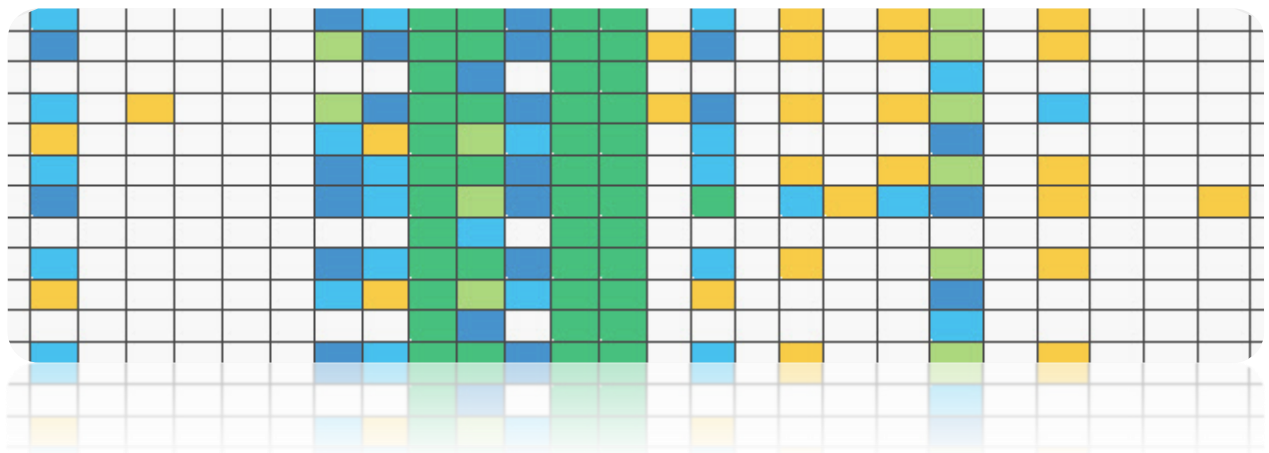


ATLAS

Interpretable prognosis for susceptibility to active anti-cancer molecular forms, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides, of transcriptome cell lines inherent in tumors



VASIL TSANOV & HRISTO TSANOV
Sofia, 2023

ATLAS

Interpretable prognosis for susceptibility to active anti-cancer molecular forms, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides, of transcriptome cell lines inherent in tumors

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Genesis 1:29

“And God said, Behold, I have given you every herb bearing seed, which is upon the face of all the earth, and every tree, in the which is the fruit of a tree yielding seed; to you it shall be for meat”ⁱ



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This book is constructed in a structure aimed at limiting animal testing in laboratory and preclinical settings.



Only refurbished computers, tablets and smart devices were used in the creation of this book. Good practices have also been applied to reduce energy consumption. The goal is to prevent additional carbon load in the world around us.

Full calculations are posted here*:

Tsanov, V., Tsanov, H. (2023). Interpretable prediction for anticancer sensitivity of glycoside amides (Version 1) [Data set], Zenodo, DOI: [10.5281/zenodo.7550371](https://doi.org/10.5281/zenodo.7550371)

*- the information is constantly updated - choose the current version at the moment

ABSTRACT

This ATLAS presents in appended form the complete monographic study (Tsanov & Tsanov, 2023) on the subject. The idea is to bring the results out of the "heavier" didactic format required for the scientific presentation and at the same time to systematize all the information needed for the subsequent clinical research.

The present excerpt from monographic work seeks to introduce the possibility that oncological diseases become chronic (like diabetes). The theoretical basis on which we refer is the fact that cancer cells feed only on carbohydrates. In turn, there is evidence that some nitrile glycosides have anti-cancer properties.

The study is divided into four parts presented in the form of goals.

Proceeding from the particular to the general, the fine molecular structure and all possible biochemical reactions of *Amygdalin* are examined. By itself, it is highly toxic to the physiologically active animal cell. Amygdalin has NO pronounced antitumor properties. In the first goal, he examines precisely the acceptable form for admission. It is concluded that the active anticancer molecular form is a stoichiometric mixture of the amide and carboxyl derivative of the nitrile glycoside.

The second goal is to study the exact action of the anti-tumor product already introduced. After an extremely precise biochemical and mathematical analysis, a series of biochemical cycles of the exact passage of the product through the digestive system, penetration into the blood, approach to cancer cells and selective passage through their cell membrane have been deduced.

The third goal is to define the chemical and pharmaceutical molecular forms. 54 methodological and/or pharmaceutical models with hundreds of variables for each are reviewed and analyzed here.

The fourth goal (it is this purpose that is separately brought out in this edition) presents the interpretable prediction of anticancer susceptibility of glycosidic amides. The affinities of the pharmaceutical form to each known cancer cell line are reviewed. The results surpass many times all anti-cancer drugs and with significantly reduced toxicity.

In an additional part, a generalized clinical control is presented. When conducting the treatment, it is also vitally important to not divert the therapy process to irreversible pathology.

Result: The cancer can become chronic and become a practically curable disease.

ABBREVIATIONS USED IN THE TEXT

AACF Active Anticancer Cell molecules Forms

AAF Active apoptotic forms

ATLAS

Interpretable prognosis for susceptibility to active anti-cancer molecular forms, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides, of transcriptome cell lines inherent in tumors

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INTRODUCTION

Assuming that some Active Anticancer Cell molecules Forms /AACF/ (secreted inside the cancer cell) could be derived from several active pharmacological forms for oral administration (in combination and/or separately) and their already considered druglikeness of the pharmaceutical form (*GPCR ligand, Ion channel modulator, Kinase inhibitor, Nuclear receptor ligand, Protease inhibitor, Enzyme inhibitor*, pharmacological and biological activity of oral active drugs (*Lipinski's Rule, Ghose Filter and CMC-50-Like Rule, Weber Filter, MDDR-Like Rule and BBB Likeness*), *QED (uwQED and wQED)*, non-laboratory and no clinical information on the chemical (*Receptor activity, Mutagenicity, Carcinogenicity, Toxicity*), *Lipophilicity, Water Solubility, Pharmacokinetics, Medical chemistry indicators*, etc.), it is still not possible to get an idea of the influence of the studied molecules on the real cancer lines.

The main research challenge is to create a sufficiently adapted methodological scheme and at the same time to maintain the general conservatism of good oncological medical practices.

The aim of the present study is to consider the possibility of enhancing the accuracy of predicting the efficacy of the studied pharmacological oral forms using models using several different sources of information, but based on empirical studies on cancer cell lines.

- ◇ -

STRUCTURAL METHODOLOGY

Conducting the experiment

The current methodological program relies on a comparative analysis of non-identical variables. In one case it values the IC₅₀, and in the other pharmacokinetic and druglikeness indicators of potential oral dosage forms.

In order to minimize the dualism in the interpretation, conditionally postulate some of the allowable values that would be reflected in the processing of a sample of data from the general population. They are:

a) Work is carried out with a statistical accuracy of 5% (thus aiming to equate the correlation of some of the indicators due to stereoisomerism). In cases where there is a functional dependence of values with different statistical accuracy, the one with the highest deviation is considered final;

b) To check the repeatability of the analysis (for each individual indicator), a minimum of 5 calculations are performed according to absolutely identical methodologies. If necessary, the tests are performed until a mean deviation of not more than $1 \cdot 10^{-2}$ is obtained;

c) Some methodologies also require the definition of physical conditions; therefore, the following are accepted as inputs: temperature (T) = 310K, $\text{pH}_{\text{oral cavity}} > 6.5 \div 7.5$; $\text{pH}_{\text{stomach}} = 0.9 \div 3.1$. Time is not a factor - it is analyzed separately.

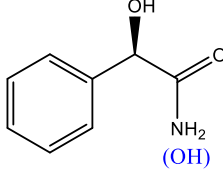
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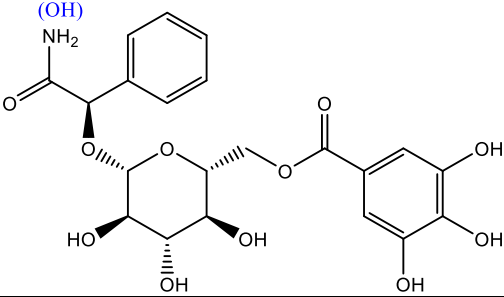
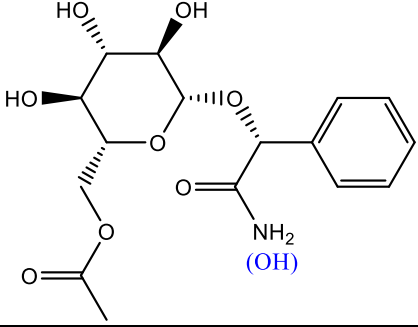
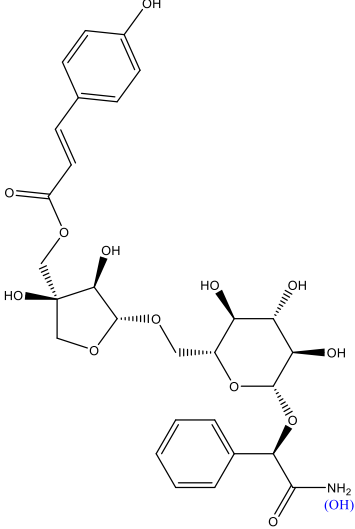
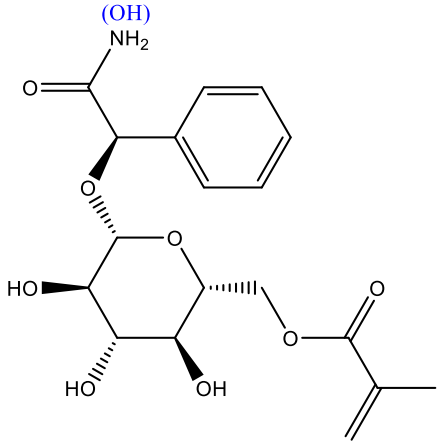
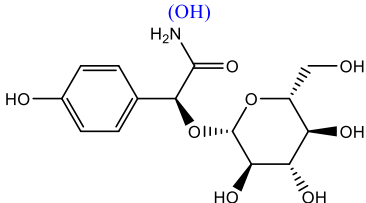
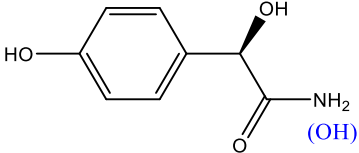
Let us divide the amide/carboxylic derivatives of natural nitrile glycosides conditionally (**Tabl.1**) into 56 groups, coinciding with the active anti-cancer molecular forms (AACF).

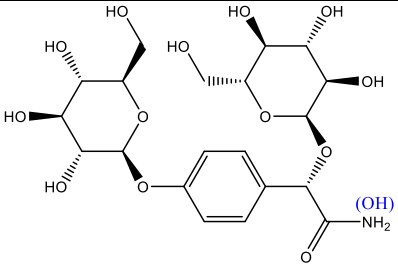
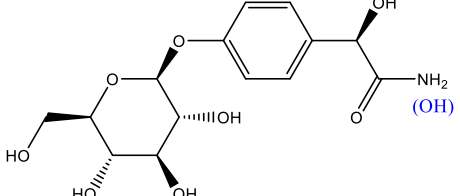
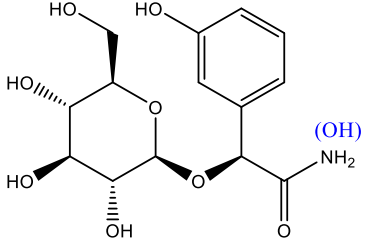
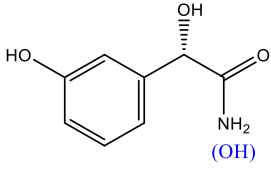
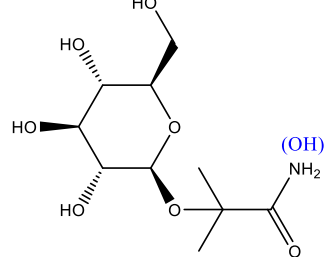
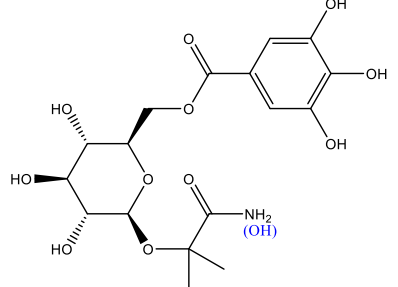
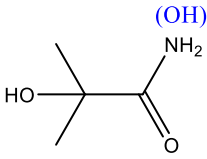
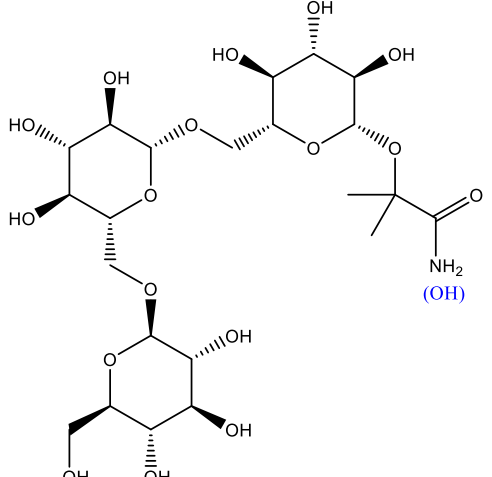
The input data represent the molecular structural properties themselves. With the exception of 9 molecules, the test substances do not have CAS numbers (Dimitrov, et al., 2016) (Yordanova, et al., 2019). In order to achieve maximum repeatability of the results, all analyzes are performed from virtually created images.

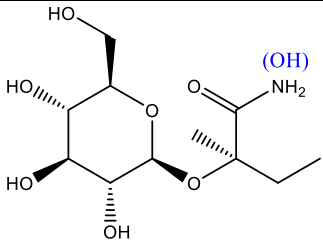
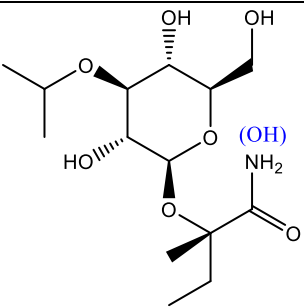
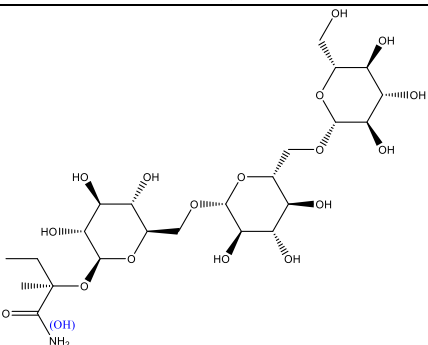
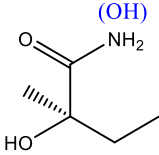
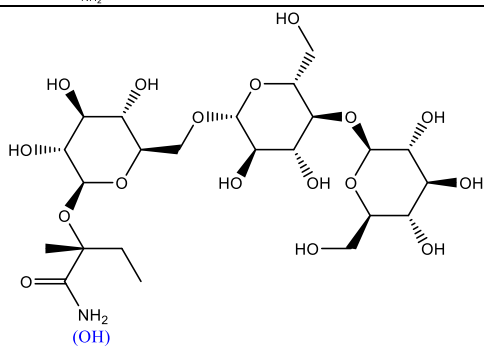
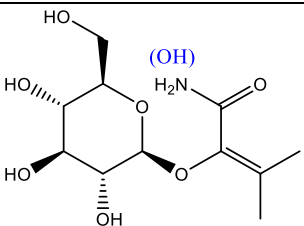
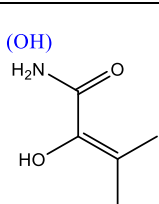
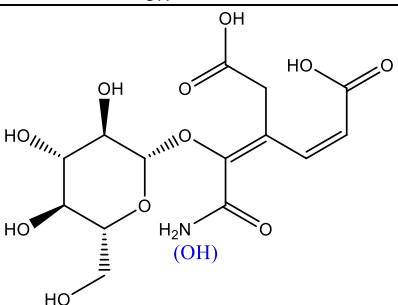
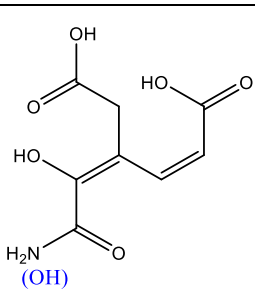
The working file formats (§III.1, Tsanov & Tsanov, 2023) used are *.cdx and *.c3xml. Standard SMILE script generation followed. Each molecular record is of the "canonical SMILES" type. Some of the isomeric forms are excluded here. Therefore, for each molecule is generated, etc. "isomeric SMILES". The data from the latter will be corrective for causes in the interpretation of the results.

