Development of new effective drugs with low side effects and definite chemical characteristics needs identification of bioactive scaffolds for further structural optimization. New synthesized derivatives of 4-hetaryl-5-amino-1-aryl-1H-1,2,3-triazoles and 3H-[1,2,3]triazolo[4,5-b]pyridines were tested for antican-cer activity using 60 human tumor cell lines within 9 cancer types. The selective influence of (5-amino-1H-1,2,3-triazol-4-yl)quinazolin-4(3H)-ones: 2-(5-amino-1-(4-chlorophenyl)-1H-1,2,3-triazol-4-yl)quinazolin-4(3H)-one and 2-(5-amino-1-phenyl-1H-1,2,3-triazol-4-yl)-6-bromoquinazolin-4(3H)-one on ovarian cancer OVCAR-4 cells with growth percentage (GP) = -4.08 and 6.63%, respectively, was found. The derivative 5,7-diamino-3-(3-(trifluoromethyl)phenyl)-3H-[1,2,3]triazolo[4,5-b]pyridine-6-carbonitrile possessed high activity towards lung cancer EKVX cells (GP = 29.14%). The compounds were shown to be less toxic than doxorubicin towards non-tumor human embryonic kidney cells of HEK293 line. Thus, the results of our study confirm the anticancer potential of compounds based on 5-amino-1-aryl-1H-1,2,3-triazoles scaffolds and their fused poly cyclic derivatives.

**Keywords:** 5-amino-1-aryl-1H-1,2,3-triazoles, 3H-[1,2,3]triazolo[4,5-b]pyridines, quinazolinones, thiazoles, 1,3,4-oxadiazoles, antiproliferative activity, anticancer activity.
in the combination with 3',4'-dimethoxyflavone or carboxyamidotriazole with 1-methyl-L-tryptophan resulted in higher anticancer activity than it was found for a single agent treatment [2].

In previous studies, we revealed two 5-aminoo-1H-1,2,3-triazole-4-carboxamides (5-amino-N-(2,5-dichlorophenyl)-1-(4-methylphenyl)-1H-1,2,3-triazole-4-carboxamide and 5-amino-N-(2,4-dimethoxyphenyl)-1-(4-fluorophenyl)-1H-1,2,3-triazole-4-carboxamide) that possessed the antiproliferative activity and were highly active towards renal cancer RXF 393 cells (GP = -13.42%) and the CNS cancer SNB-75 cells (GP = -27.30%) respectively [7] (Fig. 2). Moreover, the amino-fused 1,2,3-triazole: 5-oxo-4,5,6,7,8,9-hexahydrobenzo[4,5]thieno[3,2-e] [1,2,3]triazolo[1,5-a]pyrimidine-3-carboxamide possessed a selective action towards melanoma SK-MEL-5 cells (GP = -31.50%) [8] (Fig. 2). Other scaffolds such as 4-(1H-indole-3-carbonyl)-5-amino-1H-1,2,3-triazoles, 4-arylsulfonyl-5-amine-1H-1,2,3-triazoles, [1,2,3]triazolo[1,5-a]quinazolines demonstrated moderate or low antiproliferative activity [7, 8] (Fig. 2). Recently, ethyl 5-amino-1-[4-(6-oxo-4,5-dihydropyridazin-3-yl)phenyl]-1H-1,2,3-triazole-4-carboxylate showed potent inhibitory activity against B-Raf kinase [9]. In addition to the anticancer studies, the 5-amino-1,2,3-triazole-4-carboxamide motif was found in compounds with the activity against Trypanosoma cruzi parasite [10] and compounds inhibiting LexA autoproteolysis and the bacterial SOS response [11].

