory activity against pseudotyped viruses has been noted when quinoline 5 is competitively dosed with the clinically used non-nucleoside reverse transcriptase inhibitor (NNRTI) efavirenz. When tested against an NNRTI-resistant HIV-1 isolate, compound 5 showed a 5.1-fold decrease in the half-maximal inhibitory concentration (IC50) while the activity of the reference drug efavirenz decreased by 7.6. These results indicate that quinoline 5 is a potential lead compound in the development of new HIV-1 RNAH inhibitors.

A wide range of quinoline derivatives 9–11 (Fig. 4) have been studied as novel inhibitors of HIV penetration.

Table 1. Activity, cytotoxicity, and selectivity of 8-(naphthalen-1-yl)-substituted quinolines 5–8 against HIV-1 in the TZM-bl cell line.*

| Compound | IC50, μM | CC50, μM | SI |
|----------|----------|----------|----|
|          | Strain HXB2 | Strain YU2 | Strain 89.6 | Cell line TZM-bl |
| 5        | 6.7 ± 0.9 | 8.9 ± 0.6 | 4.7 ± 1.6 | 68.5 ± 17.1 | 14.6 |
| 6        | >100      | >100      | >100      | >100       | – |
| 7        | 61.4 ± 3.3 | 77.6 ± 10.8 | 56.6 ± 0.8 | 95.0 ± 13.1 | 1.7 |
| 8        | 12.6 ± 1.6 | 14.0 ± 0.8 | 16.8 ± 3.0 | 69.3 ± 4.9  | 5.5 |
| Efavirenz| 0.0009 ± 0.0002 | 0.0023 ± 0.0003 | – | Not effect | – |

* IC50 – half-maximal inhibitory concentration, CC50 – half-maximal cytotoxic concentration, SI – selectivity index (CC50/IC50).

Figure 2. The structures of quinoline derivatives 4a–e and their half-maximal inhibitory concentration (IC50) in regards to HIV reverse transcriptase (IC50 0.23 mg/ml for antiHIV drug nevirapine).

Figure 3. The structures of quinoline derivatives 5–8.

Compound 10a (Fig. 5) showed the highest in vitro activity against HIV-1 strains HIV-1BV59 and HIV-1UG070 in TZM-bl cell lines (IC50 3.35 ± 0.87 and 2.57 ± 0.71 μM, respectively). Its ability to inhibit entry into the target cell (IC50 1.40 ± 0.28 μM, therapeutic index
Figure 4. The structures of quinoline derivatives 9–11.

Figure 5. The structure of quinoline derivative 10a, a potential HIV inhibitor.

Of a number of synthesized 6-(1,2,3-triazol-1-yl)-substituted quinolones, compounds 12a,b (Fig. 6) were identified that are capable of inhibiting the activity of the neuraminidase of wild-type influenza virus (WT). It was noted that a change in the position of the 1,2,3-triazole fragment as well as a decrease in the size of the R substituent lead to the loss of anti-influenza activity, probably because of disruption of the patterns of interaction with targets.9

Compounds 12a,b at a concentration of 50 μM most effectively inhibited neuraminidase of wild-type influenza virus H3N2 by 89.0 and 94.8%, respectively. For comparison, oseltamivir (OST) at the same concentration inhibited neuraminidase activity by 100%. A study of the efficacy of compound 12b against circulating WT and OST-resistant influenza A and B strains showed that quinolone 12b has some advantages over OST. Although OST was more effective against wild strains, the IC50 values for compound 12b did not change significantly in the presence of OST resistance mutations. Thus, the ratio of IC50 of OST-resistant strains to IC50 of WT strains in the case of compound 12b was 0.13, 3.0, and 1.4 for influenza virus strains A (H3N2), A (H1N1), and B, respectively. Similar IC50 ratios for OST increased to 27, 380, and 5.3, respectively (Table 2). These results show that (1,2,3-triazol-1-yl)-substituted quinolones may be of interest for the development of new anti-influenza drugs against OST-resistant virus strains.