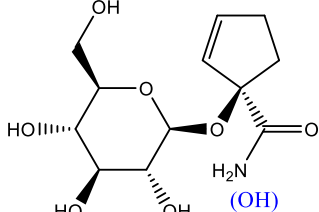
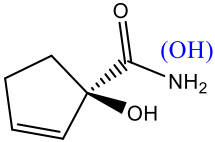
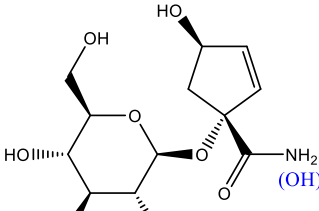
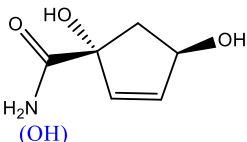
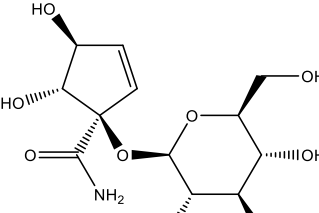
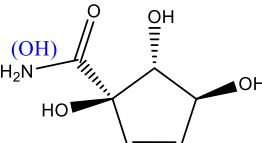
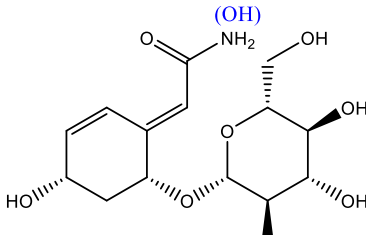
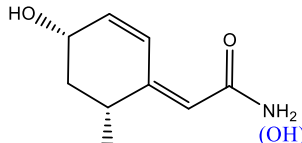
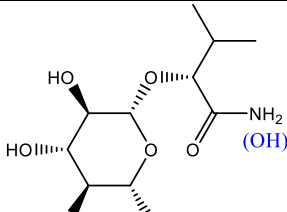
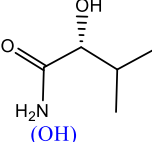
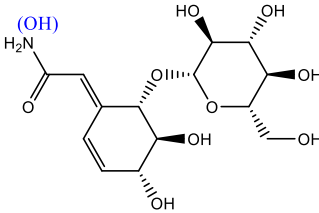
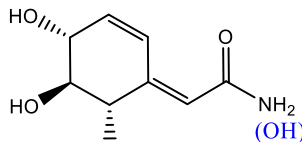
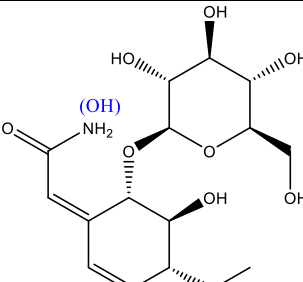
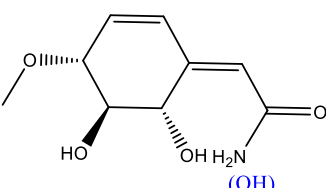
Table 1 Active pharmaceutical forms for oral use and their corresponding active anti-cancer molecules obtained after passage through the cell membrane, based on hydrolyzed amide/carboxylic acid derivatives of natural nitrile glycosides

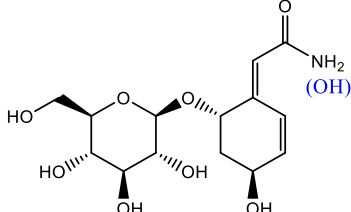
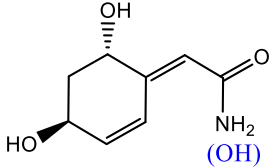
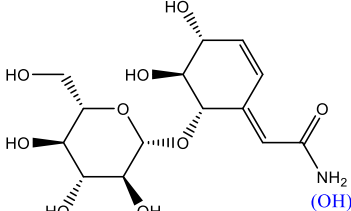
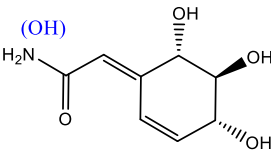
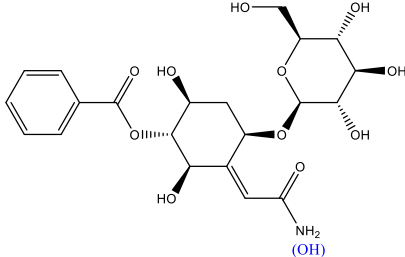
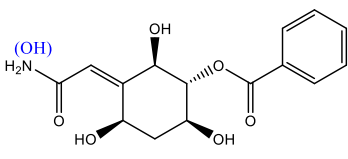
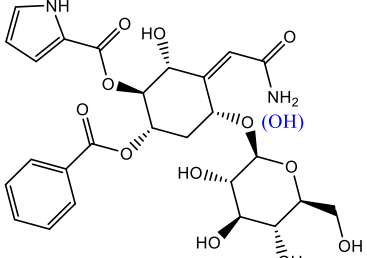
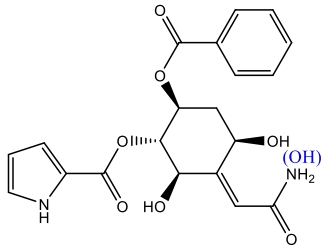
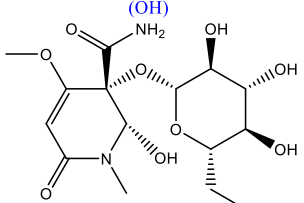
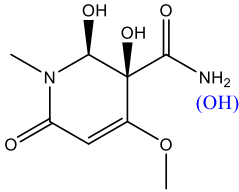
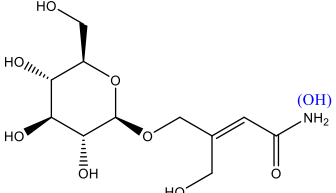
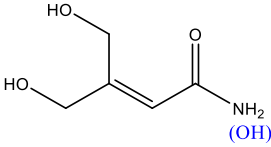
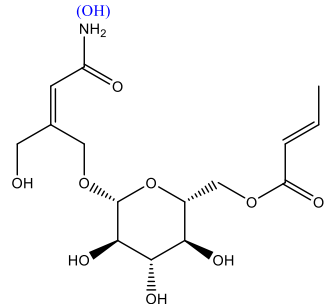
login number	active pharmaceutical forms for oral use	active anti-cancer molecular form obtained after passage through the cell membrane
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	B	
	C	
	D.	

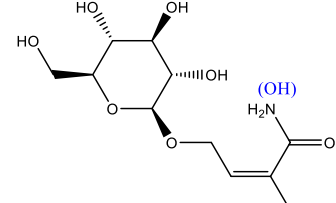
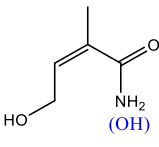
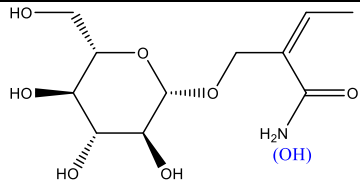
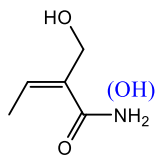
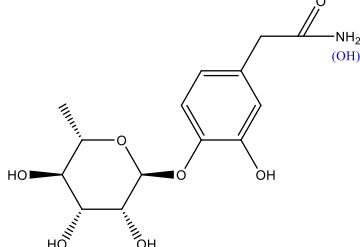
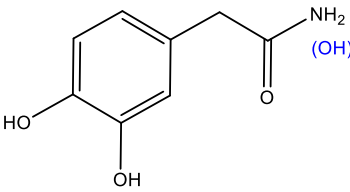
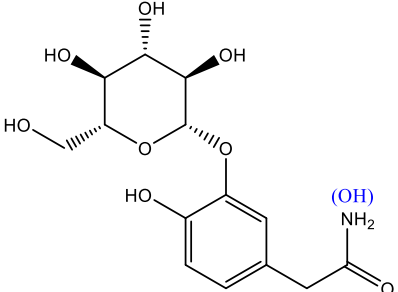
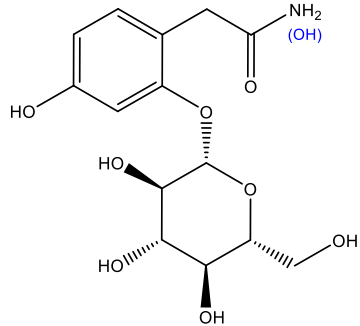
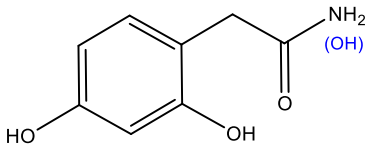
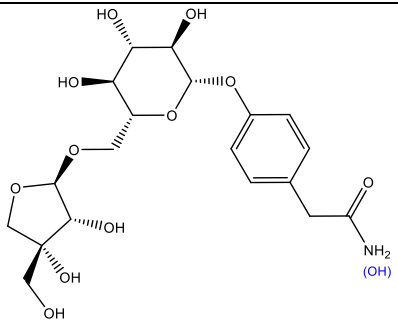
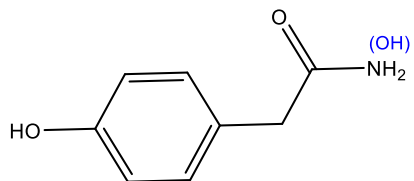
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2. A		

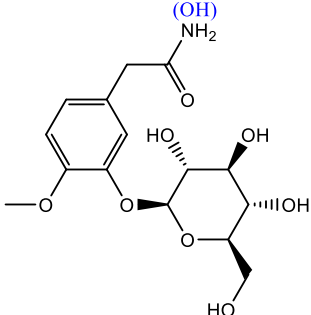
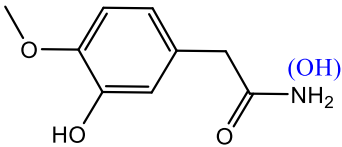
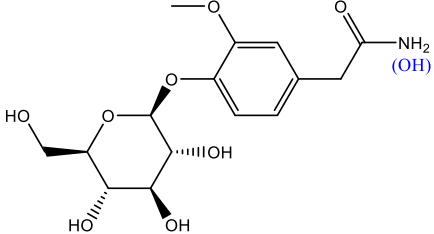
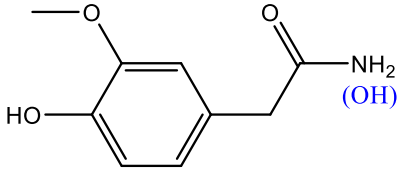
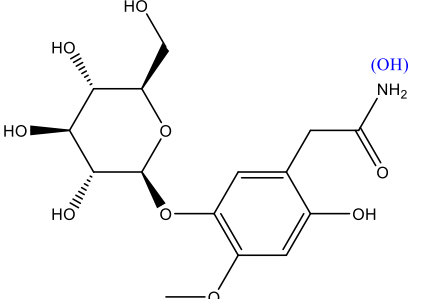
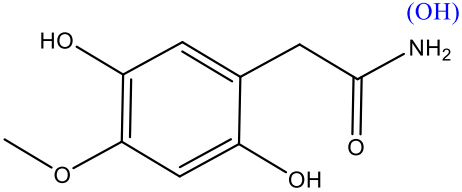
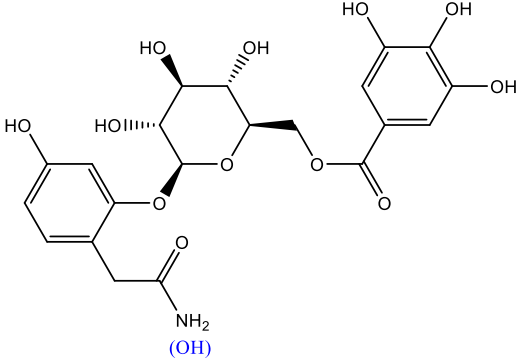
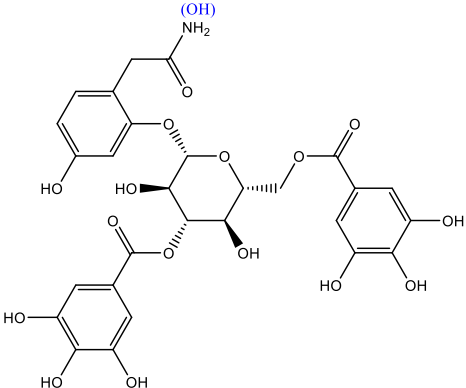
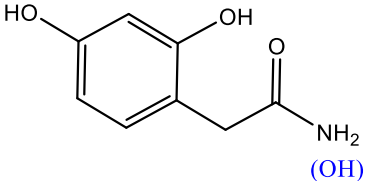
	<p>B</p> 	
	<p>C</p> 	
<p>3.</p>		
<p>4.</p>	<p>A.</p> 	
	<p>B.</p> 	
	<p>C.</p> 	