Various 5-amino-1,2,3-triazoles were obtained by using an efficient synthetic method from the
available reagents via eco-friendly base-catalyzed 
cycloaddition reaction of azides with acetonitriles 
activated by aminodicyanovinyl fragment [12, 13], 
1,3-thiazole [14, 15], 1,2,4-/1,3,4-oxadiazole [14], 
pyrroles and indoles [16] rings and with possible the 
simultaneous cascade processes leading to polycyclic 
systems [17, 18]. Additionally, the azide [3 + 2] cy-
cloadditions can be performed at room temperature 
good to excellent yields of products in the presen-
ce of catalytic amounts of pyrrolidine (5-10 mol%) 
[19, 20] according to organocatalytic methodology 
[21-24]. Those protocols might be successfully used 
for the variation of the fragment in position 4 of the 
triazole, involving the electron-withdrawing hetero-
cyclic core for an extended structure-activity inves-
tigation focused at drug-like properties. Moreover, 
the amino group formed in the reaction can be used 
for the annulation of the aromatic rings in a one-pot 
manner to synthesise the condensed polycyclic scaf-
dolds via domino-process. That allows a rapid paral-
ellel synthesis and fast generation of the combinatori-
ial libraries for screening of the biological activity 
[25, 26].

The present work was aimed on evaluation of 
the anticancer activity of new 5-amino-1-aryl-1H-
1,2,3-triazoles scaffolds and their fused derivatives 
synthesized at 20°C in a short time via azides cyclo-
condensation, as described [12, 14]. The results of 
performed in vitro study of the anticancer activity of 
the synthesized compounds towards 60 cancer cell 
lines suggested the most promising lead candidates 
with selective influence suitable for further structur-
al optimization. The utility of versatile azides and 
nitriles with substituents of different nature in the 
synthetic protocol for the 5-amino-1H-1,2,3-triazole 
derivatives allowed evaluating the dependence be-
tween structure of the side chain and anticancer ac-
tivity.

Materials and Methods

**Studied compounds.** The 5-amino-1-aryl-1H-
1,2,3-triazoles 3-6 and 3H-[1,2,3]triazolo[4,5-h] 
pyridines 7 derivatives were synthesized earlier 
at the Department of Organic Chemistry of 
Ivan Franko National University of Lviv, Ukraine 
[12, 14]. Properties and spectral characteristics of 
compounds used in this study have been presented 
in [12, 14]. The purity of compounds was established 
to be higher than 97%, based on liquid chromatog-
raphy–mass spectrometry examination. A 10 mM 
stock solution of the testing samples were prepared 
by dissolving of compounds in dimethyl sulfoxide 
(DMSO, Sigma-Aldrich, St. Louis, Missouri, USA). 
Then, working solutions of these compounds were 
prepared using culture medium. Doxorubicin (Dox) 
as purchased from Actavis S.R.L. (Bucharest, Ro-
mania) and used as a positive control.

**Cell cultures.** Human lung adenocarcinoma 
A549 cells (non-small cell lung cancer cells), human 
cervical adenocarcinoma HeLa cells, human em-
bryonic kidney HEK293 cells were obtained from 
Cell Collection of R.E. Kavetsky Institute of Ex-
perimental Pathology, Oncology and Radiobiology 
(Kyiv, Ukraine). Human ovarian carcinoma Skov3 
cells were obtained from the American Type Cul-
ture Collection (ATCC, Manassas, VA, USA) and 
were donated by Dr. Sci. O. Stasyk (Institute of Cell 
Biology, National Academy of Sciences of Ukraine, 
Lviv, Ukraine). Cells were grown in the RPMI-1640 
(PPA, Vienna, Austria) or DMEM (Sigma-Aldrich, 
St. Louis, Missouri, USA) medium supplemented 
with 10% of fetal bovine serum (Biowest, Nuaille, 
France). Cells were cultivated in the CO₂-thermo-
state at 37°C in atmosphere of 95% air and 5% CO₂.

