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Overview of the Cytotoxicity of Hydroxamic Acids, their Derivatives, and their Metal Complexes

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ABSTRACT

Hydroxamic acids (HA) and their metal complexes have attracted considerable attention because of their diverse biological properties, particularly their cytotoxic and potential anticancer activities. This narrative review summarizes the reported cytotoxic and antiproliferative effects of selected HAs, their derivatives, and related metal complexes in cancer and non-cancer cell models. It considers clinically used or clinically investigated hydroxamate-based histone deacetylase inhibitors, including vorinostat, belinostat, panobinostat, pracinostat, abexinostat, dacinostat, quisinostat, and resminostat, as well as deferoxamine, an iron-chelating siderophore containing hydroxamate groups. The reviewed studies indicate that biological activity depends on chemical structure, concentration, exposure conditions, and the experimental model. Reported mechanisms include histone deacetylase inhibition, iron chelation, reactive oxygen species generation, DNA damage, cell-cycle arrest, apoptosis, and, in selected contexts, ferroptosis. Metal coordination may alter the biological activity of hydroxamate ligands; however, cytotoxic potency alone does not establish cancer selectivity or therapeutic efficacy. Interpretation is further complicated by differences in assays and cell models. The review therefore emphasizes the need to distinguish direct cytotoxicity from protective or mechanistic effects, to verify quantitative results against primary studies, and to evaluate toxicity in non-malignant cells.

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INTRODUCTION

Cytotoxicity refers to the ability of a chemical or biological agent to damage cells, inhibit their proliferation, or induce cell death. Depending on the compound and the biological context, cytotoxic effects may involve different cellular processes, including apoptosis, necrosis, and ferroptosis [1, 2]. Although cytotoxicity can be detrimental to normal tissues, the selective induction of cell death in cancer cells represents an important objective in the development of anticancer agents. Consequently, identifying compounds that combine potent antiproliferative activity with acceptable selectivity toward malignant cells remains a major challenge in medicinal chemistry and cancer research [1, 3].

Hydroxamic acids (HA) are characterized by the functional group R–C(=O)–NHOH. Their ability to coordinate metal ions contributes to their chemical and biological properties [2]. Certain hydroxamate derivatives inhibit histone deacetylases (HDAC), enzymes involved in the regulation of gene expression, cellular differentiation, proliferation, and survival. HDAC inhibition can alter the expression of genes associated with cell-cycle regulation and apoptosis and may thereby suppress cancer-cell growth [2, 4]. Not all compounds discussed in this review are themselves hydroxamic acids; related ligands are identified separately where relevant.

Several hydroxamate-based HDAC inhibitors have been investigated for cytotoxic and antiproliferative properties, including vorinostat, belinostat, panobinostat, pracinostat, abexinostat, dacinostat, quisinostat, and resminostat [4–12]. Their effects have been evaluated in different cancer models, including leukemia, neuroblastoma, hepatocellular carcinoma, colorectal cancer, thyroid cancer, and medulloblastoma [5–12]. Deferoxamine is a distinct hydroxamate-containing iron chelator that has been studied in cancer-related experiments [13]. Importantly, the study by Chan et al. [13] examined deferoxamine as an iron-chelating agent that attenuated dihydroartemisinin-induced cytotoxicity in Molt-4 leukemia cells; it did not establish direct cytotoxicity of deferoxamine as a standalone anticancer agent. Other reported effects of hydroxamate-based compounds include oxidative stress, DNA damage, cell-cycle arrest, and programmed cell death, although the contribution of each mechanism varies by compound and experimental model.

Coordination of hydroxamate ligands with metal ions provides another approach to modifying biological activity. Metal complexation can influence molecular structure, stability, electronic properties, and interactions with biological targets [2, 3]. Selected copper, cobalt, iron, nickel, and zinc complexes have been investigated in cytotoxicity or related biological assays [1, 3, 14–16]. Complex formation may alter activity relative to the free ligand, but such changes are compound- and model-dependent. Proposed mechanisms include interactions with DNA or proteins and the modulation of oxidative stress; these mechanisms should be described as experimentally demonstrated only when supported by the relevant primary study [2, 3, 15].