A.		
B.		
5. C.		
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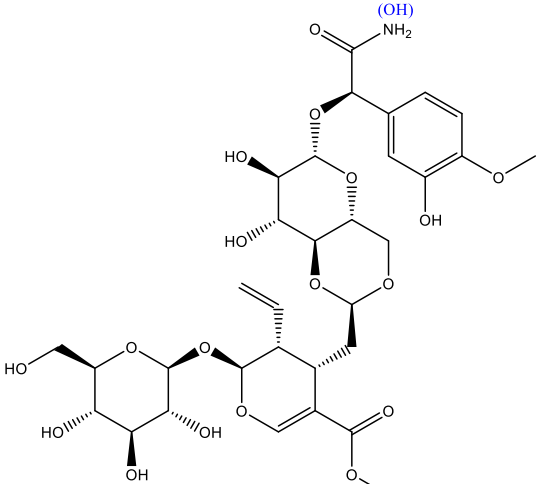
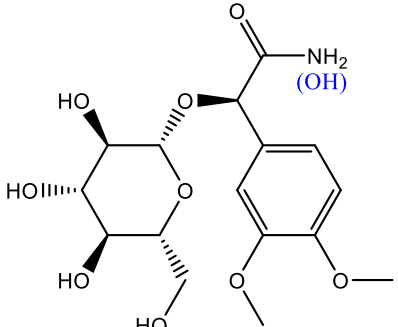
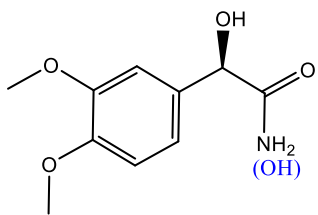
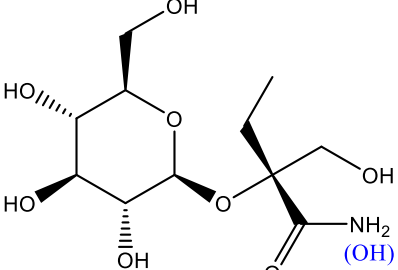
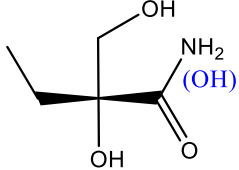
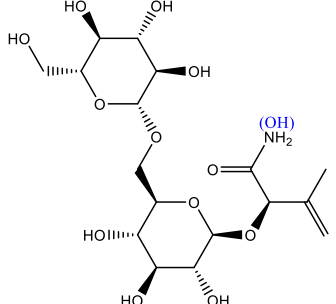
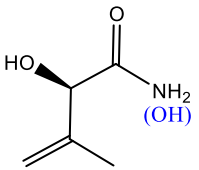
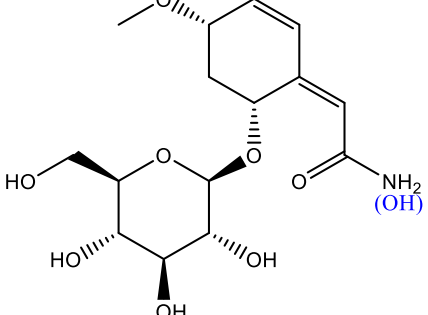
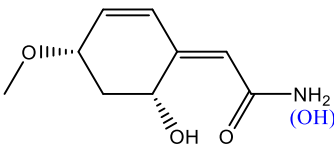
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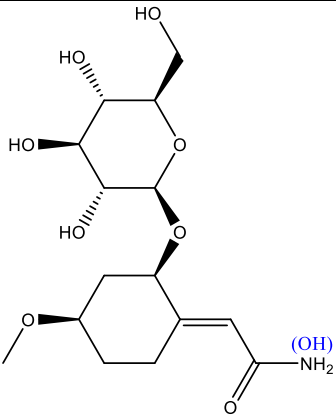
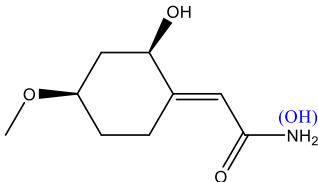
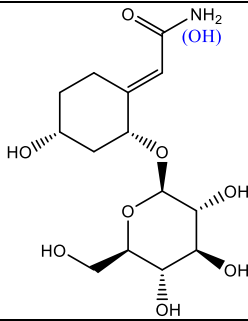
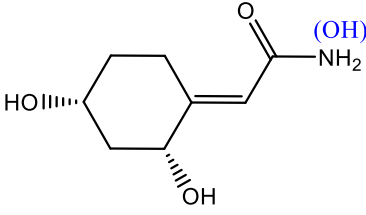
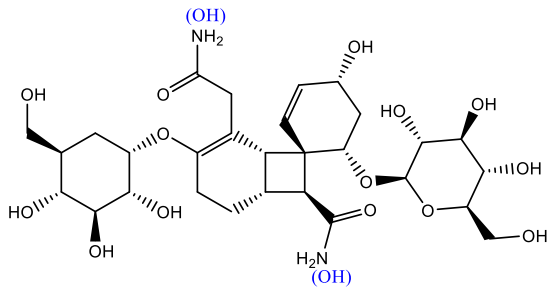
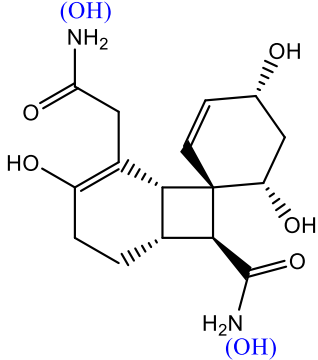
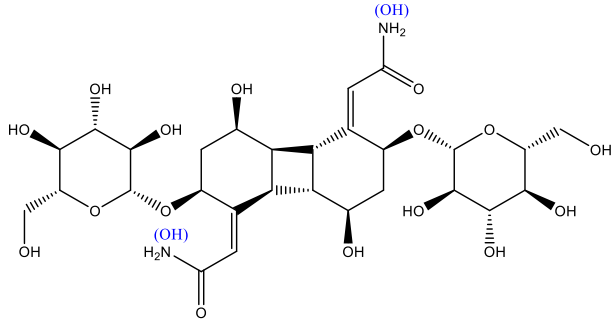
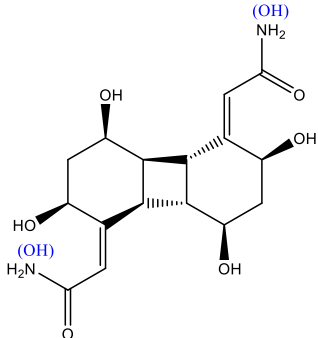
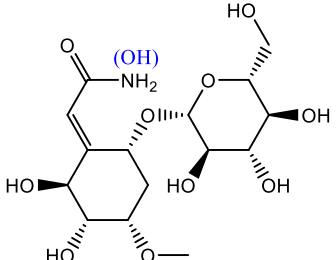
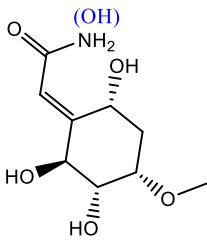
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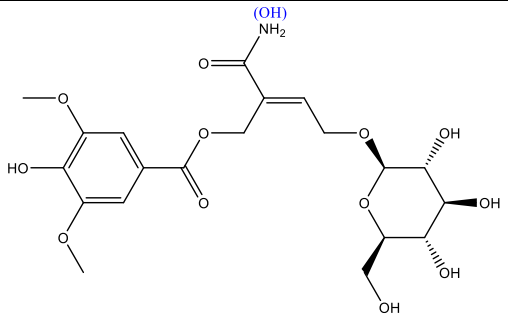
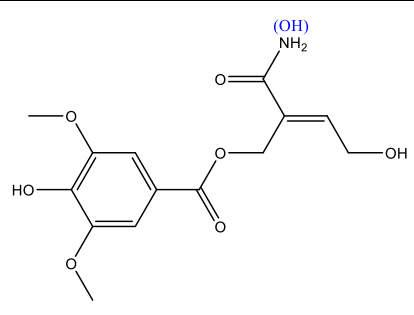
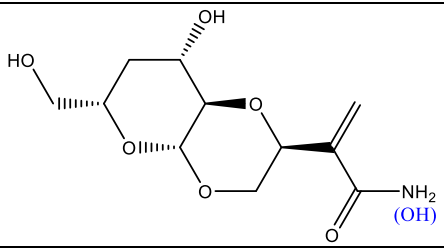
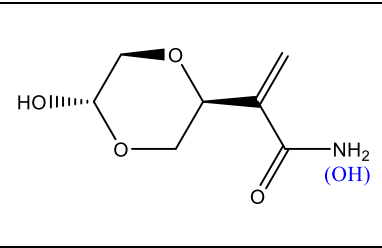
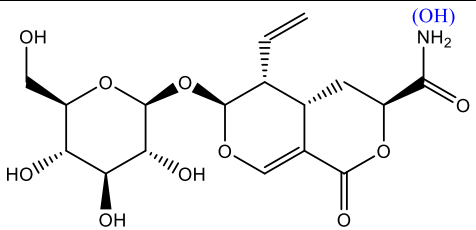
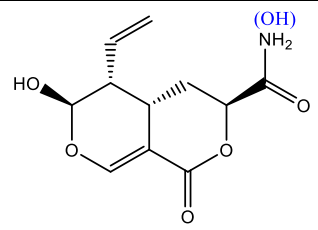
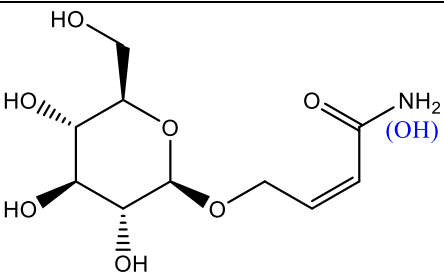
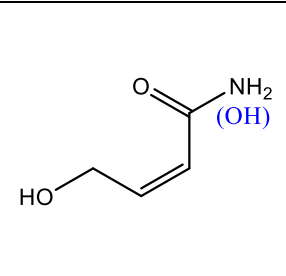
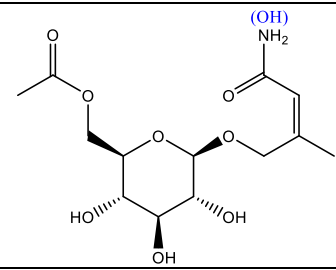
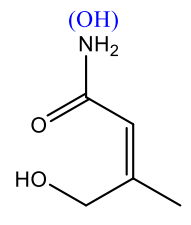
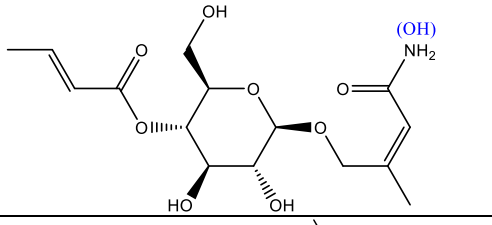
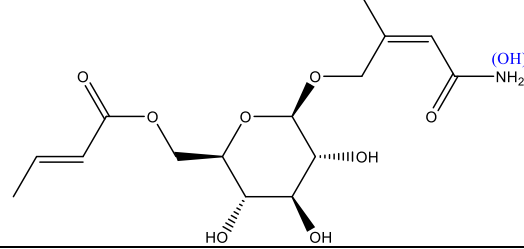
21.		
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23.	<p>B.</p> 	
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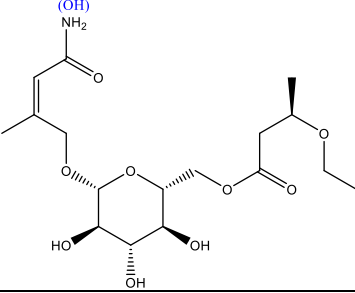
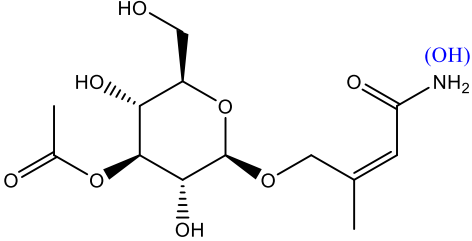
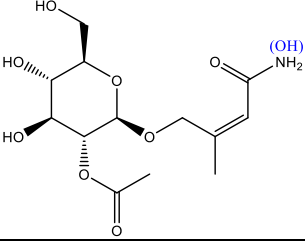
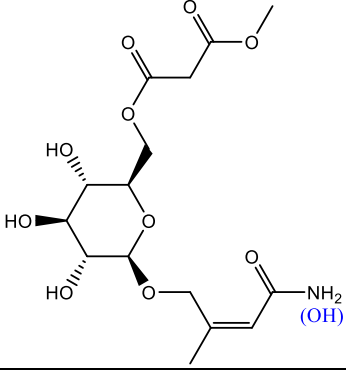
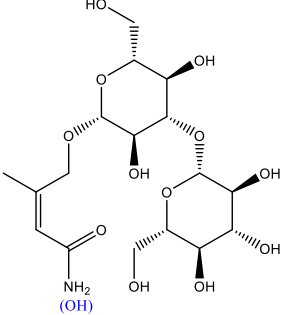
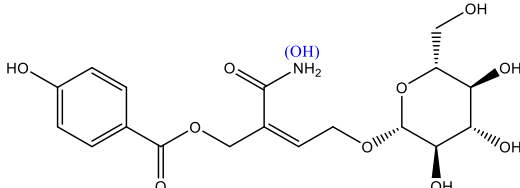
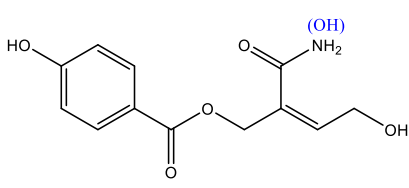
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C.		
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A.		
31.		
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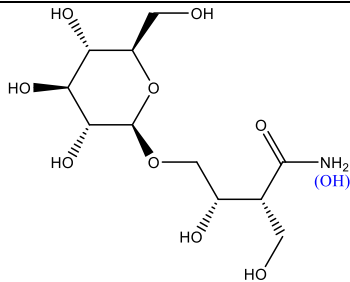
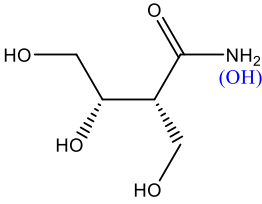
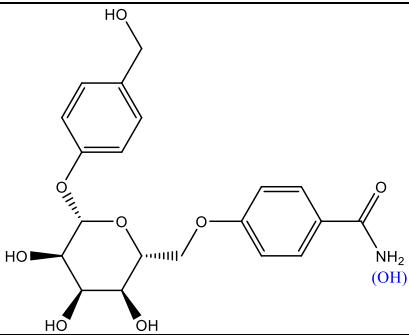
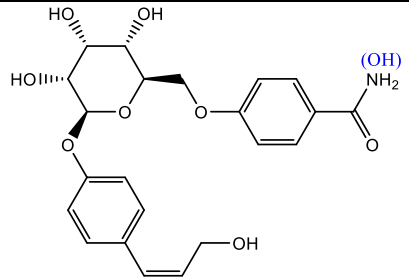
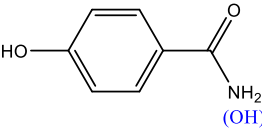
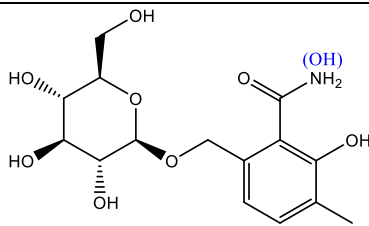
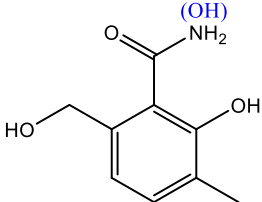
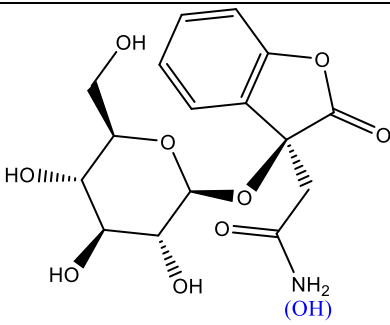
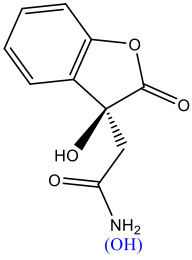
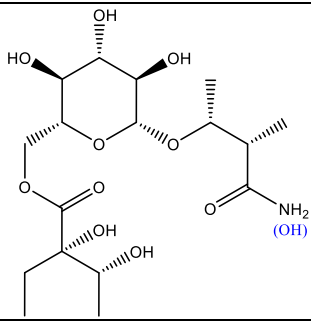
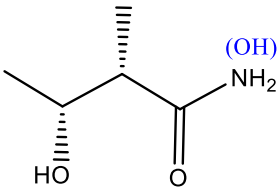
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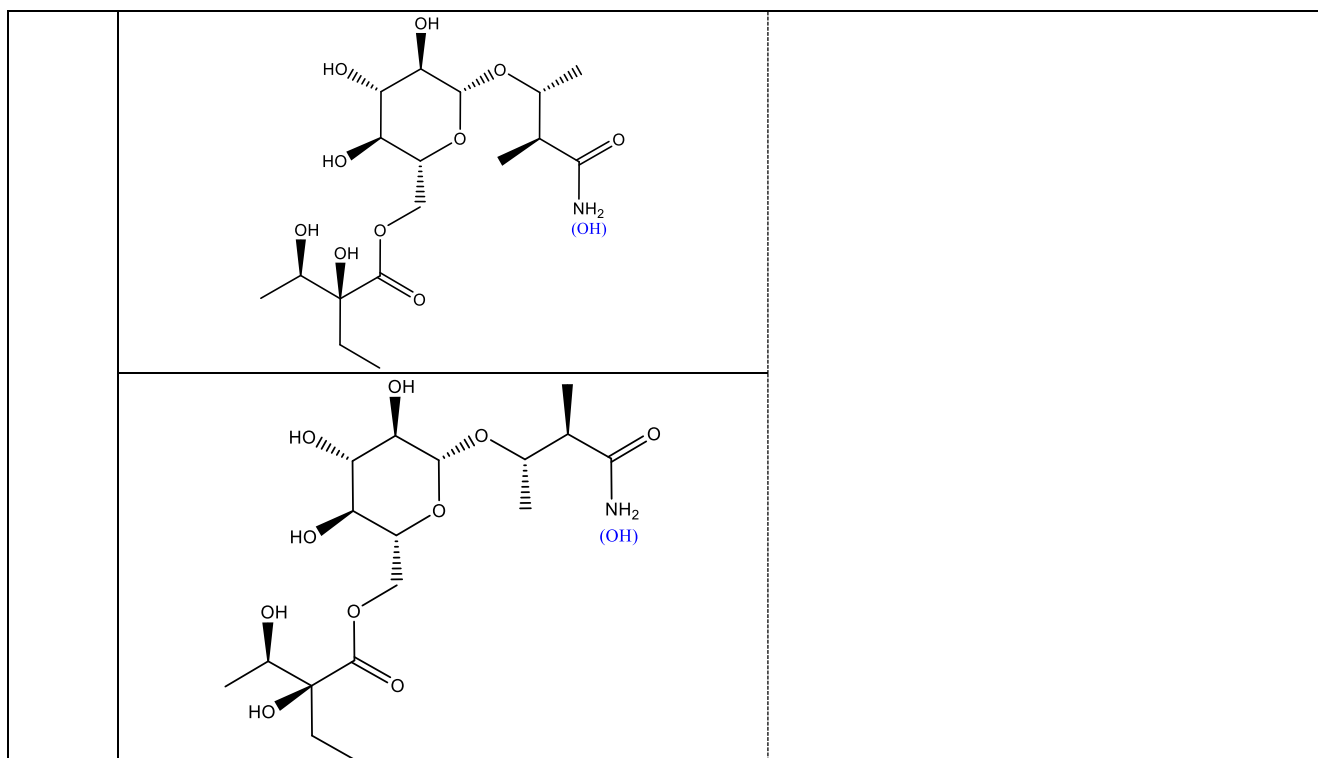
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52.		
53.	<p>A.</p>  <p>B.</p> 	
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Selection of testing algorithms:

This part of the methodology is implemented through the web-based PaccMann™ (Cadow, Born, Manica, Oskooei, & Martínez, 2020) service. The training set uses data from *Genomics of Drug Sensitivity in Cancer* (Soares, et al., 2013) (GDSC) and *Cancer Cell Line Encyclopedia* (Ghandi, Huang, & Jané-Valbuena, 2019) (CCLE). The value reported here is IC₅₀ in $\log(\mu\text{mol})$. The algorithm proposed by the researchers of PaccMann project gives for epistemic and aleatoric confidences (in absolute value) in the range of 0.850÷0.985. This is a good framework for bioactive substances and it is not necessary to anti-logarithm the value, but to analyze it directly within the limits: at IC₅₀ [$\log(\mu\text{mol})$] <1.02 might be accepted that the studied molecule has “medicinal properties”. The correction of 0.2 over the integer 1.0 comes from the statistical error of such calculations. Some of the values are negative numbers, i.e. it could be assumed that the studied molecules will also have restorative clinical properties (Sebaugh, 2011) (Gary, Zhengyin, Wensheng, & Masucci, 2012).

