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A HYPOTHESIS THAT CORRELATE MOLECULAR WEIGHTS AND NUMERICAL TECHNIQUES AS AN OPTIMIZATION TOOL TO DESIGN ESTERS WITH POTENTIAL ANTICANCER ACTIVITY

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ABSTRACT

The present work relates to a hypothesis that correlates between the molecular weights and the application of numerical techniques such as Newton-Raphson for the first time as an optimization tool to design water soluble esters with potential anticancer activity in vitro and in vivo. The results obtained from this application suggested a consistent and clear correlation between the experimental and numerical analysis results. However, further studies are required to confirm this correlation.

KEYWORDS: Hypothesis, Correlation, Molecular weights, Numerical Techniques, Esters, Anticancer Activity.

INTRODUCTION

Although thousands of potentially active anticancer agents have been evaluated during the last 50 years, however, there is always a constant need to develop new anticancer drugs. Cancer is responsible for millions of death worldwide and continues to remain one of the leading cause of death each year. According to World Health Organization (WHO), there were an estimated 15.270 million new cancer cases and 8.2 million cancer death in 2017 compared to 12.7 million and 7.6 million respectively in 2008.[1]

It is projected that the number of new cancer cases and cancer death worldwide will rise to 21 and 13 million respectively, by 2030. In the USA, there were an estimated 1.7 million new cancer cases and over half a million cancer death in 2016.[2]

With the advances in anticancer drug discovery and development in the last several decades, more than 100 anticancer drugs have been approved by the FDA (7&8). Broadly speaking; these drugs can be classified into two basic categories: cytotoxic and targeted agents based on their mechanism of action.[5-7] However, it is well established that the process of discovery and development of novel drugs is known to be time-consuming and expensive. On average, the standard process of drug discovery and development to making marketing takes 10-15 years. Furthermore, the average cost of research and development of each effective drug is estimated at $1.8 billion USD.[6] Nevertheless, there is always a constant need to develop new anticancer and new therapeutic agents.

In this context, it is not surprising that the development of new strategies over the last decades has emerged to make the process much rationale and efficient using new approaches. Therefore, as a part of our recent efforts in searching for safe, targeted, water soluble, with well tolerable and effective pharmaceutical anticancer compounds, and in order to cut short the process of drug development, which is always described complex, risky, expensive and time consuming, we synthesized some free soluble esters of {(N-(4-amino-2-butynyl)}, and fully characterized them by physico-chemical methods.

MATERIALS AND METHODS

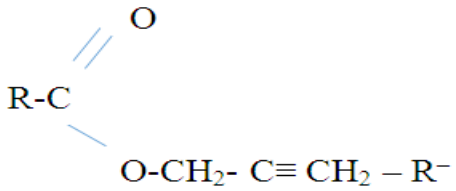
1. Chemistry

Although comprehensive investigations about organic compounds containing 2-butyne(X-CH2-C_C-CH2-X' substitutes (usually amino groups) were carried out and their biological activities as Muscarinic Receptors

inhibitors were examined[9-18], however, nothing is known about their anticancer activities. On the other hand, there is a research work about the ester of 2-butyne reported in Russian article[11] in which X represents a terephthalate or methacrylate group and X' represents N-morpholino or N-piprydino group. However, these compounds have been a target for an industrial usage as substrate for the preparation of polymers and copolymers.