Cytotoxic activity must be considered together with biological selectivity and safety. A compound that inhibits cancer-cell growth may also affect non-malignant cells, potentially limiting its therapeutic value [1, 3]. For example, copper complexes examined by Azeredo *et al*. [1] showed activity in several cancer-cell models, but some also affected peripheral blood mononuclear cells (PBMC). Differences in cell type, exposure time, concentration, and assay method limit direct comparisons across studies. Accordingly, IC_{50} values obtained under different experimental conditions should not be interpreted as directly comparable measures of clinical efficacy.

This review summarizes the reported cytotoxic properties of hydroxamic acids, hydroxamate derivatives, and selected metal complexes. It considers effects on cancer cells and, where reported, non-malignant cells; quantitative endpoints such as IC_{50}; proposed mechanisms; and the influence of metal coordination. Related non-hydroxamate ligands are explicitly distinguished from hydroxamic acids. The review also highlights limitations in the available evidence, including differences among experimental models and the need to verify numerical results against the original studies.

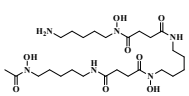
LITERATURE SEARCH AND REVIEW METHODOLOGY

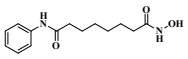
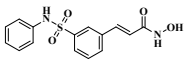
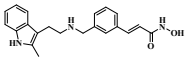
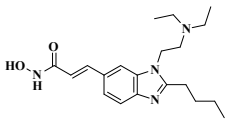
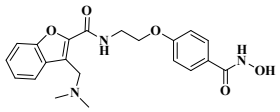
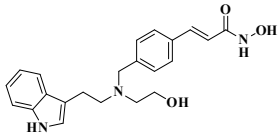
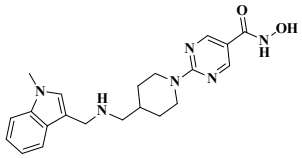
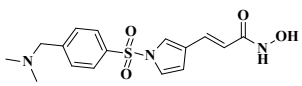
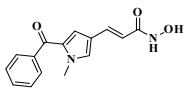
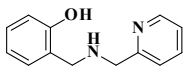
A narrative literature review was conducted to summarize published findings on HA, hydroxamate-based derivatives, and selected metal complexes in cancer-related biological research. Relevant publications were selected on the basis of their relevance to chemical identity, cytotoxicity or antiproliferative activity, experimental cell models, quantitative endpoints (including IC_{50} and percentage inhibition), and proposed mechanisms of action. Primary research articles were prioritized for extraction of experimental results, while review articles were used to provide broader context. Reference lists of relevant publications were also examined to identify additional studies. Because the studies differed in compounds, assays, exposure times, concentrations, and biological models, their results were summarized descriptively and were not pooled quantitatively. This article is a narrative review and was not designed as a systematic review or meta-analysis.

1. Cytotoxicity of HA

Cytotoxicity describes the ability of an agent to impair cell viability or proliferation. Depending on the compound and experimental context, the observed response may involve apoptosis, necrosis, ferroptosis, or reversible/irreversible growth inhibition [1, 2]. This section focuses on selected hydroxamate-based compounds and related agents investigated in preclinical or clinical cancer research. Deferoxamine (**1**) is discussed separately as an iron-chelating hydroxamate-containing siderophore, whereas vorinostat, belinostat, panobinostat, and other hydroxamate-based HDAC inhibitors are considered according to their individual development and approval status. Vorinostat and belinostat, for example, have received regulatory approval for specific hematological malignancies; regulatory status should not be generalized to all compounds in this group.

Table 1. Cytotoxic and antiproliferative effects of selected HA, derivatives, and related ligands (1–11).

Compound no.	Compound / chemical class	Cell model and reported findings	Reference
1	 Deferoxamine	Human T-lymphoblastoid leukemia cells (Molt-4): **1** (10, 20, and 30 μM) was used as an iron-chelating agent and significantly attenuated dihydroartemisinin-induced cytotoxicity. This experiment does not demonstrate direct cytotoxicity of deferoxamine alone.	[13]