Applicability of results

The structure of the analysis for an interpretable prognosis of susceptibility to active anti-cancer molecular forms of transcriptome cell lines inherent in tumors considers only the described structural relationships between the molecule and the corresponding cellular effect. PaccMann outputs individual topological (and/or structural) fragments as active. The latter, in turn, can be in the amide and carboxylic acid form and in the amide and carboxylic acid active form. On the other hand, only a part of the molecule that is not functionally significant for the process can be described. Last but not least, it should be taken into account that the concentration of the amide pharmaceutical form is 4.87 times higher than the carboxylic form, and even after the passage of the cell membrane of the cancer cell (only the amide form) carboxylic is obtained, which maybe it with anti-cancer activity.

After several mathematical¹ and stoichiometric operations of the case, the values subjected to comparative analysis acquire the following functional dependence:

$$A_{IC50} = 4.87 \times AF_{IC50} + CF_{IC50} + AAF_{IC50} + 0.25 \times ACF_{IC50}$$

where: A_{IC50} is the IC50 activity to be analyzed; AF_{IC50} - value of the IC50 analysis for the amide pharmacological form; CF_{IC50} - value of the carboxylic pharmacological form; AAF_{IC50} - value from the IC50 analysis for the active amide form and ACF_{IC50} - value from the IC50 analysis for the active carboxylic form.

Applying the permissible values (§III.3.4, Tsanov & Tsanov, 2023) it follows that the maximum value for IC50 must be less than 7.35 (with statistical accuracy included).

Presentation of results

All final values are presented in tabular form, using a “heat map” - without placing the numerical values themselves. The color identification is as follows: *dark green* (#00B050) - negative values /best effect/, *light green* (#92D050) - values from 0 to 2, *dark blue* (#0070C0) - 2.01 ÷ 4.00, *light blue* (#00B0F0) - 4.01 ÷ 6.00 and *orange* (#FFC000) - 6.01 ÷ 7.35. Unstained cells do not necessarily mean that there is no activity. The values of some of them could be in a similar order to those accepted in the analysis. However, further research and individual assessments of each individual variable in the comparison process are needed.

The two different input information flows (§III.3.4., Tsanov & Tsanov, 2023) and their differences in the analysis of data collection and packaging, determines the fact that some pairs of cell lines will get different results. That is why both possibilities are presented. They are interpreted together.

- ◇ -

RESULTS AND INTERPRETATION

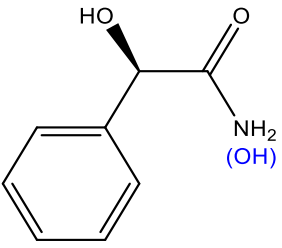
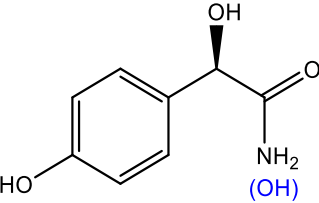
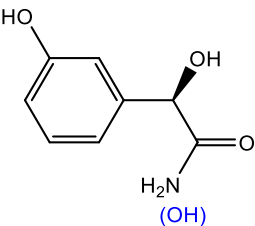
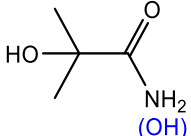
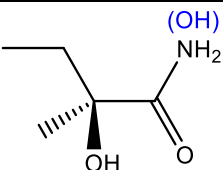
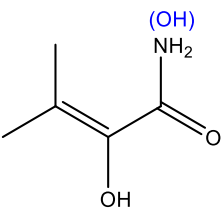
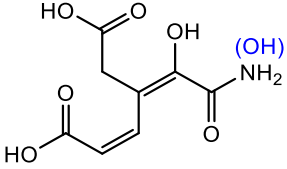
Toxication of the cancer cell

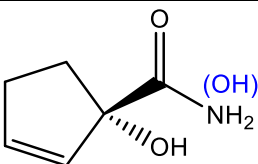
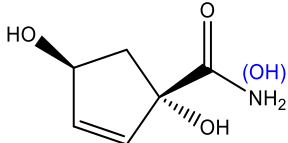
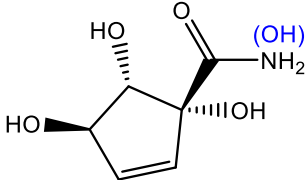
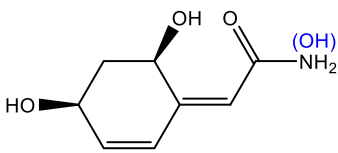
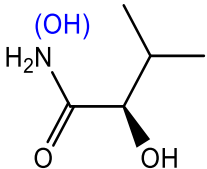
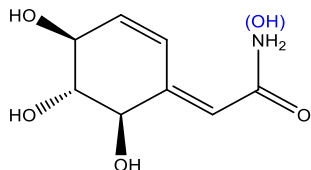
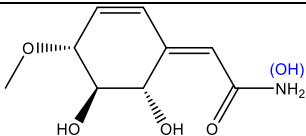
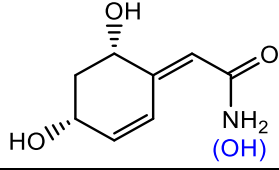
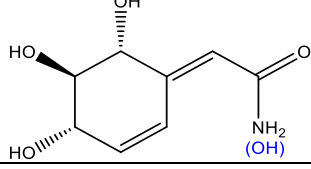
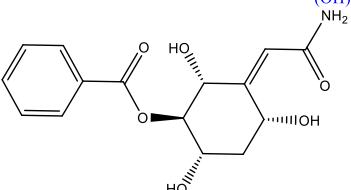
Active apoptotic form (AAF) with manifested anticancer activity is formed according to their molecular structure. For diglycoside compounds (*Amygdalin / Gentiobiose / Lucumin / Primeverose / Vicianin / Vicianose*, etc.) primary enzymatic hydrolysis (*glucanases* - which are abundant in tissue fluids) of the glycosidic bonds between the individual sugars takes place. The relationship between the secondary carbohydrate and the reaction-determining group is stronger and requires a longer reaction

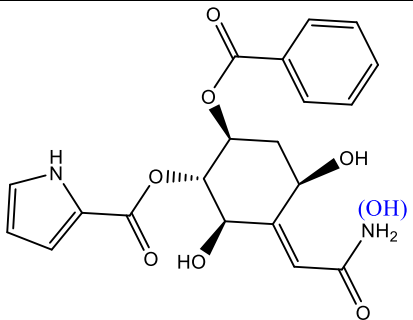
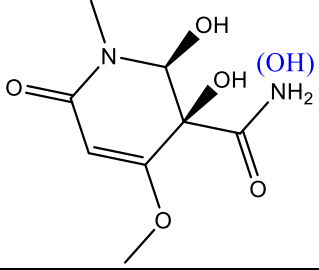
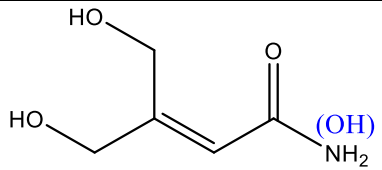
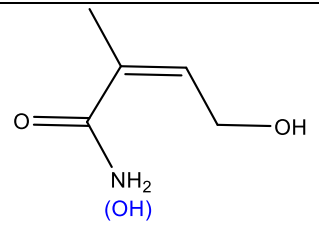
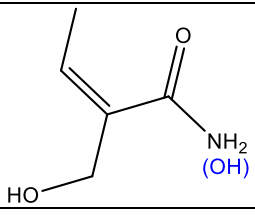
¹ No logarithmic conversion is performed, only the baseline numerical values of the IC50 analysis.

time and/or a specific enzyme such as amygdalin beta-gluconase. The latter is synthesized mainly inside the cell itself. This leads us to conclude that the passage through the cell membrane of the cancer cell (§IV.2.1-2, Tsanov & Tsanov, 2023) occurs with only one carbohydrate molecule. Once inside the cell, the only glycosidic bond is broken. This is how the AAFs themselves are created. Some of them are listed in **Tabl. 2**.

Table 2 Active apoptotic amide/carboxylic acid molecular forms, based on hydrolyzed amide/carboxylic acid derivatives of natural nitrile glycosides

chemical formula	name	natural precursor
	(R)-2-hydroxy-2-phenylacetamide	Prunasin Amygdalin Lucumin Vicianin
	(R)-2-hydroxy-2-phenylacetic acid	Sambunigrin
	(R)-2-hydroxy-2-(4-hydroxyphenyl)acetamide	Dhurrin Taxiphylllein Proteacin
	(R)-2-hydroxy-2-(4-hydroxyphenyl)acetic acid	p-Glucosyloxymendelonitrile
	(R)-2-hydroxy-2-(3-hydroxyphenyl)acetamide	Zierin
	(R)-2-hydroxy-2-(3-hydroxyphenyl)acetic acid	
	2-hydroxy-2-methylpropanamide	Linamarin
	2-hydroxy-2-methylpropanoic acid	
	(S)-2-hydroxy-2-methylbutanamide	Lotaustralin
	(S)-2-hydroxy-2-methylbutanoic acid	
	2-hydroxy-3-methylbut-2-enamide	Acacipetalin
	2-hydroxy-3-methylbut-2-enoic acid	
	(2Z,4E)-4-(2-amino-1-hydroxy-2-oxoethylidene)hex-2-enedioic acid	Triglochinin
	(2E,4Z)-3-(carboxymethyl)-2-hydroxyhexa-2,4-dienedioic acid	

	(S)-1-hydroxycyclopent-2-ene-1-carboxamide	Deidaclin Tetraphyllin A
	(S)-1-hydroxycyclopent-2-ene-1-carboxylic acid	
	(1S,4S)-1,4-dihydroxycyclopent-2-ene-1-carboxamide	Tetraphyllin B Volkenin Taraktophyllin
	(1S,4S)-1,4-dihydroxycyclopent-2-ene-1-carboxylic acid	
	(1R,4R)-1,4,5-trihydroxycyclopent-2-ene-1-carboxamide	Gynocardin
	(1R,4R)-1,4,5-trihydroxycyclopent-2-ene-1-carboxylic acid	
	(Z)-2-((4S,6R)-4,6-dihydroxycyclohex-2-en-1-ylidene)acetamide	Menisdaurin
	(Z)-2-((4S,6R)-4,6-dihydroxycyclohex-2-en-1-ylidene)acetic acid	
	(R)-2-hydroxy-3-methylbutanamide	Volkenin
	(R)-2-hydroxy-3-methylbutanoic acid	
	(E)-2-((4S,5R,6R)-4,5,6-trihydroxycyclohex-2-en-1-ylidene)acetamide	Griffonin
	(E)-2-((4S,5R,6R)-4,5,6-trihydroxycyclohex-2-en-1-ylidene)acetic acid	
	(Z)-2-((4R,5R,6S)-5,6-dihydroxy-4-methoxycyclohex-2-en-1-ylidene)acetamide	Bauhinin
	(Z)-2-((4R,5R,6S)-5,6-dihydroxy-4-methoxycyclohex-2-en-1-ylidene)acetic acid	
	(E)-2-((4R,6S)-4,6-dihydroxycyclohex-2-en-1-ylidene)acetamide	Purshianin
	(E)-2-((4R,6S)-4,6-dihydroxycyclohex-2-en-1-ylidene)acetic acid	
	(E)-2-((4S,5R,6R)-4,5,6-trihydroxycyclohex-2-en-1-ylidene)acetamide	Lithospermoside
	(E)-2-((4S,5R,6R)-4,5,6-trihydroxycyclohex-2-en-1-ylidene)acetic acid	
	(1R,2R,4R,6S,E)-3-(2-amino-2-oxoethylidene)-2,4,6-trihydroxycyclohexyl benzoate	Campyloside A
	(E)-2-((2R,3R,4S,6R)-3-(benzoyloxy)-2,4,6-trihydroxycyclohexylidene)acetic acid	

	(1S,2R,4R,6S,E)-3-(2-amino-2-oxoethylidene)-6-(benzoyloxy)-2,4-dihydroxycyclohexyl 1H-pyrrole-2-carboxylate	Campyloside B
	(E)-2-((2R,3S,4S,6R)-3-((1H-pyrrole-2-carbonyl)oxy)-4-(benzoyloxy)-2,6-dihydroxycyclohexylidene)acetic acid	
	(2S,3S)-2,3-dihydroxy-4-methoxy-1-methyl-6-oxo-1,2,3,6-tetrahydropyridine-3-carboxamide	Acalyphin
	(2S,3S)-2,3-dihydroxy-4-methoxy-1-methyl-6-oxo-1,2,3,6-tetrahydropyridine-3-carboxylic acid	
	4-hydroxy-3-(hydroxymethyl)but-2-enamide	Sutherlandin
	4-hydroxy-3-(hydroxymethyl)but-2-enoic acid	
	(Z)-4-hydroxy-2-methylbut-2-enamide	Rhodiocyanoside A
	(Z)-4-hydroxy-2-methylbut-2-enoic acid	
	(Z)-2-(hydroxymethyl)but-2-enamide	Rhodiocyanoside D
	(Z)-2-(hydroxymethyl)but-2-enoic acid	

Each of these molecules alone would not cross the cell membrane of the cancer cell. Only those related to carbohydrate and fulfilling the conditions of (§IV.2.1-2, Tsanov & Tsanov, 2023) will block and/or permanently damage her normal physiology. The use of **AAF** (**Tabl. 2**) directly for treatment will lead to severe toxic and allergic responses of the body.

By themselves, these compounds or their homologues are still used in conservative chemotherapy (Chabner & Longo, 2018) (Airley, 2009) (Priestman, 2012). Glycosides such as *Rehmapicroside*, *Loganic acid*, *HMBOA D-glucoside*, *Glucose beta-1,3-isofagamine*, *Vanillyl beta-D-glucopyranoside* and others. Although they contain **AAF** of the proposed type, they would not cross the cell membrane of the cancer cell. They do not fulfill the condition of (§IV.2.2., Tsanov & Tsanov, 2023), in the part of the amide derivative which is to be hydrolyzed by a transitional complex with a carboxylic acid.

The relative inertness of the glycosidic bond (*in vivo*) also allows the use of different amide-carboxylic glycosides simultaneously. This is also observed in nature with regard to the distribution of nitrile glycosides - they are often more than one representative in one plant. Thus, different AAFs can be injected simultaneously, at different concentrations and at different times, in order to closely

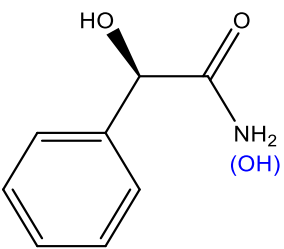
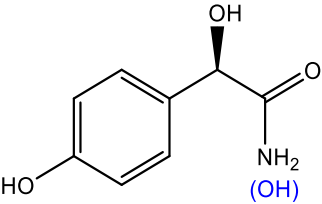
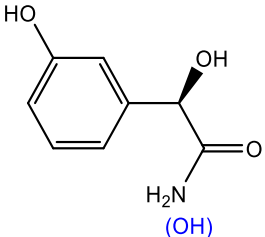
differentiate the different types of cancers, through the synergistic action of the controlled toxicity itself inside the "attacked" cell.