In recent years, data have appeared on the ability of quinoline derivatives 13–15 to inhibit the replication of arboviruses such as Zika (ZIKV) and chikungunya (CHIKV).10 2,8-Bis(trifluoromethyl)quinolines 13a and 14 (Fig. 7, Table 3) showed the highest antiZIKV activity (half-maximal inhibitory concentration (EC50) of 1.4 ± 0.09 316 ± 27 243, respectively). The structures of quinoline derivatives 12a,b are given as the process of fusion with the target cell (IC50 0.96 ± 0.28 μM, TI 55.83) as well as the replication of ZIKV in the Vero cell line.

Figure 6. The structures of quinoline derivatives 12a,b.

Figure 7. The structures of quinoline derivatives 13–15.

Table 2. Efficiency of inhibition of WT and OST-resistant influenza strains by compound 12b and the comparison drug OST

| Compound | IC50, μM | IC50 (OST-resistant strain) / IC50 (WT strain) |
|----------|---------|---------------------------------------------|
| A/H3N2 WT | 19.90 ± 1.3 | 0.15 ± 0.032 |
| A/H3N2 E119V | 2.60 ± 0.8 | 4.19 ± 0.16 | 0.13 | 27.0 |
| A/H1N1 WT | 3.50 ± 0.9 | 0.21 ± 0.011 |
| A/H1N1 H275Y | 10.60 ± 0.9 | 79.94 ± 3.2 | 3.00 | 380.0 |
| B WT | 22.00 ± 1.1 | 16.00 ± 2.9 |
| B R152 K | 30.00 ± 1.6 | 85.00 ± 5.4 | 1.40 | 5.30 |

Table 3. Activity, cytotoxicity, and selectivity of 2,8-bis(trifluoromethyl)quinolines 13–15 in respect to replication of ZIKV in the Vero cell line*

| Compound | EC50, μM | CC50, μM | SI |
|----------|---------|----------|----|
| 13a | 0.8 ± 0.06 | 195 ± 8.9 | 243 |
| 13b | 2.0 ± 0.1 | 287 ± 21 | 143 |
| 14 | 0.8 ± 0.03 | 189 ± 10 | 226 |
| 15 | 1.4 ± 0.09 | 316 ± 27 | 225 |
| Mefloquine | 3.6 ± 0.3 | 212 ± 14 | 58 |

* EC50 – half-maximal effective concentration, SI – selectivity index (CC50/IC50).
The EC50 for the DENV-2 is 3.9 and 9.2 revealed quinolones capable of inhibiting DENV-2 BHK-21 cells infected with dengue virus type 2 (DENV-2) compounds.

Phenotypic screening of 7000 compounds using BHK-21 cells infected with dengue virus type 2 (DENV-2) revealed quinolones capable of inhibiting DENV-2 BHK-21 cells infected with dengue virus type 2 (DENV-2) compounds.

Figure 8. The structures of quinoline derivatives 16a-d.

Table 4. Cytotoxicity, activity, and selectivity of compounds 16a-d against ZIKV and CHIKV in the Vero cell line

| Compound | CC50, μM | ZIKV EC50, μM | SI | CHIKV EC50, μM | SI |
|----------|---------|---------------|----|----------------|----|
| 16a      | 502 ± 4.38 | 0.76 ± 0.028 | 669.9 | 2.85 ± 0.12 | 176.2 |
| 16b      | 669 ± 4.33 | 0.75 ± 0.011 | 892.9 | 1.06 ± 0.077 | 631.7 |
| 16c      | 1113 ± 6.11 | 0.79 ± 0.005 | 1409.7 | 2.77 ± 0.18 | 402 |
| 16d      | 443 ± 5.1 | 0.81 ± 0.009 | 547.5 | 2.7 ± 0.13 | 164.2 |
| Ribavirin | 297 ± 4.95 | 3.95 ± 0.095 | 75.2 | 2.42 ± 0.49 | 122 |

The synthesized compounds 16a-d were found to have antiviral activity both in the early and post-infectious stages of the action of ZIKV and CHIKV which makes them excellent candidates for the development of antiZIKV and antiCHIKV drugs.