**Anticancer assay using NCI protocol.**

Accordingly, to the protocol of the Drug Evaluation 
Branch at the National Cancer Institute in Bethesda 
(USA), a primary antiproliferative assay was per-
formed within nine cancer types of approximately 
60 human tumor cell lines panel. The tested com-
 pounds were added to the culture at a single con-
centration (10⁻⁵ M) and left for 48 h incubation. Sul-
forhodamine B (SRB) was used as protein binding 
dye for the end-point determinations. The percent 
growth of the treated cells when compared to 
the untreated control cells was taken for each tested 
compound. The percentage of growth inhibition was 
evaluated spectrophotometrically versus controls 
(untreated cells). 100% corresponds to growth seen 
in the untreated cells, while 0% indicates a lack of 
growth over the course of the assay (i.e. equal to the 
number of cells at time zero). -100% results when all 
cells were killed.

**Cell proliferation MTT assay.** In vitro evalua-
tion of the antiproliferative activity of the syn-
thesized compounds and doxorubicin, used as a 
reference drug control, towards cancer cell lines 
was measured by using the 3-(4,5-dimethylthiazol-
2-yl)-2,5-diphenyl-tetrazolium bromide (MTT, 
Sigma-Aldrich, St. Louis, Mo, USA) test [27]. Tu-
mor and pseudo-normal cells were seeded for 24 h 
in 96-well microtiter plates at a concentration of
5,000 cells/well (100 μl/well). Then cells were incubated for next 72 h with various additions of the synthesized compounds or doxorubicin (0-100 μM). MTT that is converted to dark violet, water insoluble MTT formazan by the mitochondrial dehydrogenases, was used to determine viable cells according to the Sigma-Aldrich protocol. Absorbance Reader BioTek ELx800 (BioTek Instruments, Inc., Winooski, VT, USA) was used for reaction results measurement.

Statistical analysis. All data are presented as the mean (M) ± standard deviation (SD), n = 4. Results were analysed and illustrated with GraphPad Prism (version 6; GraphPad Software, San Diego, CA, USA). Statistical analyses were performed using two-way ANOVA with Dunnett multiple comparisons test. P-value of < 0.05 was considered as statistically significant.

Results and Discussion

Convenient synthetic protocols for cycloaddition reactions of arylazides 1 with activated acetonitriles 2 allow rapid generation of compound libraries with structural diversity (Scheme). It is noteworthy that the reaction of aryl azides 1 with acetonitriles 2 in the presence of sodium methylate in methanol occurred at 20°C and fully satisfies “click”- and “green” chemistry requirements. However, heating of the reagents is required for the highest conversion of reactants in a reaction of aryl azides 1 with acetonitriles 2c-e leading to compounds 3, 6, 7. The list of the studied substituents, as well as the full structure of compounds 3-7 tested for in vitro anticancer activity, is presented in Table 1.

Evaluation of anticancer activity in vitro. The synthesized 5-amino-1-aryl-1H-1,2,3-triazoles 3-6 and 3H-[1,2,3]triazolo[4,5-b]pyridines 7 were submitted and evaluated at the single concentration of 10^{-5} M towards a panel of the approximately 60 cancer cell lines. Human tumor cell lines were derived from nine different cancer types: leukemia, non-small cell lung, colon, CNS, melanoma, ovarian, renal, prostate and breast cancers. The primary anticancer assays were performed according to the NCI (USA) protocol (http://dtp.nci.nih.gov) described elsewhere [28-31]. The results for each compound are reported as the percentage of growth (GP) (Table 1). Range of growth (%) shows the lowest and the highest growth found among different cancer cell lines. The most active compounds are shown in Table 1.

In comparison to previously studied 5-amino-1,2,3-triazole-4-carboxamide [7], the newly synthesized compounds 3-7 displayed slight or low activity at in vitro screening (selected results are shown in Table 1). The 5-amino-4-(4-oxo-3,4-dihydroquinazolin-2-yl)-1H-1,2,3-triazoles 3 possess the inhibiting activity towards the ovarian cancer OVCAR-4 cell line. In particular, 1,2,3-triazoles 3a and 3b were found to be the most active among the quinazolines with growth inhibition GP = - 4.08 and 6.63%, respectively.