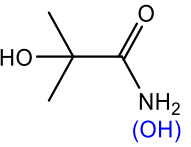
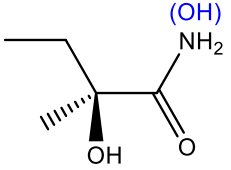
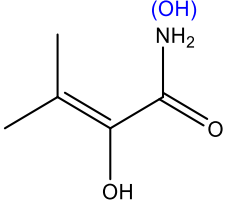
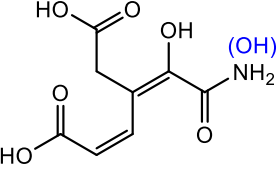
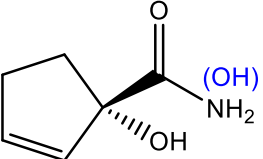
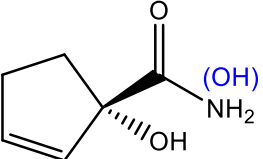
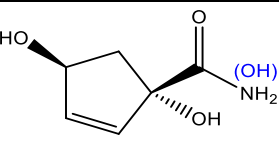
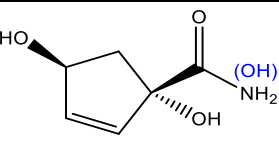
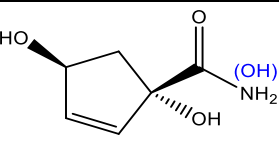
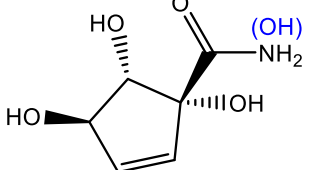
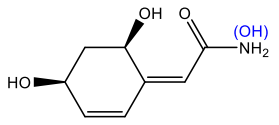
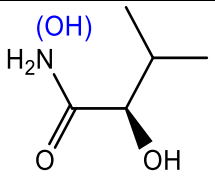
Natural nitrile glycosides would not cross the cancer cell membrane. They decompose to HCN-acid, phenyl methanol and carbohydrate. They do NOT have anticancer activity due to their inability to reach the target unchanged. These compounds, in their natural form, are extremely toxic to the human body. Their application is not a treatment, even in a higher concentration they cause irreversible pathology over the physiologically active animal cell. Dozens of their modified forms have been theoretically derived, but their amides and their carboxylic acids are the most promising for their introduction into conservative oncology. The fact is that the cancer cell itself tries to counteract it in a fairly certain way.

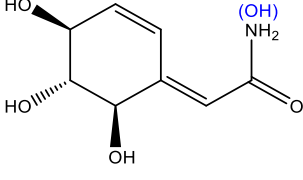
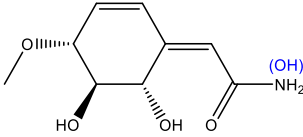
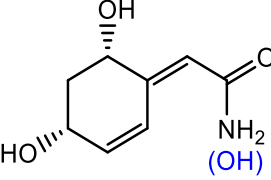
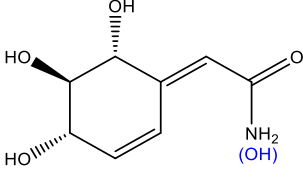
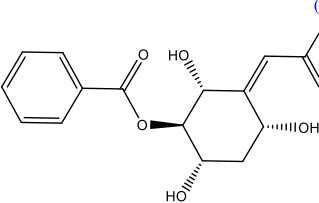
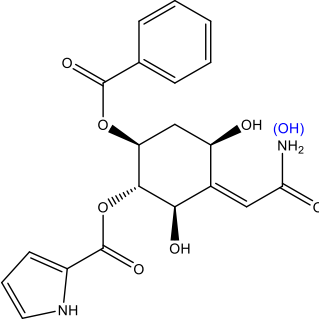
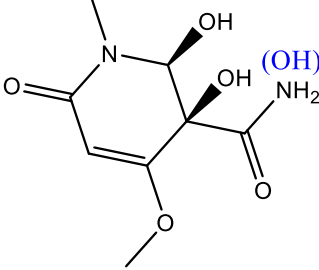
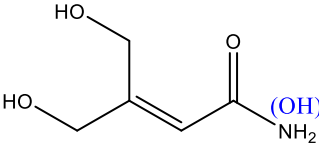
Determination of the drug dose

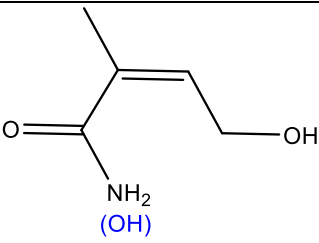
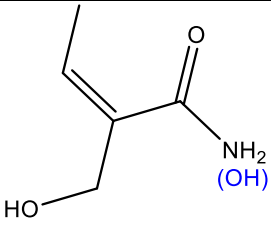
The drug dose is determined by considering all possible substances obtained by the final hydrolysis of the glycosidic bond inside the cancer cell (**Tabl. 2 & 3**).

Table 3 Nature and concentration of active anticancer cell molecules obtained after crossing the cell membrane by their natural precursors

AACF chemical formula obtained after crossing the cell membrane	natural precursor enzymatically modified to amide and carboxylic acid	AACF concentration derived from 1 mg/ml pharmacological form [mg/ml]
	Prunasin 4.87:1	0.40
	Amygdalin 4.87:1	0.27
	Lucumin 4.87:1	0.27
	Vicianin 4.87:1	0.27
	Sambunigrin 4.87:1	0.40
	Dhurrin 4.87:1	0.42
	Taxiphyllin 4.87:1	0.42
	Proteacin 4.87:1	0.31
	p-Glucosyloxymandelonitrile 4.87:1	0.42
	Zierin 4.87:1	0.42

	Linamarin 4.87:1	0.32
	Lotaustralin 4.87:1	0.35
	Acacipetalin 4.87:1	0.34
	Triglochinin 4.87:1	0.47
	Deidaclin 4.87:1	0.36
	Tetraphyllin A 4.87:1	0.36
	Tetraphyllin B 4.87:1	0.39
	Volkenin 4.87:1	0.39
	Taraktophyllin 4.87:1	0.39
	Gynocardin 4.87:1	0.41
	Menisdaurin 4.87:1	0.42
	Epiheterodendrin 4.87:1	0.35

	Griffonin 4.87:1	0.44
	Bauhinin 4.87:1	0.46
	Purshianin 4.87:1	0.42
	Lithospermoside 4.87:1	0.44
	Campyloside A 4.87:1	0.64
	Campyloside B 4.87:1	0.69
	Acalyphin 4.87:1	0.56
	Sutherlandin 4.87:1	0.44

	Rhodiocyanoside A 4.87:1	0.41
	Rhodiocyanoside D 4.87:1	0.41

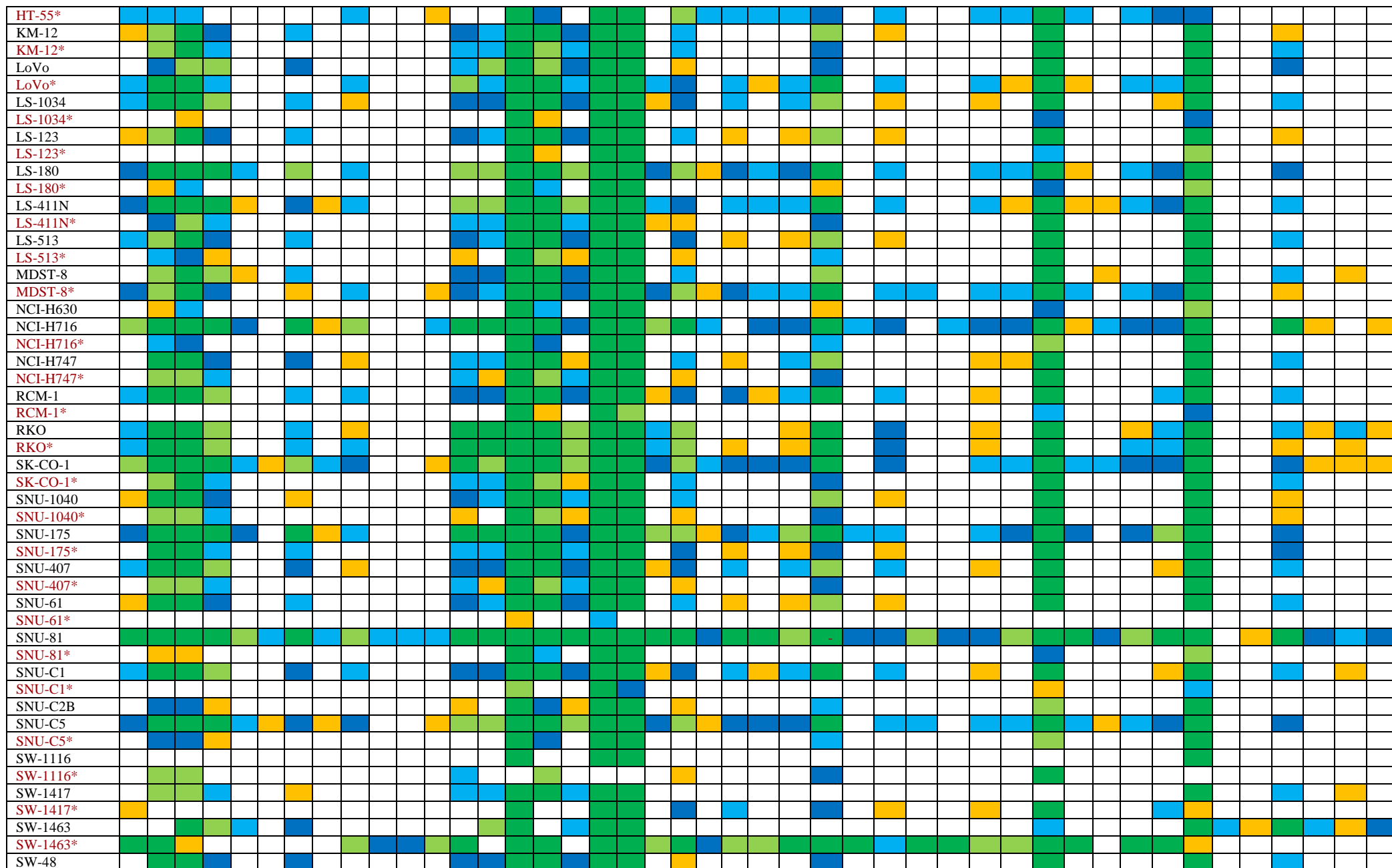
The use of two or more pharmaceutical forms would not prevent their penetration subject to the mass ratios between the active antitumor amide and the active carboxylic transfer form.

The chemical compounds listed in *Tabl. 2 & 3* and are currently used as: anti-migrane, anti-atherosclerotic, anticoagulant, treatment of HIV, anti-cancer, anti-asthmatic, anti-hypertensive, anti-epileptic, analgesic, ocular anti-inflammatory, anti-hypertensive, hypnotic, anesthetic, anti-allergic, aromatase inhibitor, anti-ulcerative, anti-neoplastic, antibacterial, anticoccidial, contraceptive, tyrosine-kinase inhibitor treatment of mast cell tumors, etc.. The difference is that with the proposed technology they are formed inside the cell itself and thus minimize their overall toxicity in the body.

- ◇ -

FINAL REZULTS

The analysis and subsequent presentation of the results was conducted according to §III.3.4, Tsanov & Tsanov, 2023:



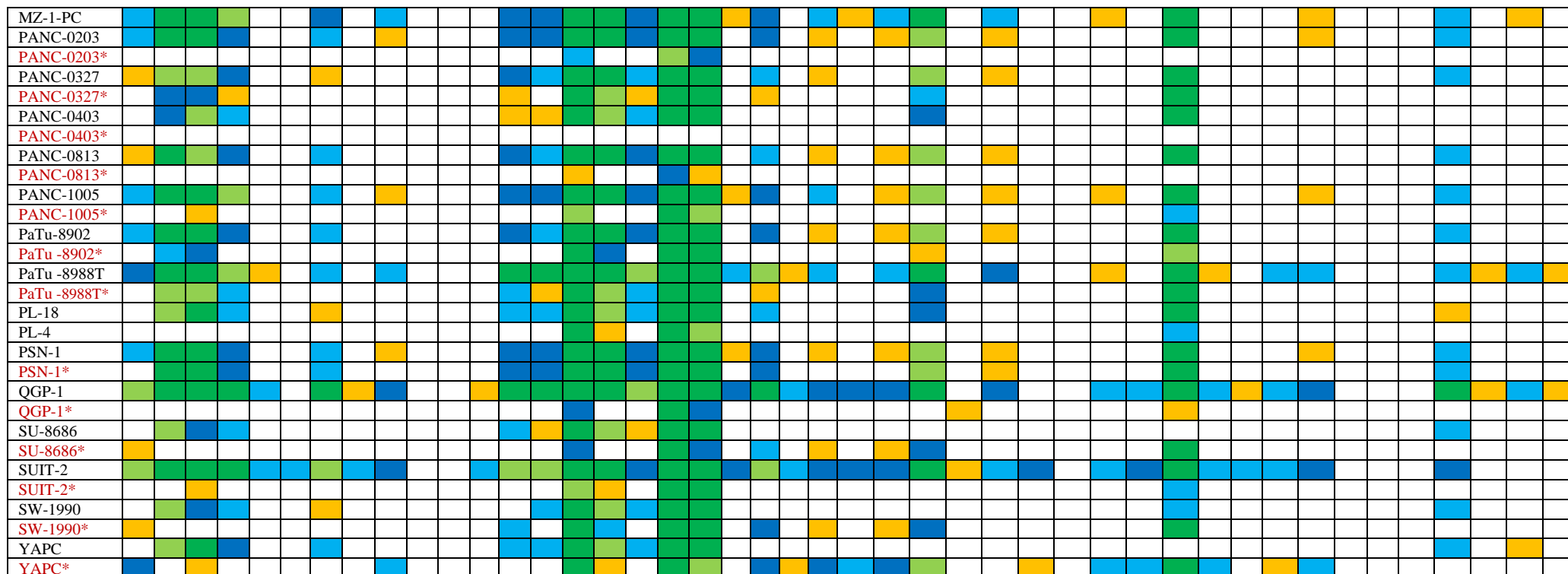
Nervous system (*Systema nervosum*)

From the data on the interpretable prognosis of sensitivity to active anti-cancer molecular forms (**Tabl. 2**) of transcriptome cell lines inherent in tumors in the Nervous system (**Tabl.4.18. a. & b.**), it is concluded that:

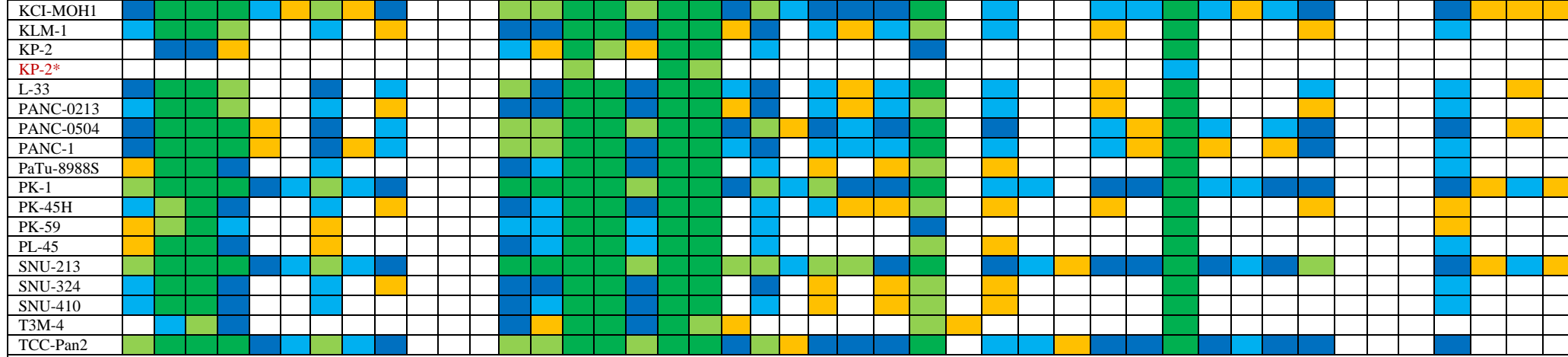
- main potential medicines: **AACF 4.C., 5.C., 23.A., 40.** and **56.B.**;
- duplication of treatment and / or substitution on medical grounds: **AACF 5.D., 19., 31.B., 34., 51.A.** and **56.A.**

Table 4.18. a Interpretable prognosis for susceptibility to active anti-cancer molecular forms, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides, of transcriptome cell lines inherent in tumors in the Nervous system: Part 1

cell line	active anti-cancer molecular forms																																										
	1								2			3	4			5				6	7	8	9	10	11	12	13	14	15	16	17	18	19	20		21	22	23		24	25	26	27
A	B	C	D	E	F	G	H	A	B	C	A		B	C	A	B	C	D	A															B	A			B	A				
glioma																																											
42-MG-BA																																											
8-MG-BA																																											
A-172																																											
AM-38																																											
Becker																																											
CAS-1																																											
CCF-STTG1																																											
D-247MG																																											
D-263MG																																											
D-336MG																																											
D-392MG																																											
D-423MG																																											
D-502MG																																											
D-542MG																																											
D-566MG																																											
DBTRG-05MG																																											
DBTRG-05MG*																																											
DK-MG																																											
DK-MG*																																											
GaMG																																											
GaMG*																																											
GB-1																																											
GB-1*																																											
GI-1																																											
GI-1*																																											
GMS-10																																											
H-4																																											
Hs-683																																											
KALS-1																																											



other




color scale of activity - in the direction of decrease: 

Table 4.26. b Interpretable prognosis for susceptibility to active anti-cancer molecular forms, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides, of transcriptome cell lines inherent in tumors in the Small intestine: Part 2

cell line	active anti-cancer molecular forms																																											
	29				30	31			32	33	34	35	36	37	38	39	40	41	42	43	44	45								46	47	48	49	50	51		52	53		54	55	56		
	A	B	C	D		A	B	C														A	B	C	D	E	F	G	H						A	B		A	B		A	B		
HuTu-80																																												
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color scale of activity - in the direction of decrease:																																												

Other pharmaceutical forms would be highly active and potential agents for personalized therapies.