Compound no.	Compound / chemical class	Cell model and reported findings	Reference
2	 Vorinostat	Ewing sarcoma cell lines A4573 and TC32: IC_{50} = 200 ng/mL; reported effects included DNA damage, ROS production, oxidative stress, cell-cycle arrest, and apoptosis. Hepatocellular carcinoma/hepatoma cell lines SMMC-7721, BEL7402, and HepG2: IC_{50} = 0.47, 9.49, and 2.39 µM, respectively. HCT-116 colorectal cancer cells: IC_{50} = 2.14 µM.	[5–7]
3	 Belinostat	Thyroid cancer cell lines: IC_{50} values are summarized separately in **Table 2**. Growth inhibition via apoptosis was reported.	[8]
4	 Panobinostat	Thyroid cancer cell lines: IC_{50} values are summarized separately in **Table 2**. The source reports greater cytotoxicity for compound **4** than for compound **3** in the tested thyroid cancer cell lines.	
5	 Pracinostat	HCT-116 colorectal cancer cells: IC_{50} = 0.48 µM. No antiproliferative effect was observed in normal human fibroblasts at concentrations up to 100 µM. In rat renal fibroblasts, no cell death was observed at 500 nM in the reported study.	[7, 9]
6	 Abexinostat	Human neuroblastoma cell lines SKNBE and SMS-KCNR: IC_{50} = 83.5 nM and 196.4 nM, respectively.	[10]
7	 Dacinostat	Medulloblastoma model: IC_{50} < 200 nM in D283 cells. The source reports significant tumor-growth suppression in mice at 20 mg/kg, without weight loss.	[11]
8	 Quisinostat	Medulloblastoma model: IC_{50} < 200 nM in D283 cells. The source reports significant tumor-growth suppression in mice at 20 mg/kg, without weight loss.	
9	 Resminostat	Hepatocellular carcinoma cell lines HepG2, SMMC-7721, and Hep3B: cytotoxic and antiproliferative effects were reported at concentrations ranging from 0.1 to 5 µM.	[12]
10	 (*E*)-3-(5-benzoyl-1-methyl-1*H*-pyrrol-3-yl)-*N*-hydroxyacrylamide	Murine erythroleukemia model: dose-dependent growth inhibition was reported at 4.8 and 24 µM, with increased hemoglobin accumulation at 4.8 µM. The source reports no cytotoxicity after 48 h at 1.2, 2.4, 4.8, and 24 µM.	[17]
11		Colo-205, NCI-H460, U-937-derived cells, and healthy peripheral blood mononuclear cells (PBMC): the related aminophenolic ligand HBPA (not a HA),	[1]

Compound no.	Compound / chemical class	Cell model and reported findings	Reference
	2-[(pyridin-2-ylmethyl)amino]phenol (HBPA; related aminophenolic/pyridyl ligand, not a HA	as well as *N*-hydroxybenzamide (NHBA), was reported to be non-cytotoxic at concentrations up to 100 µM.	

Note. IC_{50}, half-maximal inhibitory concentration; ROS, reactive oxygen species.

Table 2. IC_{50} values (µM) of belinostat (3) and panobinostat (4) in thyroid cancer cell lines.

Thyroid cancer cell line	Belinostat (3)	Panobinostat (4)	Reference
Cal62	0.47	0.033	[8]
Hth7	0.40	0.015	
Hth83	0.72	0.034	
C643	0.97	0.071	
SW1736	0.38	0.035	
T241	0.75	0.065	
T351	1.10	0.050	
BHP2-7	0.77	0.037	
T238	6.80	1.50	

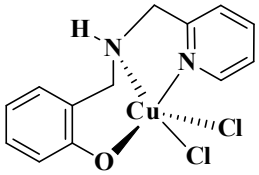
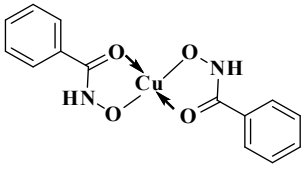
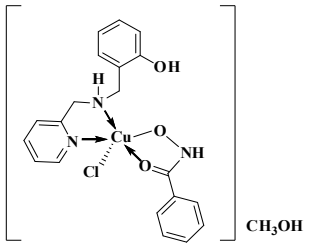
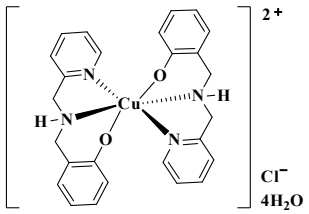
The mechanisms associated with hydroxamate-containing compounds are diverse and should not be conflated. However, several hypotheses have been put forward and experimentally verified. Iron chelation can restrict the availability of iron required for DNA synthesis and cell proliferation, whereas iron-dependent radical formation can contribute to the cytotoxicity of compounds such as dihydroartemisinin. In the study by Chan *et al.* [13], Compound **1** (**Table 1**) attenuated dihydroartemisinin-induced cytotoxicity in Molt-4 cells, supporting an iron-dependent component of dihydroartemisinin action. This experiment does not demonstrate that compound **1** itself was cytotoxic. More generally, iron depletion may affect proliferating cells, but selective toxicity toward cancer cells must be demonstrated experimentally rather than inferred solely from iron requirements.