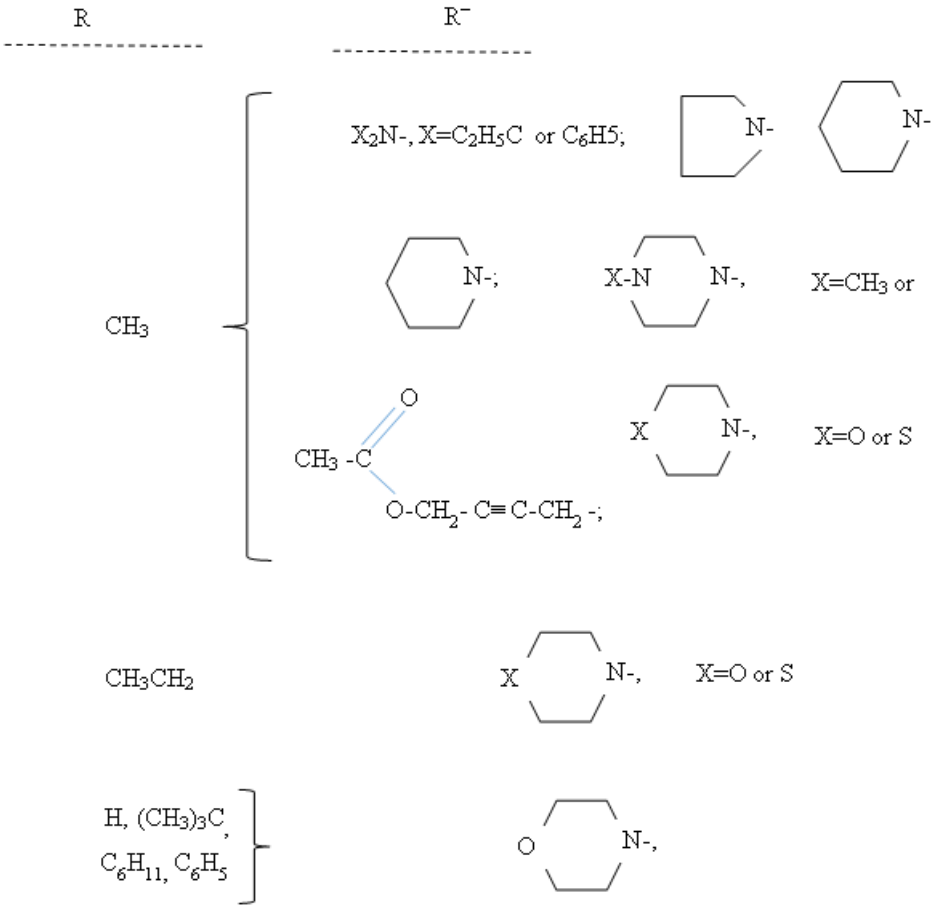
Furthermore, upon searching in the literature during the period between 1966 up to date, we found that there were apparently several articles[12] mentioned about the chemistry of 2-butyne esters with similar structures to our compounds, those listed in Table.1 vide infra. Nevertheless, no work found on anti-tumor activity of esters were found in the literature, a part of fairly related work on the three compounds containing an acetylenic nucleus (propargl group), which are considered to possess antitumor activity.[12]

We, therefore, presenting in this paper the synthesis, characterization and antitumor activity in vitro and in vivo of some of these esters {N-(4-amino-2butynyl)} with the general formula:



Where R=H, methyl, ethyl, trimethylmethyl, cyclohexyl, phenyl.
R^{--} =N-tertiary or N-quaternary analogous amino group

Table 1: The esters prepared in this work.



1.1. Preparations of Esters

The {N-(4-amino-2-butynyl1)} esters were prepared by the successive conversions of propargyl alcohol into the corresponding propargyl ester by a simple esterification with a respective organic acid with or without organic solvent. The propargyl ester was then converted into the corresponding N-(4-amino-2-butynyl) ester by Mannish condensation with paraformaldehyde, acetic acid or a respective acid and Cu2Cl2 (as a catalyst) in dioxane.