Figure 9. The structures of fluoroquinolones 17 and 18.

Diarylpyrazolyl-substituted quinoline 19 (Fig. 10), which exhibited higher inhibitory activity against DENV-2 (IC50 0.81 μM, SI >246.91), compared with ribavirin (IC50 12.61 μM, SI 4.47) was considered12 as a potential antiviral drug against DENV. It was shown that compound 19 also effectively inhibits other serotypes of DENV, reduces the clinical manifestations of the disease and mortality in mice infected with DENV.

The quinoline skeleton is currently considered as the basis for the development of effective antiviral drugs for the prevention and treatment of non-polio enterovirus infection. As a result of the search for drugs with direct action on the conservative multifunctional viral protein C2 involved in membrane rearrangement, viral assembly, and viral RNA replication, compound 20 was identified (Fig. 10) demonstrating high broad-spectrum antiviral activity against 5 tested strains of non-polio enteroviruses (two EV-D68 strains (USA, Kentucky and USA, Missouri), two EV-A71 strains (Taiwan, Tainan and USA, Alaska), and one CVB3 strain), and also showed high microsomal stability with a half-life of 114.7 min13 (Table 5). Research of this kind is a step forward in the development of sought-after antiviral drugs against non-polio enteroviruses.

Quinoxalines exhibiting antiviral activity

Potential inhibitors of HIV-1 integrase were designed and then synthesized based on a created pharmacophore model and 3D analysis of quantitative structure–activity relationship (3D-QSAR). 2,3-Diaryl-substituted quinoxalines 21a,b (Fig. 11) showed the best antiHIV activity and low toxicity (Table 6). It was noted that lipophilic and bulky substituents at positions 2 and 3 of the quinoxaline fragment increase the activity of compounds 21a,b against HIV as compared to unsubstituted quinoxalines or quinoxalines with less bulky substituents.14
Virtual library screening, molecular docking, and 3D-QSAR study allowed the structure to be optimized followed by synthesis of quinoxalines 22 and 23 (Fig. 12) which showed high antiviral activity against wild and mutant (K103N) HIV reverse transcriptase. Compound 22 showed the highest antiviral activity against wild and mutant (K103N) HIV reverse transcriptase.

Quinoxalines 24a, b and 25a, b were identified to have low toxicity and high activity in the micromolar level ranging from 0.06–3.8 μM. The sulfanyl group. Compound 24a which showed the highest activity against the CV-B5 virus (EC50 0.09 ± 0.01 μM) was chosen as the lead compound. 16 Given the high activity in combination with low cytotoxicity, quinolines 24a, b and 25a, b can serve as the basis for the development of new drugs for treatment of infections caused by enteroviruses.

**Quinazolines exhibiting antiviral activity**

Data on 4-substituted quinazolines with antiviral activity are limited to isolated examples. 2-Sulfanylquinazolines 26a, b containing the chalcone fragment (Fig. 14) (EC50 156.4 and 138.1 μg/ml, respectively) are superior to ribavirin (EC50 436.0 μg/ml) in activity against tobacco mosaic virus.

A number of 4-arylaminoquinazolines 27a–d effectively suppress the replication of human cytomegalovirus. Conjugates 28a, b and 29a, b of 4-arylaminoquinazolines with the sesquiterpene lactone artemisinin were synthesized (Fig. 15) exhibiting antimalarial action. It was shown that derivatives 28a, b and 29a, b are superior in anti-cytomegalovirus activity to ganciclovir.