On the contrary, the selective influence of [1,2,3]triazolo[4,5-b]pyridines 7a-i on single lung cancer cell line was observed, specifically the compound 7a was highly active on EKVX cell line (GP = 29.14%). Unfortunately, compounds of another 3 scaffolds types 4, 5, and 6 were found to possess

![Scheme. Synthesis of 5-amino-1-aryl-1H-1,2,3-triazole scaffolds](image-url)
**Table 1. Anticancer screening data at 10^{-3} M for selected 5-amino-1-aryl-1H-1,2,3-triazoles**

<table>
<thead>
<tr>
<th>No</th>
<th>Compound</th>
<th>Mean GP%</th>
<th>Range GP %</th>
<th>The most sensitive cell lines</th>
<th>Growth, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>3a</td>
<td>![Compound Image]</td>
<td>76.65</td>
<td>-4.08 to 106.52</td>
<td><strong>OVCAR-4 (Ovarian Cancer)</strong> SNB-75 (CNS Cancer) T-47D (Breast Cancer) 549/ATCC (Lung Cancer)</td>
<td>-4.08 24.75 27.21 40.57</td>
</tr>
<tr>
<td>3b</td>
<td>![Compound Image]</td>
<td>81.27</td>
<td>6.63 to 111.89</td>
<td><strong>OVCAR-4 (Ovarian Cancer)</strong> U251 (CNS Cancer) NCI-H460 (Lung Cancer) HCT-116 (Colorectal Cancer)</td>
<td>6.63 38.57 43.51 48.22</td>
</tr>
<tr>
<td>3c</td>
<td>![Compound Image]</td>
<td>97.08</td>
<td>68.84 to 119.80</td>
<td>SR (Leukemia) NCI-H522 (Lung Cancer) HCT-116 (Colorectal Cancer)</td>
<td>69.47 68.84 70.27</td>
</tr>
<tr>
<td>4</td>
<td>![Compound Image]</td>
<td>94.78</td>
<td>75.95 to 108.76</td>
<td>NCI-H522 (Lung Cancer) T-47D (Breast Cancer) A549/ATCC (Lung Cancer) UACC-257 (Melanoma)</td>
<td>75.95 76.44 77.80 79.56</td>
</tr>
<tr>
<td>5</td>
<td>![Compound Image]</td>
<td>96.04</td>
<td>74.39 to 109.30</td>
<td>A498 (Kidney Cancer) NCI-H522 (Lung Cancer) A549/ATCC (Lung Cancer)</td>
<td>74.39 76.10 79.77</td>
</tr>
<tr>
<td>6</td>
<td>![Compound Image]</td>
<td>100.47</td>
<td>65.45 to 116.00</td>
<td>NCI-H522 (Lung Cancer)</td>
<td>69.47 68.84 70.27</td>
</tr>
<tr>
<td>7a</td>
<td>![Compound Image]</td>
<td>87.34</td>
<td>29.14 to 120.64</td>
<td><strong>EKVX (Lung Cancer)</strong> HS 578T (Breast Cancer) T-47D (Breast Cancer) MCF7 (Breast Cancer)</td>
<td>29.14 46.17 48.07 60.15</td>
</tr>
<tr>
<td>7b</td>
<td>![Compound Image]</td>
<td>90.52</td>
<td>45.26 to 110.76</td>
<td><strong>EKVX (Lung Cancer)</strong> HS 578T (Breast Cancer) A549/ATCC (Lung Cancer) NCI-H522 (Lung Cancer)</td>
<td>31.52 45.26 77.20 65.56</td>
</tr>
<tr>
<td>7c</td>
<td>![Compound Image]</td>
<td>91.48</td>
<td>38.20 to 122.37</td>
<td><strong>EKVX (Lung Cancer)</strong> NCI-H522 (Lung Cancer)</td>
<td>38.20 72.33</td>
</tr>
<tr>
<td>7d</td>
<td>![Compound Image]</td>
<td>98.96</td>
<td>39.08 to 122.40</td>
<td><strong>EKVX (Lung Cancer)</strong> HS 578T (Breast Cancer)</td>
<td>39.08 75.96</td>
</tr>
<tr>
<td>7e</td>
<td>![Compound Image]</td>
<td>97.49</td>
<td>43.45 to 118.96</td>
<td><strong>EKVX (Lung Cancer)</strong> T-47D (Breast Cancer)</td>
<td>43.45 73.57</td>
</tr>
</tbody>
</table>
activity below moderate (mostly active on NCI-H522 Lung Cancer cell line) (Table 1). In general, it can be concluded that the replacement of the amide moiety at position 4 of 1H-1,2,3-triazole leads to a decrease or loss of its anticancer activity. Nevertheless, triazoles 3 and 7 studied in current work, with the partially preserved amide moiety remained active. Meanwhile, the introduction of other bioisosters such as thiazole and oxadiazole, led to a decrease in activity, and moreover, the replacement with sulfo- or aryl substituent generally led to activity disappearance (Fig. 3). The introduction of the donor substituent in the aryl moiety at position 1, and any limitation of rotation of this moiety due to the substituent in the ortho position to triazole also assist the activity decrease. Such data suggest a possibility of the same mechanism of action of the studied scaffolds in comparison to known carboxamidotriazole (Fig. 1).