The following esters are selected from the group above and comprising:
L2= {N-(4-morpholino-2-butnyl)} acetate
L4= {N-(4-morpholino-2-butynyl)} benzoate
L6= {N-(4-morpholino-2-butynyl)} formats
L9= {N-(4-diethylamino-2-butynl)} acetate
L12= {N-(4-diphenylamino-2-butynl)} acetate
L18= {N-(4-morpholino-2-butnyl)} pivalate
L20=N, N'-{(4, 4-piprazino)-bis-(2-butynl)} diacetate

L22= {N-(4-morpholino-2-butynl0} cyclohexyl carboxylate

2. Biology

2.1. Cell lines

A panel of tumor cell lines consists bladder(BXF RT112); colon(CXF COLO205); gastric(GXA MKN45); head and neck(HNXF CAL-27); non-small cell lung(LXFA 526L,LXFL-529L); breast(MAXF MDA-MB-231, MAXF SK-BR-3); pancreatic(PAXF 1657L), and renal cancer(RXF SN12C). Non-PDX-derived cell lines were either kindly provided by the NCI (Bethesda, MD), or were purchased from ATCC (Rockville, MD) DSMZ (Branuschweig, Germany), or JCRB (Japanese Collection of Research Biosources Cell Bank, Japan)m whereas, LXFA 526L, LXFL 529L and PAXF 1657L were established at former Oncotest GmbH in Freiburg (Table 2).

Table 2: Authenticated cell lines used for the study.

	Cell Line				STR
#	type		name	origin	Analysis
1	Bladder	BXF	RT112	DSMZ	authentic
2	Colon	CXF	COLO 205	NCI	authentic
3	Gastric	GXA	MKN45	JCRB #0254	authentic
4	Head & Neck	HNXF	CAL-27	DSMZ	authentic
5	Lung	LXFA	526L	Xenograft, Freiburg	authentic
6	Lung	LXFL	529L	Xenograft, Freiburg	authentic
7	Mammary	MAXF	MDA-MB-231	ATCC	authentic
8	Mammary	MAXF	SK-BR-3	ATCC, HTB-30	authentic
9	Pancreas	PAXF	1657L	Xenograft, Freiburg	authentic
10	Renal	RXF	SN12C	NCI	authentic
JCRB: Japanese Collection of Research Biosources					
ATCC : American Type Culture Collection, Rockville, MD, USA					
NCI: National Cancer Institute, Bethesda, MD, USA.					
DSMZ : Deutsche Sammlung von Mikroorganismen und Zellkulturen, Braunschweig, Germany.					

2.2. Cell Culture

Cell lines were routinely passaged once or twice weekly and maintained in culture for up to 20 passages. All cells were grown at 37°C in a humidified atmosphere with 5% CO_2 in RPMI 1640 medium (25 mM HEPES, with L-glutamine, #FG1385, Biochrom, Berlin, Germany) supplemented with 10% (v/v) fetal calf serum (Sigma, Taufkirchen, Germany) and 0.1 mg/mL gentamicin (Life Technologies, Karlsruhe, Germany).

2.3. Anti-Cancer Assay

A modified propidium iodide (PI) based monolayer assay was used to assess the anti-cancer activity of the test articles.[21] Briefly, cells were harvested from exponential phase cultures, counted and plated in 96 well flat-bottom microtiter plates at a cell density of 6,000 to 12,000 cells/well dependent on the cell line's growth rate. After a 24 h recovery period, to allow the cells to resume exponential growth, 10 µl of culture medium (4 control wells/cell line/plate) or of culture medium with test articles were added. The 8 compounds were applied at ten concentrations in half-log increments to 0.3 (v/v). After four days of treatment, cells were next washed with 200 µl PBS to remove dead cells and debris, then 200 µl of a solution containing 7 µg/ml propidium iodide (PI) and 0.1% (v/v) Triton X-100 was added. After an incubation period of 1-2 hours at room temperature, fluorescence (FU) was measured using the Enspire Multimode Plate Reader (excitation $\lambda = 530$ nm, emission $\lambda = 620$ nm) to quantify the amount of attached viable cells.

2.4. Calculation of IC50 and IC70

IC_{50} and IC_{70} values were calculated by 4 parameters non-linear curve fit using Oncotest Warehouse Software. For calculation of mean IC_{50} and IC_{70} values the geometric mean was used.