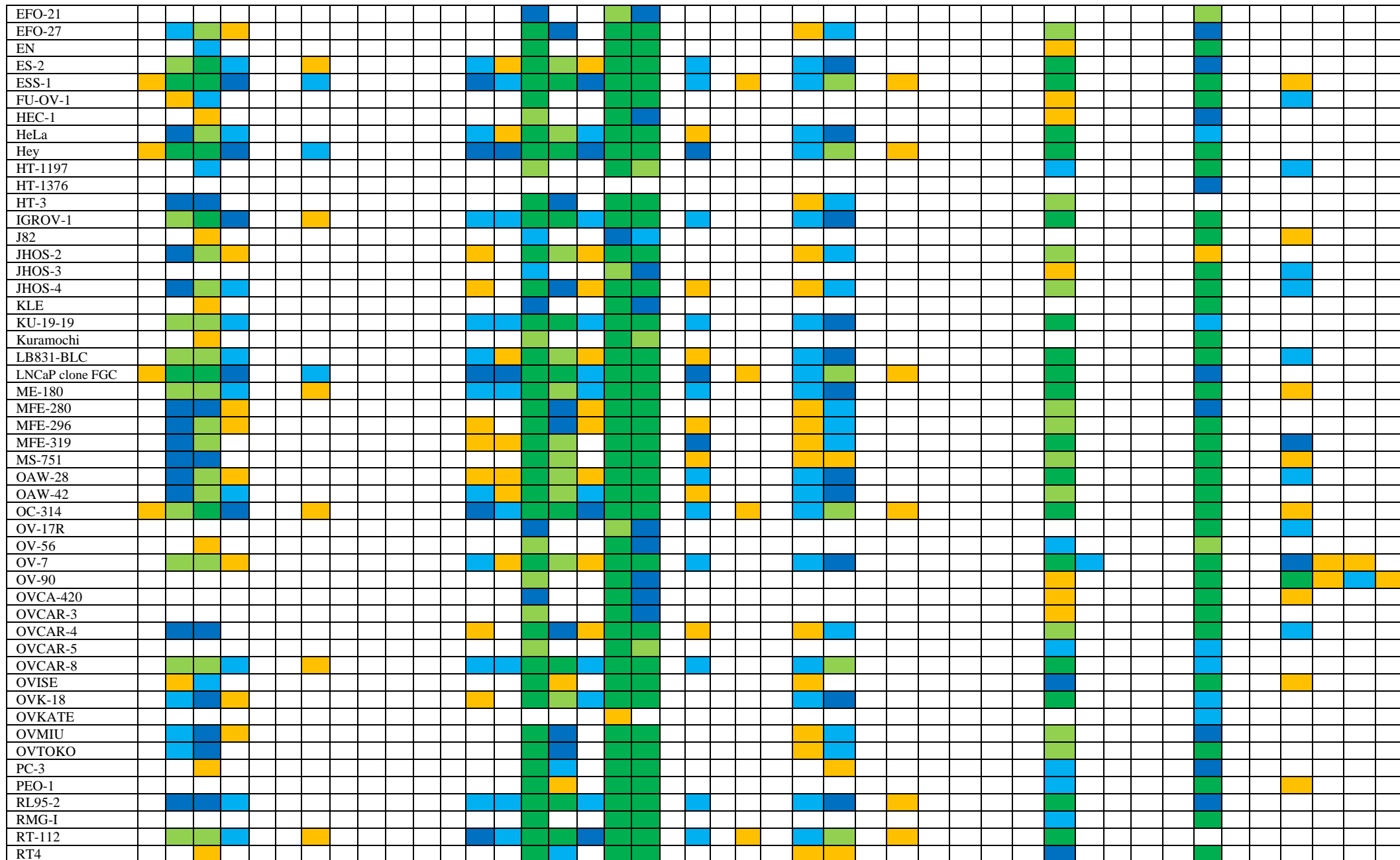
Soft tissue (*Mollis textus*)

From the data on the interpretable prognosis of sensitivity to active anti-cancer molecular forms (**Tabl. 2**) of transcriptome cell lines inherent in tumors in the Soft tissue (**Tabl.4.27. a & b.**), it is concluded that:

- main potential medicines: **AACF 1.C., 4.B., 5.C. and 40.**;
- duplication of treatment and / or substitution on medical grounds: **AACF 1.D., 4.C., 5.D., 12., 19., 23.A., 30., 31.A., 31.B., 31.C., 32., 33., 34., 36., 37., 38., 39., 42., 43., 44., 45.B., 45.E., 46., 48., 49., 51.A., 51.B., 56.A. and 56.B.**

Table 4.27. a Interpretable prognosis for susceptibility to active anti-cancer molecular forms, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides, of transcriptome cell lines inherent in tumors in the Soft tissue: Part 1

cell line	active anti-cancer molecular forms																																														
	1								2			3	4				5				6	7	8	9	10	11	12	13	14	15	16	17	18	19	20		21	22	23		24	25	26	27	28		
	A	B	C	D	E	F	G	H	A	B	C		A	B	C	D	A	B	C	D												A	B			A	B										
Ewings sarcoma-peripheral primitive neuroectodermal tumour																																															
A-673																																															
fibrosarcoma																																															
HT-1080																																															
HT-1080*																																															
SW-684																																															
leiomyoblastoma																																															
G-402																																															
G-402*																																															
leiomyosarcoma																																															
SK-UT-1																																															
SK-UT-1*																																															
liposarcoma																																															



RESULTS ANALYSIS

In *Fig.1* present (§III.3.4.2, *Tsanov & Tsanov, 2023*) the descriptive dependences of the interpretable predictions of sensitivity to active anti-cancer molecular forms of transcriptional cell lines inherent in tumors of different nature.

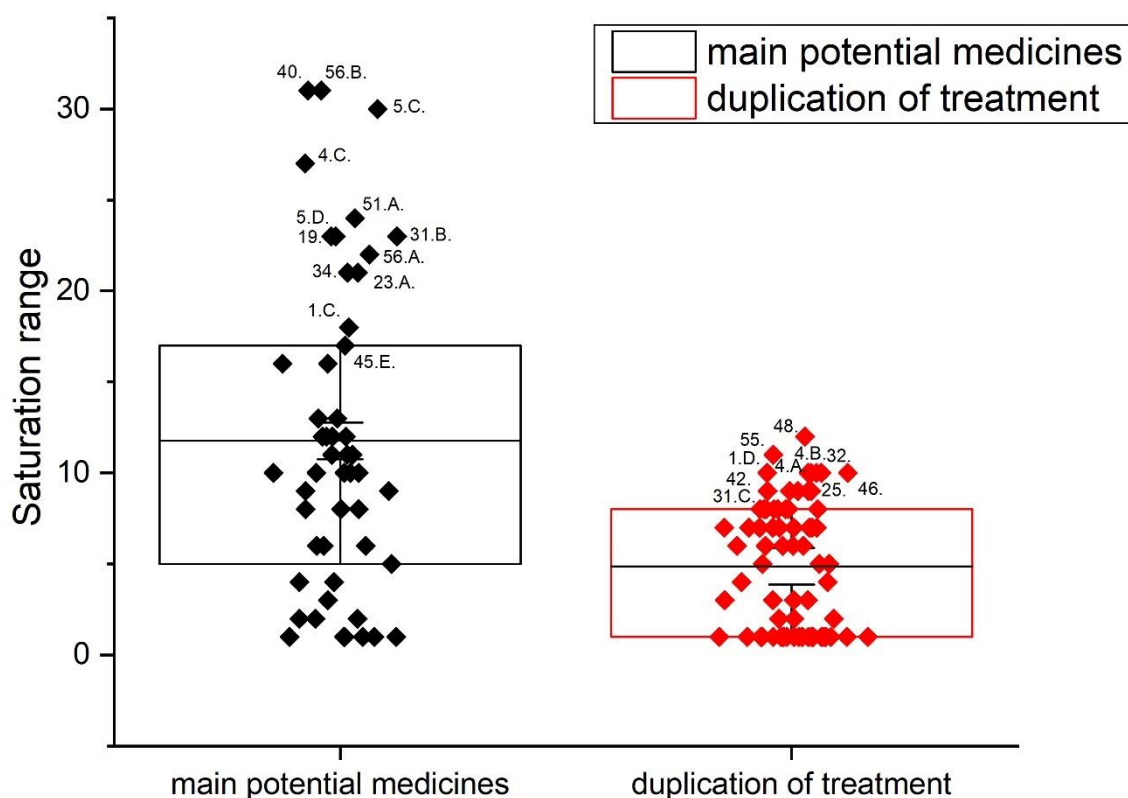


Figure 1 Schematic representation of the descriptive dependences of the interpretable predictions of sensitivity to active anti-cancer molecular forms of transcriptional cell lines inherent in tumors

They highlight some molecular forms that are extremely active. These are: for basic pharmaceutical forms /sorted by total activity/ – **40., 56.B., 5.C., 4.C., 51.A., 5.D., 19., 31.B., 56.A., 34., 23.A., 1.C.** and **45.E.**; for substitute forms – **48., 55., 1.D., 4.A., 4.B., 32., 42., 25., 31.C.** and **46.**

The final breakdown by active group and pharmaceutical form is presented in the front section.

CLINICAL CONTROL

Correlation of bio constants

The bio constants of the human organism should not be accepted as a dogma, but simply used as benchmarks of the norm. Deviation from them should not necessarily be considered pathology. The human body adapts extremely well to the environment and strives to respond adequately to each stimulus.

Especially when performing chemotherapy on cancer patients, it is necessary to monitor the overall reference picture of the patient. It is good to avoid interpreting individual deviations from the physiologically healthy organism and to direct the treatment of cancer in the direction of "suppression" of individual symptoms.

In **Tabl. 5** the control forms that the clinician must comply with before and during chemotherapy are indicated. These are reference correlations that would directly affect the release of the active anticancer molecular form (**Tabl. 2**) within the cancer cell.

- A. **VOLUME OF BLOOD:** it is directly related to the fluid ratio, and secondarily to the water content in the body - hence the change in a number of physicochemical parameters

When the total blood volume increases, it is important for the treating physician to rule out diagnoses:

- chronic leucosis;
- uremia (due to the change in nitrogen balance, which will prevent the transport of amide derivatives in the body) - is often accompanied by hyperkalemia and hyperchloremia.

When the total blood volume decreases, it is necessary to exclude the diagnoses:

- acidosis - increased water content is also reported;
- tubular acidosis - **IMPORTANT:** do not rush with renal tubular acidosis - there may also be hypophosphatemia.

B. HEMATOCRIT:

In case of a decrease in the value of the hematocrit in the blood, it is obligatory for the attending physician to reject the diagnoses:

- spherocytic anemia - at the beginning of treatment with amide / carboxylic derivative of nitrile glycosides there is a **!!! REAL POSSIBILITY FOR CRISIS !!!** ;
- this condition is often an indicator of brain metastases.

C. NATREMIA:

Particular attention should be paid to cases of hyponatremia. Here the attending physician needs to comply with diagnoses such as:

- cystic fibrosis (CP) / mucoviscidose/- in these cases treatment should begin with a very low concentration of the dosage form;
- this condition is often an indicator of lung cancer.

D. KALEMIA:

To some extent, hyperkalemia has a synergistic effect on the action of the studied dosage forms. It is good for the doctor to maintain higher blood potassium reference values. In cases where this is difficult, two circumstances must be taken into account:

- to reject Cushing's syndrome, by control test and for hyperchloremia - gives both increase and decrease;
- is often observed together with hypophosphatemia. If necessary, to introduce phosphorus preparations into the body.

E. CHLORAEMIA:

Hypochloraemia alters the ionic and electrostatic activity of both amides and carboxylic acids - especially when they are in low concentrations in the blood. It is good to consider:

- presence of liver cirrhosis - the analysis should be done at least 4 hours after glucose infusion and diuretics taken. The results should be differentiated from Hepato-renal syndrome.

F. CALCEMIA:

Hypercalcemia can suppress the spread of the drug form. Treatment should be resected and any comorbidities considered:

- extensive metastases;
- osteorenal sarcoma;
- breast carcinoma with bone metastases during treatment with ANDROGENS and ESTRONES;
- sarcoidosis.

G. SIDERINEMIA:

Hyposiderinemia could delay the detection of a more acidic environment around the cancer cell and from there slow down the action of the drug under study. The clinician should be aware that this is often accompanied by:

- ballast leukemia;
- carcinomas;
- uterine fibroids;
- myelosis and lymphadenosis (in terminal stage);
- erythraemia vera (in terminal stage).

Correction of iron in the blood is one of the most important factors in treatment with these doses.

Table 5 Correlation data of microcomponents in human blood that affect the digestibility and activity of amides and carboxylic acids derivatives of natural nitrile glycosides

control form	raise		reduction	
	indicator	indicator control	indicator	indicator control
volume of blood	chronic leucosis		acidosis	increased water content is also reported
	uremia‡	hyperkalemia	tubular acidosis*	hypophosphatemia
		hyperchloremia		
hematocrit			spherocytic anemia ☀	indicator of brain metastases
natremia ▲			Mucoviscidose **	indicator of lung cancer
kalemia			to reject Cushing's syndrome ◇	often seen with hypophosphatemia
chloraemia			indicator and of liver cirrhosis §	
calcemia	extensive metastases			
	osteorenal sarcoma			
	breast carcinoma with bone metastases during treatment with ANDROGENS and ESTROGENES			
	sarcoidosis			
siderinemia			ballast leukemia	
			carcinomas	
			uterine fibroids	
			myelosis and lymphadenosis (in terminal stage)	
			erythraemia vera (in terminal stage)	
total iron-binding capacity	bone marrow hypoplasia		hemosiderosis	
			malignant tumors	
cupremia	malignant tumors	increase in iodine in the blood		
	malignant melanoma			
	hemochromatosis			
	malignant hemopathy (T-leucosis)	increase of sulfates in the blood		
	meligenic lymphomas			
	treatment with estrogen			
	cirrhosis of the liver	reduction of iodine in the blood		
Zinc content in the blood	in the serum			
	erythraemia vera		bone marrow hypoplasia	
	atrophic cirrhosis of the liver		lymphadenosis	
	acute leucosis		myelosis	
	in erythrocytes			

	bone marrow hypoplasia	myelosis
	erythraemia vera	lifenosis
<p>*- IMPORTANT: do not rush with renal tubular acidosis - there may also be hypophosphatemia *- due to a change in the nitrogen balance ☀- REAL POSSIBILITY FOR CRISIS ! *- MANDATORY- the treatment should begin with a very low concentration of the dosage form ▲- to rule out myxedema as a concomitant disease ◇- control test and for hyperchloremia - gives both increase and decrease §- the analysis should be performed at least 4 hours after glucose infusion and diuretics taken. The results should be differentiated from Hepato-renal syndrome</p>		

H. TOTAL IRON-BINDING CAPACITY

In case of increased content of total iron-binding capacity, it is necessary for the clinician to take into account the possible presence of:

- bone marrow hypoplasia,

and at reduced content with:

- hemosiderosis;
- malignant tumors.