The antiproliferative activity of three 5-amino-1-aryl-1H-1,2,3-triazoles (3a, 3b, and 7a) were evaluated in human carcinoma cell lines of different tissue origin: ovarian (Skov3), cervical (HeLa), lung (A549) and towards human embryonic kidney HEK293 cells using the MTT test. It was found that compound 3a possessed the highest cytotoxic

<table>
<thead>
<tr>
<th>No</th>
<th>Compound</th>
<th>Mean GP%</th>
<th>Range GP %</th>
<th>The most sensitive cell lines</th>
<th>Growth, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>7f</td>
<td></td>
<td>96.08</td>
<td>43.61 to 119.29</td>
<td>EKVX (Lung Cancer), T-47D (Breast Cancer), HS 578T (Breast Cancer), MCF7 (Breast Cancer)</td>
<td>43.61, 67.15, 68.79, 71.42</td>
</tr>
<tr>
<td>7g</td>
<td></td>
<td>95.58</td>
<td>44.78 to 122.77</td>
<td>EKVX (Lung Cancer), HS 578T (Breast Cancer), NCI-H522 (Lung Cancer), A549/ATCC (Lung Cancer)</td>
<td>44.78, 61.63, 69.36, 81.56</td>
</tr>
<tr>
<td>7h</td>
<td></td>
<td>98.00</td>
<td>50.24 to 119.60</td>
<td>EKVX (Lung Cancer), NCI-H522 (Lung Cancer), A549/ATCC (Lung Cancer)</td>
<td>50.24, 77.24, 84.51</td>
</tr>
<tr>
<td>7i</td>
<td></td>
<td>93.43</td>
<td>63.00 to 115.30</td>
<td>EKVX (Lung Cancer), NCI-H522 (Lung Cancer), UO-31 (Kidney Cancer)</td>
<td>63.00, 65.83, 70.22</td>
</tr>
</tbody>
</table>