2.5. Data Evaluation

An assay was considered fully evaluable if the following quality control criteria were fulfilled:

- Z'-factor calculated within the assay plate ≥0.5.[22]
- Fluorescence intensity of >500 U from the untreated control wells, equivalent to a control/background ratio >3.0
- Coefficient of variation in the growth control wells ≤30%

2.6. Sigmoidal Concentration Response Curve

Drug effects were expressed in terms of the percentage of the fluorescence signal, obtained by comparison of the

mean signal in the treated wells with the mean signal of the untreated controls (expressed as T/C-value [%]):

$$\frac{T}{C}[\%] = \frac{\text{mean fluorescence signal}_{\text{treated group}}}{\text{mean fluorescence signal}_{\text{control group}}} \cdot 100$$

Sigmoidal concentration-response curves were fitted to the data points obtained for each compound using 4 parameters non-linear curve fit (Oncotest Data Warehouse Software). IC values are reported as absolute and relative IC_{50} and absolute IC_{70} values. The absolute IC_{50} value reflects the concentration of the test compound that achieves T/C=50%. The absolute IC_{70} value gives the concentration of the test compound that achieves T/C=30%.

2.7. In Vitro Anti-Cancer Activity

The in vitro anti-cancer activity of some selected ester compounds was investigated in vitro in a panel of 10 human cancer cell lines by using a 2D monolayer assay. Results are summarized for compound L4 in Table 3A&B giving the absolute and relative IC50 and IC70 as well as all individual measurement points.

2.8. In Vivo Anti-Cancer Activity

BDF1 male mice 5 weeks old were used in group of 8 animals. Cells were implanted s.c. in a volume of 0.5 ml. The three concentrations 200,100 and 50 mg/kg were given i. Pat day 1 and continued for 9 days. The anticancer activity was estimated according to NCI tumor panel screening method.[23]

2.9. Acute Toxicity Study

The single dose toxicity study of L4 was examined in mice and guinea pigs.

2.10. In Vitro Effect of Mutagenicity

Test strains of Salmonella typhimurium TA1530 and TA1537 were used to test the mutagenic effect of these compounds using concentrations up to a dose of 50 ug/ml.

2.11. Numerical Technique Optimization

We have adopted the Newton-Raphson (N-R) method[25] as the most suitable numerical technique to test our hypothesis about correlation between molecular weight of a chemical compound and its anticancer activity.

The aim of the current study is to investigate whether there is a possible relation between the anticancer activity of the ester compounds obtained both in vitro and in vivo and applying a mathematical model using the optimization method of Newton-Raphson (N-R) as a tool to confirm the anticancer activity obtained on these esters. Up to our knowledge, such technique was not used before and we are the first to use this application in this study.

3. RESULTS AND DISCUSSION

The series of ester compounds showed marked anticancer activity against most of the cell lines used (IC50 ranging from 0.016 - 0.242 ug/ml), however, the most active of these compounds was L4, as can be noticed in Table 4 and Fig.2.

Table 3: In vitro activity of L4 in 10 tumor cell lines.

A. IC values [0.3%/v/v)]

L4 / Cell Line		Exp. no.	Unit	Abs. IC50	Abs. IC70
BXF	RT112	QA1477-P1456452-7	%/v/v	0,012	0,012
CXF	COLO 205	QA1478-P1456096-7	%/v/v	0,013	0,014
GXA	MKN45	QA1479-P1457204-7	%/v/v	0,022	0,028
HNXF	CAL-27	QA1480-P1457405-7	%/v/v	0,017	0,020
LXFA	526	QA1481-P1457434-7	%/v/v	0,023	0,025
LXFL	529	QA1482-P1456819-7	%/v/v	0,016	0,021
MAXF	MDA-MB-231	QA1483-P1457463-7	%/v/v	0,017	0,021
MAXF	SK-BR-3	QA1484-P1456297-7	%/v/v	0,013	0,014
PAXF	1657	QA1485-P1456127-7	%/v/v	0,016	0,019
RXF	SN12C	QA1486-P1457492-7	%/v/v	0,012	0,013