I. CUPREMIA:

Hypercupremia would significantly increase the need for a higher drug dose. She herself is also an indicator for:

- malignant tumors - as a control sample can be used and the increase in iodine in the blood; - malignant melanoma;
- hemochromatosis;
- malignant hemopathy (T-leukemia) - as a control sample can be used and the increase of sulfates in the blood;
- meligenic lymphomas;
- treatment with estrogen;
- cirrhosis of the liver - as a control sample can be used and the reduction of iodine in the blood.

J. ZINC CONTENT IN THE BLOOD

The content of zinc in the blood determines its extremely complex role in cancer. Its compounds are both inhibitors and promoters. It can displace a number of metals from organometallic biologically active substances, but at the same time its coordination compounds in an in vivo medium are volatile.

We recommend a mandatory blood test for zinc in the blood before starting chemotherapy according to the studied experimental methodology. The following reference deviations must be taken into account:

a. in the serum:

increased serum zinc concentration:	decreased serum zinc concentration:
<ul style="list-style-type: none"> - erythraemia vera; - atrophic cirrhosis of the liver; - acute leukemia. 	<ul style="list-style-type: none"> - bone marrow hypoplasia; - lymphadenosis; - myelosis.

b. in erythrocytes:

increased concentration of zinc in erythrocytes:	decreased concentration of zinc in erythrocytes:
<ul style="list-style-type: none"> - bone marrow hypoplasia; - erythraemia vera. 	<ul style="list-style-type: none"> - myelosis; - lifadenosis.

Chemoprevention and Homeopathy

The proposed methodological program for conservative treatment of oncological diseases does not contradict the good medical practices for chemotherapy. In order to improve the general condition of patients, chemoprevention (Lele, 2021) and/or homeopathy could be applied, but not at the expense of a varied diet, incl. table salt, water, culinary acidifiers and fats. Alternative medicine should only be used to treat individual symptoms, not syndromes.

- ◇ -

THE CONCLUSIONS OF THE PREVIOUS STUDIES

On the first goal (Tsanov & Tsanov, 2020)

Our legacy of the *Hunza people* and the knowledge from tens-of-thousands of scientists who created modern synthesis and biochemistry make the production of nitrile amide into a routine (especially with nitrile hydratase). Thus, humanity holds in its hands a huge medicinal resource that can provide treatment for diseases of all parts of conservative medicine (including all listed in **Section 1.1.**).

The hydrolyzed to amide/carboxylic acid nitrile/cyanide carbohydrates will occupy one of the fundamental steps of countless future clinical practices. This is the purpose of our modest research!

Other substances in these groups with pronounced biological activity (including anti-tumor) are the hydrolyzed nitrile groups of *Linamarin*, (*R*)-*Lotaustralin*, *S-Sambunigrin*, etc., to their amide/carboxylic acid.

The second goal (Tsanov & Tsanov, 2021)

- 1) The amide derivatives of nitrile glycosides are potential chemical compounds with anti-cancer activity;
- 2) the cancer cell seeks to shift the hydrolysis of these derivatives in a direction that would not pass through its cell membrane;
- 3) the amide-carboxylic derivatives of nitrile glycosides can deliver extremely toxic compounds inside the tumor cell itself and thus block and / or permanently damage its normal physiology;
- 4) the use of these compounds in oncology could turn cancer from a lethal to a chronic disease (such as diabetes). The cause and conditions of the disease are not eliminated, but the number of cancer cells could be kept low for a long time (even a lifetime).

On the third goal (Tsanov & Tsanov, 2022)

- 1) Amides resulting from the hydrolysis of nitrile glycosides would be able to cross the cell membrane of a cancer cell and thus cause its cellular response;
- 2) the pharmaceutical form must represent the exact amide/carboxylic acid ratio for the corresponding active anticancer cell form;
- 3) clinical concentrations are more than 7 times higher than those of nitrile glycosides due to their reduced toxicity;
- 4) no significant deviations are observed, on a theoretical level, in the complex use of several pharmaceutical forms together and/or sequentially.

APPLYING THE RESULTS

The results of the analysis show that the studied molecular forms do NOT contradict conservative oncology. Their activity is significant and many times exceeds a number of approved products for treatment (§IV.3.2., p.718; Tsanov & Tsanov, 2023) (Tsanov & Tsanov, 2022).

This anti-cancer agents could best be administered orally (§IV.1., p.57; Tsanov & Tsanov, 2023).. Their toxicity is many times less than that of most references accepted in clinical chemotherapy (§IV.3.1., p.85; Tsanov & Tsanov, 2023) (Tsanov & Tsanov, 2020).

After analyzing the conclusions on the previous goals of the research (§IV.1÷3., p.718; Tsanov & Tsanov, 2023) (Tsanov & Tsanov, 2021), taking into account the presented clinical control, the data for the interpretable prognosis for susceptibility to active anti-cancer molecular forms of transcriptome cell lines inherent in tumors - for each anatomical system, good medical practices and the data from **Fig. 1.**, we conclude that the treatment of oncological diseases should be carried out in four stages (**Fig.2.**):

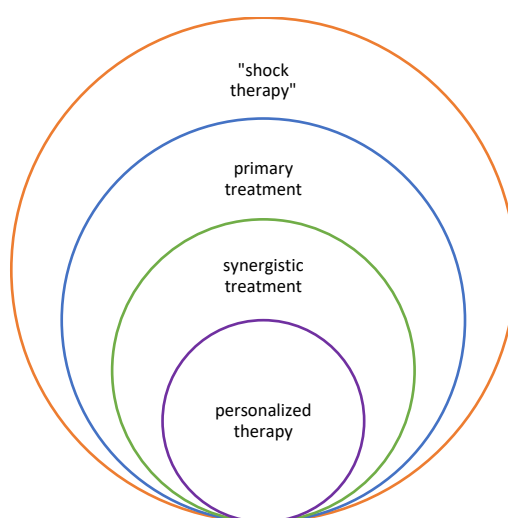
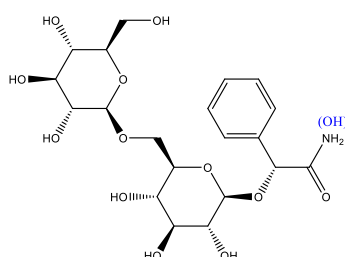


Figure 2 Schematic representation of the sequence of treatment with amide and carboxyl derivatives of natural nitrile glycosides of cancer cell lines

- “shock therapy” - in the case of a primary diagnosis of the presence of oncological disease and/or observation of pathological activity of cancer cells (including the growth of tumor tissues), the patient should be treated with:



(R)-2-phenyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)acetamide

(R)-2-phenyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)acetic acid

Thus, the active anticancer molecule released inside the cell itself will be ((*R*)-2-hydroxy-2-phenylacetamide, which in turn would pass in a small amount into the carboxyl form - (*R*)-2-hydroxy-2-phenylacetic acid (§IV.2.3.2.). Applying pharmaceutical form **1.B. (Tabl. 1.)**, the concentration of the amide toxic to the cancer cell is 0.40 mg/ml, compared to the pharmaceutical oral form (§IV.2. 4., Tsanov & Tsanov, 2023). Based on the analysis for determining the medicinal dose of the amide and carboxylic derivatives of amygdalin (§IV.1. 3., Tsanov & Tsanov, 2023), it follows that the treatment dose for the ratio, **-amide: -carboxylic = 4.87: 1** is:

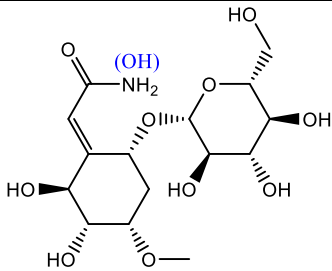
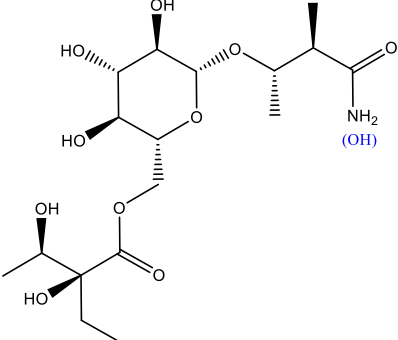
210-465 mg PO q6-8hr; not to exceed 2.1 g/day.

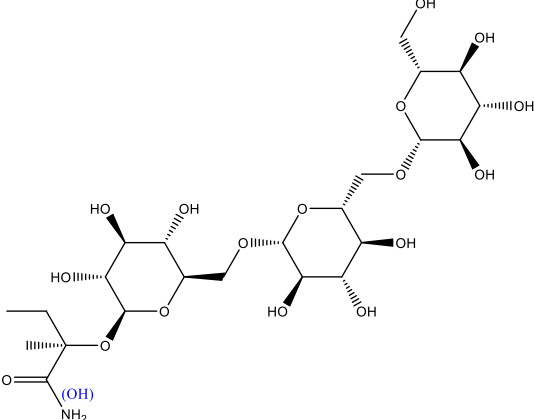
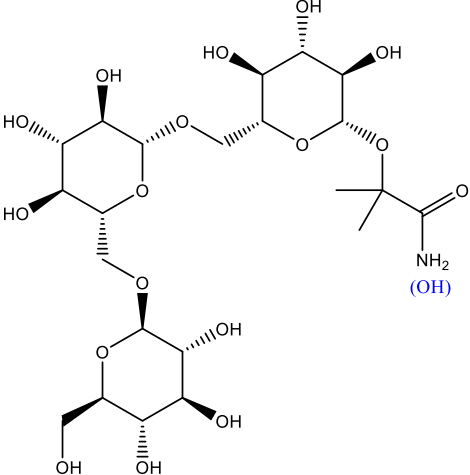
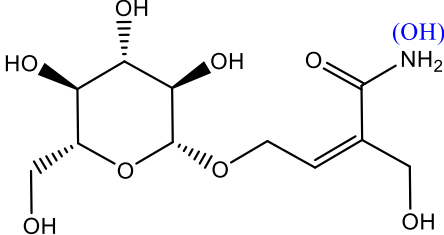
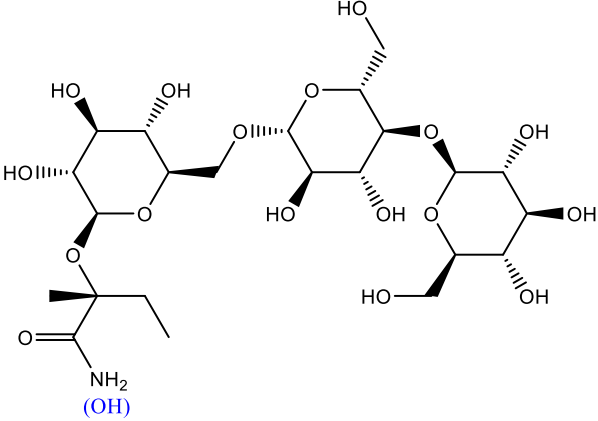
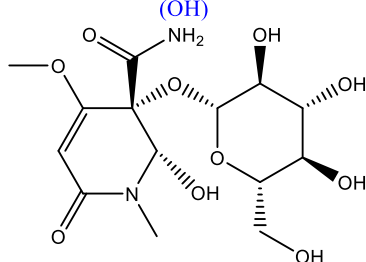
!!! If the patient shows allergic symptoms, he should undergo diuretic cleansing, after consultation with a nephrologist - after which the treatment should be started again. If similar symptoms appear again, this stage of treatment can be skipped **!!!**

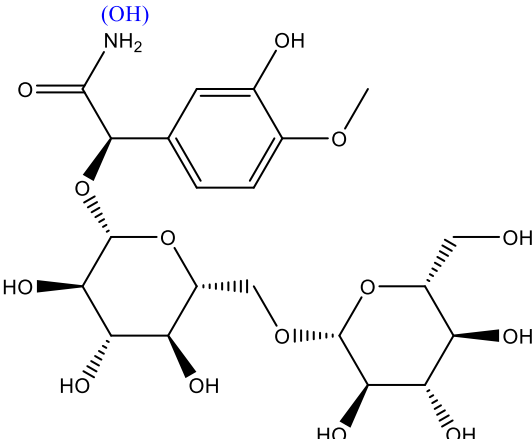
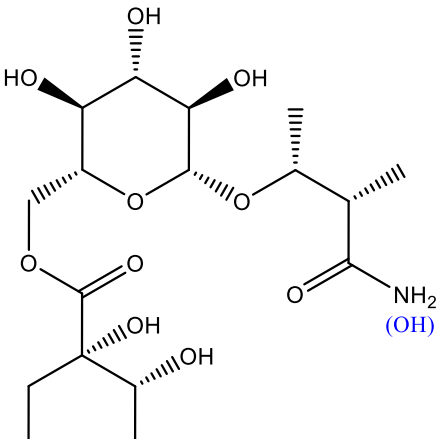
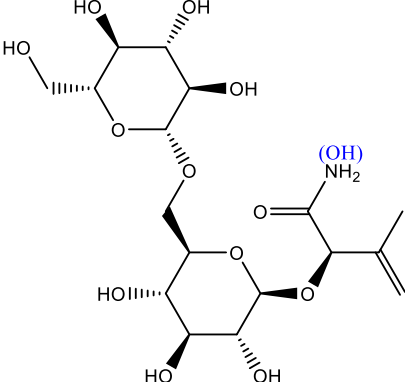
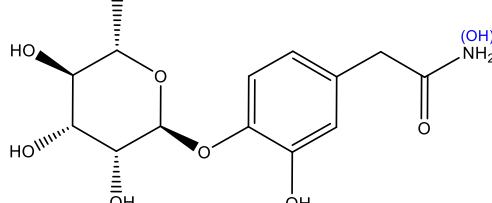
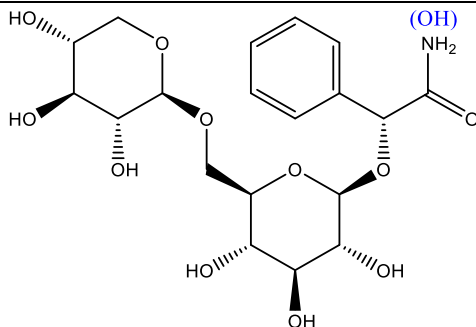
- primary treatment – after making the final diagnosis and determining the exact active cell lines, the clinical oncologist must proceed to the actual treatment - **Introduction of the main active pharmaceutical form into the body.**

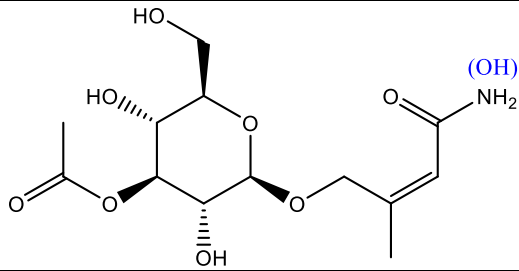
This concept includes clinical selection from a pre-calculated set of substances active enough to influence the physiological activity of the cancer cell. This stage aims to "attack" the cancer cells in a wider range of cell lines, incl. those that overlap with other cancer formations (including metastases), which at this moment may be in another stage of their development. The choice is made between the chemical compounds from the **Tabl. 6.**

Table 6 Basic pharmaceutical forms for oral use for primary treatment of cancer, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides

login number*	active pharmaceutical forms for oral use**	UPAC component names**
40.		<p>(Z)-2-((2S,3R,4S,6R)-2,3-dihydroxy-4-methoxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)cyclohexylidene)acetamide</p> <p>(Z)-2-((2S,3R,4S,6R)-2,3-dihydroxy-4-methoxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)cyclohexylidene)acetic acid</p>
56.B.		<p>((2R,3S,4S,5R,6R)-6-(((2S,3R)-4-amino-3-methyl-4-oxobutan-2-yl)oxy)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)methyl (2S,3R)-2-ethyl-2,3-dihydroxybutanoate</p> <p>(2R,3S)-3-(((2R,3R,4S,5S,6R)-6-(((2S,3R)-2-ethyl-2,3-dihydroxybutanoyl)oxy)methyl)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)-2-methylbutanoic acid</p>