Fig. 3. Structure-activity dependence
Fig. 4. Level of cytotoxicity of three 5-amino-1-aryl-1H-1,2,3-triazoles (3a, 3b, and 7a) towards human carcinoma cell lines of different tissue origin: ovarian (Skov3), cervical (HeLa), lung (A549), and towards non-tumor cells (HEK293). After a total experimental time (72 h), cell vitality was detected by the MTT assay. *\(P \leq 0.05\); **\(P \leq 0.01\); ***\(P \leq 0.001\) (difference compared with the no treated control cells). Dox – doxorubicin

action towards studied tumor cells. Compound 3a inhibited growth of human ovarian carcinoma Skov3 cells with the IC\(_{50}\) of 9.1 μM (Fig. 4, Table 2). The IC\(_{50}\) value for compound 3a was 78.1 μM in human cervical adenocarcinoma HeLa cells, and 28.7 μM – in human lung adenocarcinoma A549 cells (Fig. 4, Table 2). The IC\(_{50}\) value for A549 cells was 99.6 μM for compound 3b (Fig. 4, Table 2). At the highest dose of 100 μM, compound 3b inhibited the growth of Skov3 cells by 12.6 %, and the growth of HeLa cells – by 27.8% (Fig. 4, Table 2). Compound 7a inhibited growth of HeLa cells with the IC\(_{50}\) of 20.4 μM (Fig. 4, Table 2). Compound 7a inhibited Skov3 and A549 cells growth by 26.5 and 44.8%, respectively (Fig. 4, Table 2). Doxorubicin demonstrated higher cytotoxicity towards Skov3, HeLa and A549 tumor cells (IC\(_{50}\) was 0.8 μM, 0.6 μM, and 0.6 μM, respectively, Fig. 4, Table 2).

We have also studied the toxicity of three 5-amino-1-aryl-1H-1,2,3-triazoles (3a, 3b, and 7a) towards human embryonic kidney HEK293 cells. HEK293 cells were relatively non-sensitive to the action of compounds 3a, 3b and 7a. The IC\(_{50}\) level was above 100 μM under compounds 3a and 3b treatment, while IC\(_{50}\) for compound 7a was 38.6 μM (Fig. 4, Table 2). The IC\(_{50}\) value of doxorubicin was 0.5 μM that indicates high cytotoxic effect of the chemotherapeutic drug towards HEK293 cells (Fig. 4, Table 2).

Conclusion. Anticancer activity in vitro for the selected 5-amino-1-aryl-1H-1,2,3-triazoles and their fused polycyclic derivatives 3H-[1,2,3]triazolo[4,5-b]pyridines was evaluated. New 5-amino-1-aryl-1H-1,2,3-triazole scaffold (5-amino-1H-1,2,3-triazol-4-yl)quinazolin-4(3H)-one) was found to possess the antitumor activity with a selective influence on
Taking into account these results, further structure optimization to design more selective and active anticancer agents among 5-amino-1-aryl-1H-1,2,3-triazoles is in progress.

The obtained results allowed identifying the most active (5-amino-1H-1,2,3-triazol-4-yl)quinoxalin-4(3H)-ones: 2-(5-amino-1-(4-chlorophenyl)-1H-1,2,3-triazol-4-yl)quinolin-4(3H)-one and 2-(5-amino-1-phenyl-1H-1,2,3-triazol-4-yl)-6-bromouquinolin-4(3H)-one towards ovarian cancer OVCAR-4 cells with GP = -4.08 and GP = 6.63%, respectively. The 5,7-diamino-3-(3-(trifluoromethyl)phenyl)-3H-[1,2,3]triazolo[4,5-b]pyridine-6-carbonitrile possessed a significant cytotoxic activity towards lung cancer EKVX cells (GP = 29.14%). The most prominent cytotoxic effect was demonstrated by the 2-(5-amino-1-(4-chlorophenyl)-1H-1,2,3-triazol-4-yl)quinolin-4(3H)-one compound towards human ovarian carcinoma Skov3 cells. 2-(5-amino-1-phenyl-1H-1,2,3-triazol-4-yl)-6-bromoquinolin-4(3H)-one and 5,7-diamino-3-(3-(trifluoromethyl)phenyl)-3H-[1,2,3]triazolo[4,5-b]pyridine-6-carbonitrile compounds possessed a remarkable cytotoxic activity towards lung cancer EKVX cells, however, their toxicity was less compared with doxorubicin’s one. These compounds were also less toxic than doxorubicin towards human embryonic kidney HEK293 cells. 2-(5-amino-1-phenyl-1H-1,2,3-triazol-4-yl)-6-bromoquinolin-4(3H)-one and 5,7-diamino-3-(3-(trifluoromethyl)phenyl)-3H-[1,2,3]triazolo[4,5-b]pyridine-6-carbonitrile compounds also showed a remarkable cytotoxic activity towards cervical and lung carcinoma cell lines, however, their toxicity was less compared with doxorubicin’s one. These compounds were also less toxic than doxorubicin towards human embryonic kidney HEK293 cells. A search for more selective and active anticancer agents among 5-amino-1H-1,2,3-triazoles is conducted.