The antiviral activity of 2-substituted quinazolinones 30 obtained by cycloadition of C-(diethoxypyrophosphoryl)-N-methyl nitro ene 31 to 3-substituted 2-vinylquinazolin-3(3H)-ones 32 in regards to a wide range of DNA and RNA viruses was studied. Several derivatives were active against two types of varicella zoster virus (TK+ and TK-) with EC50 values of 5.4–13.6 μM, as well as against human cytomegalovirus (EC50 8.94–13.2 μM). 19 (Table 7).

3-Aryl- and 3-benzyl-substituted compounds 30b–i are superior in activity against the TK+ (07-1) strain of varicella zoster virus compared to reference drugs acyclovir and brivudine (EC50 39.2 and 31.9 μM,
respectively); in this case, activation by a viral enzyme is required. At the same time, the activity of quinazolinones 30b–i against the TK+ (OKA) strain was found to be 360–587 times lower than that of the above reference drugs. It was noted that 6-bromo-substituted 2-isoxazolidinylquinazolin-4(3H)-ones are superior in activity to analogs without

**Figure 15.** Structures of quinazoline derivatives 27–29 and their activity against human cytomegalovirus (laboratory strain AD169-GFP) (for ganciclovir, EC50 2.60 ± 0.50 μM).

**Table 7.** Activity of 2-isoxazolidinyl-substituted quinazolin-4(3H)-ones 30a–s against varicella zoster virus and human cytomegalovirus

| Compound | X | R | EC50, μM | EC50, μM |
|----------|---|---|---------|---------|
| cis-30a  | Br | H | >100    | >100    |
| trans-30a| Br | H | >100    | 66.87   |
| trans-30b| Br | Bn| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30c| Br | OCNMe₂CH₂| 10.3 ± 1 | 5.4 ± 1.0 |
| trans-30d| Br | OCNMe₂CH₂| 8.3 ± 1  | 5.8 ± 1.4 |
| trans-30e| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30f| Br | Me| >4      | >4      |
| trans-30g| Br | Et| >20     | >20     |
| trans-30h| Br | Bn| >100    | >100    |
| trans-30i| Br | Bn| >100    | >100    |
| trans-30j| Br | Me| >4      | >4      |
| trans-30k| Br | Et| >20     | >20     |
| trans-30l| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30m| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30n| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30o| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30p| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30q| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30r| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| trans-30s| Br | OCNMe₂CH₂| 13.5 ± 7.1 | 13.6 ± 9.1 |
| Acyclovir | | | 1.55 ± 1.0 | 39.2 ± 3.6 |
| Ganciclovir| | | 16.9 ± 6.9 | 7.7 ± 0.9 |
| Brivudine | | | 0.023 ± 0.008 | 31.9 ± 16.1 |
| Cidofovir | | | 1.5 ± 0.2 | 1.7 ± 0.4 |
a substituent in the benzene ring. Compounds 30d-f-i are comparable in activity against AD-169 and Davis strains of human cytomegalovirus (EC50 8.94–13.2 μM) to ganciclovir (EC50 16.9 and 7.7 μM) but inferior to cidofovir (EC50 1.5 and 1.7 μM)19 (Table 7).

Based on 1-allylquinazoline-2,4-diones containing substituted benzoyl or benzyl groups at position 3, isoxazolidine derivatives 33 and 34 were obtained (Fig. 16) which showed high activity against varicella zoster virus and human cytomegalovirus.21

![Figure 16. Structures of quinazoline derivatives 33 and 34 and their activity against varicella zoster virus and human cytomegalovirus.](image)

Table 8. Cytotoxicity, activity, and selectivity of quinazoline derivatives 35a-c in relation to hepatitis B virus in the HepG2 cell line

| Compound | IC50, mM | SI | IC50, mM | SI |
|----------|---------|----|---------|----|
| 35a      | 71.51   | 4.07 | 17.57   |
| 35b      | 21.13   | 1.54 | 13.72   |
| 35c      | 15.79   | 0.71 | 22.74   |
| Lamivudine >100 | <0.1 | >1000 |