Iron-mediated free radical formation plays a crucial role in the cytotoxic mechanism (ferroptosis). The same applies to iron depletion via a chelating agent (reduced cell division) [5]. The presence of iron is essential for DNA synthesis during mitosis in rapidly dividing cancer cells. Cancer cells exhibit high expression of transferrin receptors, which enable them to absorb more iron than normal cells. This high level of free iron in cancer cells makes a chelating agent selectively toxic to them [13].

2. Cytotoxicity of HA complexes

Studies of metal complexes containing hydroxamate or related ligands have been investigated for different biological endpoints. Co(II) and Cu(II) benzohydroxamate complexes studied by Coelho *et al.* [18], were evaluated for antimycobacterial activity, including activity against intramacrophage *Mycobacterium tuberculosis*. The authors reported that none of the tested compounds showed a cytotoxic effect at 200 µg/mL. This concentration is a tested concentration at which cytotoxicity was not detected; it is not an IC_{50} value. The study is an antimicrobial investigation and should not be presented as evidence of anticancer activity. It is distinct from the anticancer cytotoxicity results reported by Azeredo *et al*. [1] for copper complexes **12**–**15** (**Table 2**). In that study, complex **15** showed IC_{50} values of 20–27 µM across the tested cancer-cell models and an IC_{50} of 18 µM in PBMC, indicating limited selectivity under the reported conditions (**Table 3**) [1].

Table 3. Cytotoxicity (IC_{50}, µM) of HA-derived metal complexes 12–15 in cancer and normal-cell models.

Complex no.	Chemical structure	Colo-205	NCI-H460	U-937	PBMC	Reference
12		>250	56	>400	45	[1]
13		>100	38	42	82	
14		35	32	31	36	
15		20	25	27	18	

Note. IC_{50}, half-maximal inhibitory concentration; Colo-205: colon cancer cell line; NCI-H460: lung carcinoma cell line; U-937: monocyte cell line derived from a lymphoma; and PBMC: normal peripheral blood mononuclear cells.

Table 4. Reported cytotoxic effects of HA-derived metal complexes 16–18.

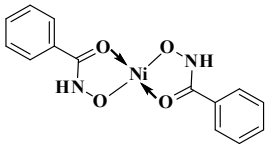
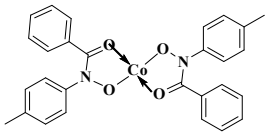
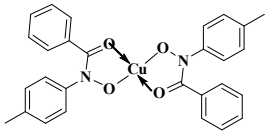
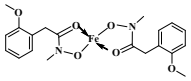
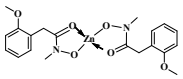
Complex no.	Chemical structure	Cell model and reported findings	Reference
16		Human colorectal carcinoma cells (HCT-116; the source table spells this as "HTC116"): IC_{50} = 60 µM.	[14]
17		Human breast cancer cells (MCF-7); MTT assay, 48 h; concentration range: 0–360 µM. Reported percentage inhibition: 52.80% (the corresponding test concentration is not specified in the source excerpt); IC_{50} = 38.09 µM.	[15]
18		Human breast cancer cells (MCF-7); MTT assay, 48 h; concentration range: 0–360 µM. Reported percentage inhibition: 83.97% (the corresponding test concentration is not specified in the source excerpt); IC_{50} = 89.52 µM.	

Table 5. Toxicity of iron(II) and zinc(II) complexes 19 and 20 in Artemia after 24 h.

Complex no.	Chemical structure	Test concentration	Exposure time	Reported mortality	Reference
19		10 µg/mL	24 h	16.66%	[16]
20		10 µg/mL	24 h	96.66%	

Note. These values represent the reported mortality in Artemia after 24 h; they are not mammalian-cell IC_{50} measurements.

For the Co(II) and Cu(II) benzohydroxamate complexes described by Coelho *et al.* [18], molecular docking suggested a possible interaction with urease, an enzyme relevant to the intraphagosomal survival of *Mycobacterium tuberculosis*. This is a computationally proposed interaction, not direct experimental proof of urease inhibition or an established anticancer mechanism. Separately, Azeredo *et al.* [1] reported findings consistent with ferroptosis-related cell death for complex **15**; these observations should be interpreted within the specific experimental conditions and cell models used in that study.