**Table 2. Cytotoxicity indicator (IC₅₀) of three 5-amino-1-aryl-1H-1,2,3-triazoles (3a, 3b, and 7a) and doxorubicin (Dox) towards human carcinoma cell lines of different tissue origin: ovarian (Skov3), cervical (HeLa), lung (A549) and towards pseudo-normal HEK293 cells**

<table>
<thead>
<tr>
<th>Cell line</th>
<th>IC₅₀, μM</th>
<th>IC₅₀, μM</th>
<th>IC₅₀, μM</th>
<th>Dox</th>
</tr>
</thead>
<tbody>
<tr>
<td>Human ovarian carcinoma Skov3 cells</td>
<td>9.1</td>
<td>&gt;100</td>
<td>&gt;100</td>
<td>0.8</td>
</tr>
<tr>
<td>Human cervical adenocarcinoma HeLa cells</td>
<td>78.1</td>
<td>&gt;100</td>
<td>20.4</td>
<td>0.6</td>
</tr>
<tr>
<td>Human lung adenocarcinoma A549 cells</td>
<td>28.7</td>
<td>99.6</td>
<td>&gt;100</td>
<td>0.6</td>
</tr>
<tr>
<td>Human embryonic kidney HEK293 cells</td>
<td>&gt;100</td>
<td>&gt;100</td>
<td>38.6</td>
<td>0.5</td>
</tr>
</tbody>
</table>

**Conflict of interest.** Authors have completed the Unified Conflicts of Interest form at http://ukrbiochemjournal.org/wp-content/uploads/2018/12/coi_disclosure.pdf and declare no conflict of interest.

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на 60 клітинних лініях NCI60 в межах 9 типів раку. Виявлено селективний вплив (5-аміно-1H-[1,2,3]-триазол-4-іл)хіназоліну-(4H)-онів: 2-(5-аміно-1-(4-хлорфеніл)-1H-[1,2,3]-триазол-4-іл)хіназоліну-(4H)-ону і 2-(5-аміно-1-феніл-1H-[1,2,3]-триазол-4-іл)-6-бромхіназоліну-(4H)-ону на клітини OVCAR-4 раку яєчника з GP = -4,08 та 6,63% відповідно. Виявлено, що 5,7-діаміно-3-(3-(трифторметил)феніл)-3H-[1,2,3]-триазоло[4,5-b]пірідин-6-карбонітрил мав значну активність щодо клітин EKVX раку легенів (GP = -30,74%). Встановлено, що сполуки були менш токсичні порівняно з доксорубіцин щодо непухлинних клітин HEK293 нирки ембріона людини. Такі результати є важливими для отримання більш селективних і активних протипухлинних засобів на основі 5-аміно-1-арил-1H-[1,2,3]-триазолів та їх конденсованих поліциклічних похідних.

Ключові слова: 5-аміно-1-арил-1H-[1,2,3]-триазоли, 3H-[1,2,3]-триазоло[4,5-b]пірідини, хіназоліни, тіазоли, 1,3,4-оксадіазоли, 1,2,3-триазолів та їх метаболіти.

References


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