5.C.		<p>(R)-2-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)butanamide</p> <p>(R)-2-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)butanoic acid</p>
4.C.		<p>2-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)propanamide</p> <p>2-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)propanoic acid</p>
51.A.		<p>(Z)-2-(hydroxymethyl)-4-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)but-2-enamide</p> <p>(Z)-2-(hydroxymethyl)-4-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)but-2-enoic acid</p>
5.D.		<p>(R)-2-(((2S,3R,4S,5S,6R)-6-(((2R,3R,4R,5S,6R)-3,4-dihydroxy-6-(hydroxymethyl)-5-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)tetrahydro-2H-pyran-2-yl)oxy)methyl)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)-2-methylbutanamide</p> <p>(R)-2-(((2S,3R,4S,5S,6R)-6-(((2R,3R,4R,5S,6R)-3,4-dihydroxy-6-(hydroxymethyl)-5-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)tetrahydro-2H-pyran-2-yl)oxy)methyl)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)-2-methylbutanoic acid</p>
19.		<p>(2S,3S)-2-hydroxy-4-methoxy-1-methyl-6-oxo-3-(((2R,3S,4R,5R,6S)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)-1,2,3,6-tetrahydropyridine-3-carboxamide</p> <p>(2S,3S)-2-hydroxy-4-methoxy-1-methyl-6-oxo-3-(((2R,3S,4R,5R,6S)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)-1,2,3,6-tetrahydropyridine-3-carboxylic acid</p>

31.B.		<p>(R)-2-(3-hydroxy-4-methoxyphenyl)-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)acetamide</p> <p>(R)-2-(3-hydroxy-4-methoxyphenyl)-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)acetic acid</p>
56.A.		<p>((2R,3S,4S,5R,6R)-6-(((2R,3S)-4-amino-3-methyl-4-oxobutan-2-yl)oxy)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)methyl (2S,3R)-2-ethyl-2,3-dihydroxybutanoate</p> <p>(2S,3R)-3-(((2R,3R,4S,5S,6R)-6-(((2S,3R)-2-ethyl-2,3-dihydroxybutanoyl)oxy)methyl)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)-2-methylbutanoic acid</p>
34.		<p>(R)-3-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)but-3-enamide</p> <p>(R)-3-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)but-3-enoic acid</p>
23.A.		<p>2-(3-hydroxy-4-(((2S,3R,4R,5R,6S)-3,4,5-trihydroxy-6-methyltetrahydro-2H-pyran-2-yl)oxy)phenyl)acetamide</p> <p>2-(3-hydroxy-4-(((2S,3R,4R,5R,6S)-3,4,5-trihydroxy-6-methyltetrahydro-2H-pyran-2-yl)oxy)phenyl)acetic acid</p>
1.C.		<p>(R)-2-phenyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2S,3R,4S,5R)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)acetamide</p> <p>(R)-2-phenyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((2S,3R,4S,5R)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)acetic acid</p>

45.E.		<p>(2R,3R,4S,5R,6R)-2-(((Z)-4-amino-2-methyl-4-oxobut-2-en-1-yl)oxy)-3,5-dihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-4-yl acetate</p>
		<p>(Z)-4-(((2R,3R,4S,5R,6R)-4-acetoxy-3,5-dihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)-3-methylbut-2-enoic acid</p>
<p>*- sorted by general activity - descending ** - this table represents a sample of Tabl. 2 and the data from §IV.4. 2. of (Tsanov & Tsanov, 2023) *** - it is the most active isomeric molecular form. Any kind of optical isomerism is allowed.</p>		

The clinical approach here requires the selection of an active pharmaceutical form (**Tabl. 6**) against the Interpretable prediction of sensitivity to active anticancer molecular forms of transcriptomic cell lines intrinsic to tumors. Here the rule of greater activity is observed.

EXAMPLE:

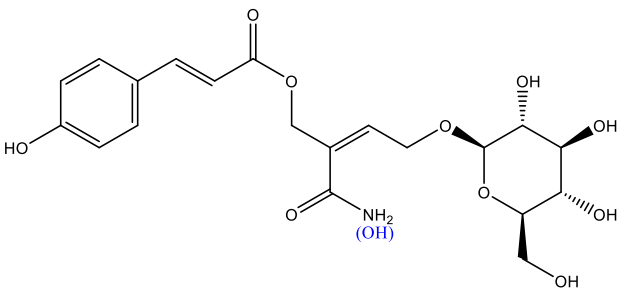
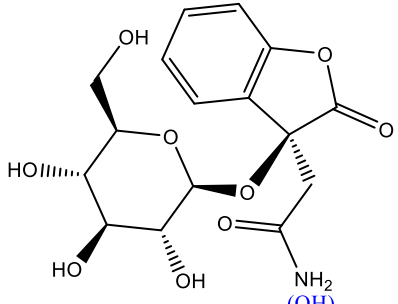
for leukemia (§FINAL RESULTS/ Leukemia):

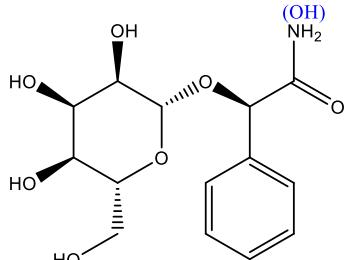
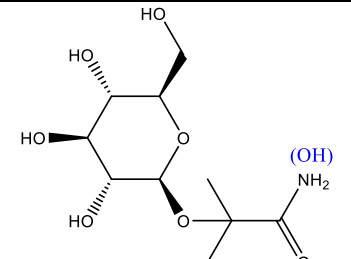
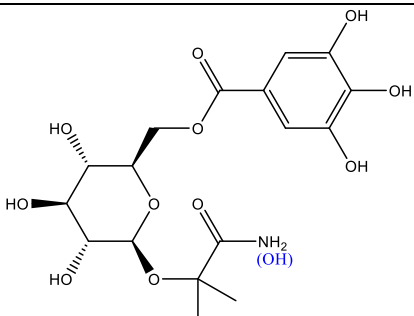
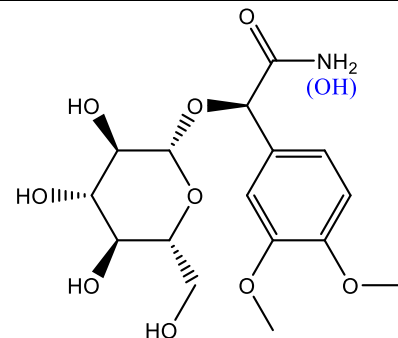
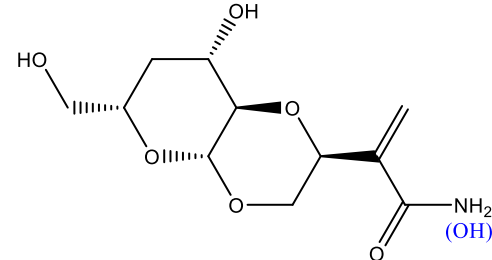
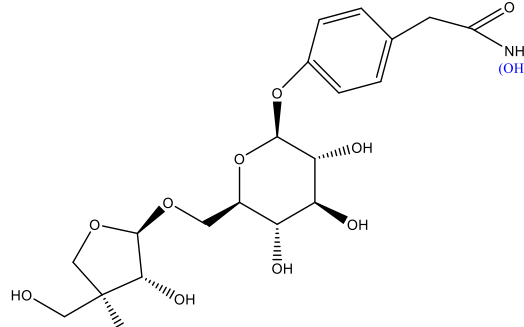
- basic potential medicines: AACF 1.C., 4.C., 5.C., 5.D., 19, 25, 30, 31.B., 31.C., 34, 39, 40, 45.E, 50, 56.A. and 56.B.;
- duplication of treatment and / or substitution on medical grounds: AACF 1.D., 4.A., 4.B., 5.A., 5.B., 7, 12, 32, 33, 42, 43, 44, 45.B., 49, 50, 51.A. and 51.B..

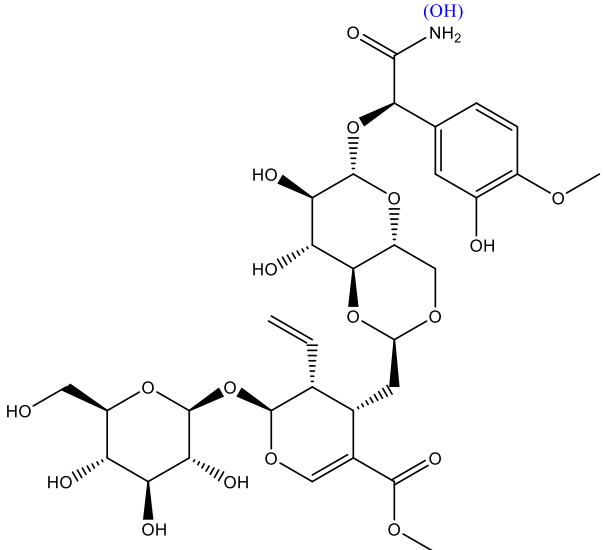
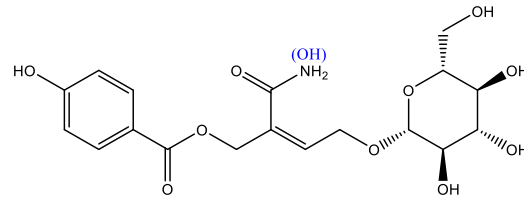
According to **Tabl. 7** it follows that the choice of be:

- basic potential medicines: AACF 40 /in this case the 1st of general activity/;
- and **Tabl. 7** then:
- duplication of treatment and / or substitution on medical grounds: AACF 1.D. /in this case third in overall activity /.

Table 7 Substitute pharmaceutical forms for oral use for primary treatment of cancer, based on amides/carboxylic acids - hydrolysis derivatives of natural nitrile glycosides

login number*	active pharmaceutical forms for oral use**	UPAC component names**
48.		<p>(Z)-2-carbamoyl-4-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)but-2-en-1-yl (E)-3-(4-hydroxyphenyl)acrylate</p>
		<p>(Z)-2-(((E)-3-(4-hydroxyphenyl)acryloyl)oxy)methyl-4-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)but-2-enoic acid</p>
55.		<p>2-((S)-2-oxo-3-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)-2,3-dihydrobenzofuran-3-yl)acetamide</p>
		<p>2-((S)-2-oxo-3-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)-2,3-dihydrobenzofuran-3-yl)acetic acid</p>

1.D.		(R)-2-phenyl-2-(((2S,3R,4R,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)acetamide
		(R)-2-phenyl-2-(((2S,3R,4R,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)acetic acid
4.A.		2-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)propanamide
		2-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)propanoic acid
4.B.		((2R,3S,4S,5R,6S)-6-((1-amino-2-methyl-1-oxopropan-2-yl)oxy)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)methyl 3,4,5-trihydroxybenzoate
		2-methyl-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(((3,4,5-trihydroxybenzoyl)oxy)methyl)tetrahydro-2H-pyran-2-yl)oxy)propanoic acid
32.		(R)-2-(3,4-dimethoxyphenyl)-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)acetamide
		(R)-2-(3,4-dimethoxyphenyl)-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)acetic acid
42.		2-((2S,4aR,6S,8S,8aR)-8-hydroxy-6-(hydroxymethyl)hexahydro-6H-pyrano[2,3-b][1,4]dioxin-2-yl)acrylamide
		2-((2S,4aR,6S,8S,8aR)-8-hydroxy-6-(hydroxymethyl)hexahydro-6H-pyrano[2,3-b][1,4]dioxin-2-yl)acrylic acid
25.		2-(4-(((2S,3R,4S,5S,6R)-6-(((2R,3R,4R)-3,4-dihydroxy-4-(hydroxymethyl)tetrahydrofuran-2-yl)oxy)methyl)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)phenyl)acetamide
		2-(4-(((2S,3R,4S,5S,6R)-6-(((2R,3R,4R)-3,4-dihydroxy-4-(hydroxymethyl)tetrahydrofuran-2-yl)oxy)methyl)-3,4,5-trihydroxytetrahydro-2H-pyran-2-yl)oxy)phenyl)acetic acid

31.C.		<p>methyl (2S,3R,4S)-4-(((2R,4aR,6S,7R,8R,8aS)-6-((R)-2-amino-1-(3-hydroxy-4-methoxyphenyl)-2-oxoethoxy)-7,8-dihydroxyhexahydropyrano[3,2-d][1,3]dioxin-2-yl)methyl)-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)-3-vinyl-3,4-dihydro-2H-pyran-5-carboxylate</p>
46.		<p>(R)-2-(((2R,4aR,6S,7R,8R,8aS)-7,8-dihydroxy-2-(((2S,3R,4S)-5-(methoxycarbonyl)-2-(((2S,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)-3-vinyl-3,4-dihydro-2H-pyran-4-yl)methyl)hexahydropyrano[3,2-d][1,3]dioxin-6-yl)oxy)-2-(3-hydroxy-4-methoxyphenyl)acetic acid</p> <p>(Z)-2-carbamoyl-4-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)but-2-en-1-yl 4-hydroxybenzoate</p> <p>(Z)-2-(((4-hydroxybenzoyl)oxy)methyl)-4-(((2R,3R,4S,5S,6R)-3,4,5-trihydroxy-6-(hydroxymethyl)tetrahydro-2H-pyran-2-yl)oxy)but-2-enoic acid</p>
<p>*- sorted by general activity – descending ** - this table represents a sample of <i>Tabl. 2</i> and the data from §IV.4. 2. of (<i>Tsanov & Tsanov, 2023</i>) *** - it is the most active isomeric molecular form. Any kind of optical isomerism is allowed.</p>		

If for some reason (financial, logistical, allergic, etc.) the first form cannot be used, then the next molecular form is chosen and so on. The same applies if the activity is too great and this leads to complications in other diseases (including chronic ones for the patient). Then apply (with the same selection) – the substitute per oral pharmaceutical forms in the main treatment of cancer (*Tabl. 7*).

Based on the analysis for determining the medicinal dose of the amide and carboxylic derivatives of amygdalin (§IV.1. 3., *Tsanov & Tsanov, 2023*), it follows that the treatment dose for the ratio, - **amide: -carboxylic = 4.87: 1** is:

210-655 mg PO q6-8hr; not to exceed 2.3 g/day.


!!! If the patient shows allergic symptoms, he should undergo diuretic cleansing, after consultation with a nephrologist - after which the treatment should be started again **!!!**

- synergistic treatment – the very genesis of the treatment we offer is based on chronification of the disease, i.e. its transformation into controlled over time, by means of taking medicinal forms.

That is why there will inevitably come a time when the activity of the preparation will sharply decrease. At this point, the body's clinical response will be directed against the treated substances. That's why we have planned for a certain period of time to replace the medicinal products with those that will

preserve the overall activity. Medicinal substances are divided into two tables for each type of cancer, according to **§IV.4.1.** – conditionally accepted for **Part 1** and **Part 2**. Thus, the clinical doctor can easily select the substitute substances, observing the rule of substitutability according to tables, i.e. if he has chosen a medicinal form from the table. with a **Part 1** title, he makes a choice with a **Part 2** title (following exactly the same rules).

- Personalized therapy - in a small number of cases, it is necessary to act individually according to the patient's current anamnesis. For this purpose, after the application of the general therapy and / or in combination with it, the specific agent for the respective cell lines must be taken (**§.4.1.**, *Tsanov & Tsanov, 2023*). The ratio of amide to carboxylic acid should again be 4.87:1.

The doctor is only required to comply with the color assessment of activity (activity decreases in order (**§.III.3.4.1.**, *Tsanov & Tsanov, 2023*) - )

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AUTHOR'S NOTES

With the present scientific work we have tried to present in a more generalized form our long-term theoretical research. We tried to draw every value, every dependence and every conclusion precisely, in a form that is not subject to any personal view and/or to be enslaved to a generally "accepted" opinion.

Natural nitrile glycosides would not cross the tumor cell membrane. They decompose to HCN-acid, phenyl methanol and carbohydrate. They do NOT have antitumor activity due to their inability to reach the target unchanged. These compounds, in their natural form, are extremely toxic to the human body. Applying them is not a cure, even at higher concentrations they do more harm than good. Theoretically, we have derived dozens of their modified forms, but their amides and carboxylic acids are the most promising for their introduction in conservative oncology. The fact is that the tumor cell itself is trying to counteract in a way that is quite safe for it.

The knowledge that humanity has gained from the millennial battle between it and tumors, combined with the development of mathematics, statistical and quantum molecular thermodynamics, molecular topology and geometry, clinical oncology, pathophysiology, etc., with the unequivocal contribution of thousands of scientists, we tried to we present this thesis as a sentence and the most modest way to try to confirm and prove it.

- ◇ -

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

HUMAN AND ANIMAL RIGHTS

No Animals/Humans were used for studies that are the basis of this research.

CONSENT FOR PUBLICATION

Not applicable.

AVAILABILITY OF DATA AND MATERIALS

The authors confirm that the data supporting the findings of this study are available within the article.

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CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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