![Figure 17. The structures of quinazoline derivatives 35a-c.](image)

It was found that derivatives of 6-iodo-3-(3-trifluoromethylphenyl)quinazolin-4(3H)-ones containing thiosemicarbazone, pyrazole, or azomethine fragments in position 2 have weak or moderate activity against H5N1 influenza virus and are inferior to the reference drug zanamivir.22

A wide range of 2-benzylsulfanyl-3-(thiophen-2-ylmethyl)quinazolin-4(3H)-ones were studied, and it was shown that compounds 35a-c (Fig. 17) are the most promising for the development of non-nucleoside drugs on their basis for the treatment of hepatitis B virus (Table 8).

![Figure 18. Structures of 3-hydroxy-6-(1,2,3-triazolyl)quinazoline-2,4(1H,3H)-diones 36a,b and their activity and selectivity against vaccinia virus (comparison drugs brivudine and cidofovir) and adenovirus type 2 (comparison drugs cidofovir, zalcitabine, and alovudine).](image)
Figure 19. The structures of quinazoline derivatives 37–43.

Table 9. Activity, cytotoxicity, and selectivity of 2,4-disubstituted quinazolines 37–43 in relation to influenza A/WSN/33 (H1N1) virus in the cell line HEK293T-Gluc

| Compound | IC<sub>50</sub>, μM | CC<sub>50</sub>, μM | SI |
|----------|-------------------|------------------|----|
| 37       | 3.70 ± 0.82       | > 100            | 27.03 |
| 38       | 8.64 ± 1.76       | > 100            | 11.57 |
| 39       | 4.19 ± 0.43       | > 100            | 23.87 |
| 40       | 7.18 ± 1.89       | > 100            | 13.93 |
| 41a      | 1.88 ± 0.10       | 23.28 ± 2.91     | 12.38 |
| 41b      | 1.29 ± 0.01       | 59.94 ± 3.04     | 46.46 |
| 41c      | 9.04 ± 0.57       | 15.86 ± 0.58     | 1.75 |
| 42a      | 3.88 ± 0.47       | 36.64 ± 2.24     | 9.44 |
| 42b      | 3.43 ± 0.54       | > 100            | 29.15 |
| 43a      | 6.84 ± 0.68       | 29.43 ± 0.95     | 4.30 |
| 43b      | 3.83 ± 0.15       | > 100            | 26.11 |
| 43c      | 5.00 ± 1.37       | > 100            | 20.00 |
| 43d      | 11.47 ± 0.54      | > 100            | 8.72 |
| Ribavirin| 15.36 ± 0.93      | > 100            | 6.51 |

Figure 20. The structures of 2-(thiophen-2-yl)-2,3-dihydroquinazolin-4(1H)-ones 44.

To conclude, quinoline and quinoxaline derivatives are active against a large number of RNA viruses. Quinolines and quinoxalines are of interest for the development of drugs that block one of the stages of HIV life cycle. Among them, new inhibitors of reverse transcriptase, RNase H and HIV integrase, as well as inhibitors of HIV penetration into the target cell have been identified. Quinolines and quinoxalines have also been found to be active against RNA viruses such as arboviruses and enteroviruses. Quinoline derivatives are promising for the development of new anti-influenza drugs against oseltamivir-resistant virus strains.

Quinazolin-4-one derivatives are characterized by activity against DNA viruses: adenovirus, hepatitis B virus, varicella zoster virus, and cytomegalovirus. Apparently, the structural similarity with purines determines the ability to inhibit DNA polymerase in the same way as acyclovir. Recently, successful examples of the design of anti-influenza agents based on 4-substituted quinazolines are noted.

The data presented in the review indicate the enormous potential of benzazines in the design of drugs suitable for the treatment of diseases caused by RNA and DNA viruses.

This work was supported by the Ministry of Science and Higher Education of the Russian Federation (project No. FEUZ-2020-0058 (N687.428.223/20)).
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