CONCLUSION

This review summarizes evidence on the cytotoxic and antiproliferative properties of HA, hydroxamate derivatives, and selected metal complexes. Several hydroxamate-based HDAC inhibitors have shown activity in different cancer-cell models. Reported mechanisms include HDAC inhibition, iron chelation, reactive oxygen species generation, DNA damage, cell-cycle arrest, apoptosis, and ferroptosis. The findings also demonstrate why cytotoxic potency must be interpreted alongside effects on non-malignant cells. Deferoxamine requires a distinct interpretation: in the cited Molt-4 study, it was used to attenuate dihydroartemisinin-induced cytotoxicity rather than demonstrated as a directly cytotoxic agent [13]. In addition, the benzohydroxamate complexes studied against intramacrophage Mycobacterium tuberculosis [18] provide antimicrobial, not anticancer, evidence.

Future studies should clarify the mechanisms underlying the biological effects of these compounds, establish structure–activity relationships, and compare cancerous and non-cancerous cells under standardized conditions. Particular attention should be given to the influence of metal identity, ligand structure, stability, and cellular uptake on biological activity. Quantitative results should be reported with the corresponding concentration, exposure time, assay, cell model, and uncertainty where available. Further preclinical investigations are needed before the compounds discussed here can be considered therapeutically useful. Overall, hydroxamic acids and related metal complexes remain valuable research scaffolds, but their selectivity, safety, and therapeutic relevance require further validation.

DECLARATIONS

Conflict of Interest: The author declares no conflict of interest.

Funding: No external funding was received for the preparation of this review.

Author Contributions: Ibrahima Sory Sow conceived the review, conducted the literature analysis, and prepared the manuscript.

Data Availability: No new datasets were generated or analyzed in this review.

Ethical Approval: Not applicable; this review did not involve new experiments on humans or animals.

REFERENCES

1. Azeredo, N.F.B.; Borges, F. V.; Mathias, M.S.; Resende, J.A.L.C.; Franco, R.W.A.; Kanashiro, M.M.; Horn, A.; Fernandes, C. Effect of the hydroxamate group in the antitumoral activity and toxicity toward normal cells of new copper(II) complexes. *BioMetals* 2021, *34*, 229–244, doi:10.1007/s10534-020-00275-9.
2. Kovacic, P.; Edwards, C.L. HAs (therapeutics and mechanism): chemistry, acyl nitroso, nitroxyl, reactive oxygen species, and cell signaling. Journal of Receptor and Signal Transduction Research 2011, 31(1), 10–19. doi:10.3109/10799893.2010.497152.
3. Guk, D.A.; Krasnovskaya, O.O.; Beloglazkina, E.K. Coordination compounds of biogenic metals as cytotoxic agents in cancer therapy. Russian Chemical Reviews 2021, 90(12), 1566–1623. doi:10.1070/RCR5016.
4. Grassadonia, A.; Cioffi, P.; Simiele, F.; Iezzi, L.; Zilli, M.; Natoli, C. Role of Hydroxamate-Based Histone Deacetylase Inhibitors (Hb-HDACIs) in the Treatment of Solid Malignancies. Cancers 2013, 5(3), 919–942. doi:10.3390/cancers5030919.

5. Sampson, V.B.; Vetter, N.S.; Kamara, D.F.; Collier, A.B.; Gresh, R.C.; Kolb, E.A. Vorinostat enhances cytotoxicity of SN-38 and temozolomide in ewing sarcoma cells and activates STAT3/AKT/MAPK pathways. *PLoS One* 2015, *10*, 1–19, doi:10.1371/journal.pone.0142704.
6. Liao, B.; Zhang, Y.; Sun, Q.; Jiang, P. Vorinostat enhances the anticancer effect of oxaliplatin on hepatocellular carcinoma cells. *Cancer Med.* 2018, *7*, 196–207, doi:10.1002/cam4.1278.
7. Ganai, S.A. Histone deacetylase inhibitor pracinostat in doublet therapy: a unique strategy to improve therapeutic efficacy and to tackle herculean cancer chemoresistance. *Pharm. Biol.* 2016, *54*, 1926–1935, doi:10.3109/13880209.2015.1135966.
8. Chan, D.; Zheng, Y.; Tyner, J.W.; Chng, W.J.; Chien, W.W.; Gery, S.; Leong, G.; Braunstein, G.D.; Koeffler, H.P. Belinostat and panobinostat (HDACI): In vitro and in vivo studies in thyroid cancer. *J. Cancer Res. Clin. Oncol.* 2013, *139*, 1507–1514, doi:10.1007/s00432-013-1465-6.
9. Kang, S.W.; Lee, S.M.; Kim, J.Y.; Kim, S.Y.; Kim, Y.H.; Kim, T.H.; Kang, M.S.; Jang, W.H.; Seo, S.K. Therapeutic activity of the histone deacetylase inhibitor SB939 on renal fibrosis. *Int. Immunopharmacol.* 2017, *42*, 25–31, doi:10.1016/j.intimp.2016.11.008.
10. Sholler, G.S.; Currier, E.A.; Dutta, A.; Slavik, M.A.; Illenye, S.A.; Mendonca, M.C.F.; Dragon, J.; Roberts, S.S.; Bond, J.P. PCI-24781 (abexinostat), a novel histone deacetylase inhibitor, induces reactive oxygen species-dependent apoptosis and is synergistic with bortezomib in neuroblastoma. *J. Cancer Ther. Res.* 2013, *2*, 21–41, doi:10.7243/2049-7962-2-21.
11. Zhang, S.; Gong, Z.; Oladimeji, P.O.; Currier, D.G.; Deng, Q.; Liu, M.; Chen, T.; Li, Y. A high-throughput screening identifies histone deacetylase inhibitors as therapeutic agents against medulloblastoma. *Exp. Hematol. Oncol.* 2019, *8*, 30–39, doi:10.1186/s40164-019-0153-x.
12. Fu, M.; Shi, W.; Li, Z.; Liu, H. Activation of mPTP-dependent mitochondrial apoptosis pathway by a novel pan HDAC inhibitor resminostat in hepatocellular carcinoma cells. *Biochem. Biophys. Res. Commun.* 2016, *477*, 527–533, doi:10.1016/j.bbrc.2016.04.147.
13. Chan, H.W.; Singh, N.P.; Lai, H.C. Cytotoxicity of dihydroartemisinin toward Molt-4 cells attenuated by N-tert-butyl-alpha-phenylnitrone and deferoxamine. Anticancer Res. 2013, 33(10), 4389–4393. PMID: 24123007. (No DOI is indexed in PubMed.)
14. Hassan, L.R.; Ramasamy, K.; Lim, S.M.; Bahron, H.; Tajuddin, A.M. Synthesis and characterization of benzoHA metal complexes and their cytotoxicity study. Jurnal Teknologi 2018, 80(6), 87–94. doi:10.11113/jt.v80.12293.
15. Thakur, Y.; Agrawal, R.; Tripathi, M.; Siddiqi, M.K.; Mohapatra, E.; Khan, R.H.; Pande, R. Exploring the DNA binding efficacy of Cobalt(II) and Copper(II) complexes of HAs and explicating their anti-cancer propensity. *J. Mol. Struct.* 2019, *1197*, 691–706, doi:10.1016/j.molstruc.2019.07.099.
16. Khan, S.N.; Ali, I.; Baloch, D.; Shah, W.A.; Ghaffar, A. Synthesis, spectroscopic characterization and antimicrobial activities of N-methyl-o- methoxybenzoHAand its metal (Fe, Co, Ni, Zn) complexes. *Pure Appl. Biol.* 2021, *10*, 225–233, doi:10.19045/bspab.2021.100023.
17. Ragno, R.; Mai, A.; Massa, S.; Cerbara, I.; Valente, S.; Bottoni, P.; Scatena, R.; Jesacher, F.; Loidl, P.; Brosch, G. 3-(4-Aroyl-1-methyl-1H-pyrrol-2-yl)-N-hydroxy-2-propenamides as a New Class of Synthetic Histone Deacetylase Inhibitors. 3. Discovery of Novel Lead Compounds through Structure-Based Drug Design and Docking Studies. *J. Med. Chem.* 2004, *47*, 1351–1359, doi:10.1021/jm031036f.
18. Coelho, T.S.; Halicki, P.C.B.; Silva Jr., L.; de Menezes Vicenti, J.R.; Gonçalves, B.L.; Almeida da Silva, P.E.; Ramos, D.F. Metal-based antimicrobial strategies against intramacrophage Mycobacterium tuberculosis. Lett. Appl. Microbiol. 2020, 71(2), 146–153. doi:10.1111/lam.13298.