B. Test/Control values [%] at each test concentration

L4 /		Exp.	Test/Control (%) at Drug Concentration [%(v/v)]									
Cell Line		no.	9,5E-06	3,0E-05	9,5E-05	3,0E-04	9,5E-04	3,0E-03	9,5E-03	3,0E-02	9,5E-02	3,0E-01
BXF	RT112	QA1477-P1456452-7	96	97	101	98	99	98	96	0	2	3
CXF	COLO 205	QA1478-P1456096-7	97	98	100	103	103	104	100	6	8	7
GXA	MKN45	QA1479-P1457204-7	97	100	100	100	99	100	97	25	7	7
HNXF	CAL-27	QA1480-P1457405-7	97	98	102	97	94	98	95	7	4	3
LXFA	526	QA1481-P1457434-7	101	100	95	102	103	98	100	12	6	10
LXFL	529	QA1482-P1456819-7	106	102	106	102	100	99	87	17	14	7
MAXF	MDA-MB-231	QA1483-P1457463-7	109	100	113	102	115	101	97	12	4	8
MAXF	SK-BR-3	QA1484-P1456297-7	95	96	97	100	93	97	95	11	12	12
PAXF	1657	QA1485-P1456127-7	99	103	100	104	101	99	94	17	17	15
RXF	SN12C	QA1486-P1457492-7	93	92	93	96	92	92	90	3	5	5

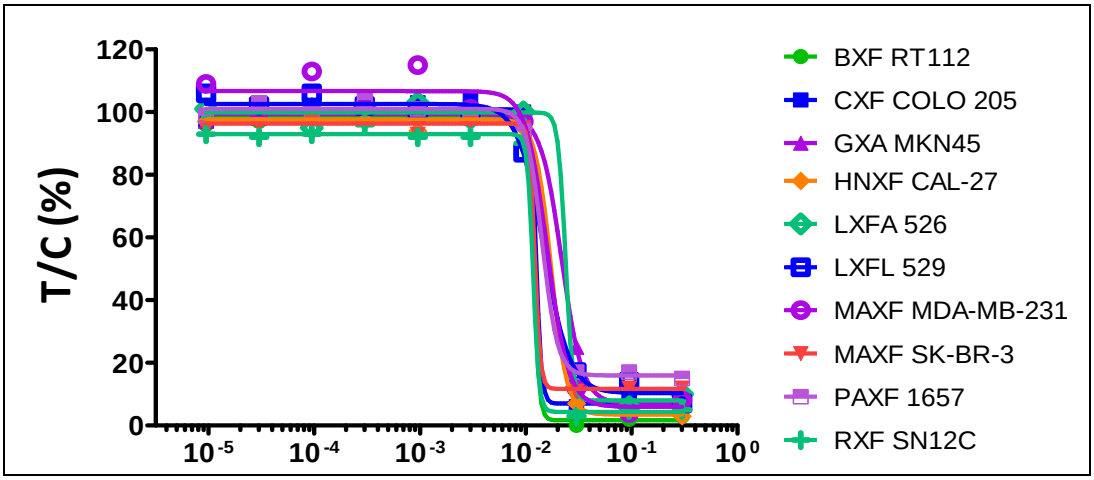


Fig 1: The dose-response curves for L4.

The in vivo anticancer activities of eight compounds were tested in B16-melanoma. The results indicated that only one of these esters (L4) showed relevant activity at doses 200, 100 and 50 mg/kg and the Test/Control (T/C) values were 141 and 141 and 124%, respectively, whereas other compounds showed no in vivo activity in B16- melanoma. The in vitro activity of L4 compound was therefore, confirmed when tested in vivo using B16 melanoma at both 100 and 200 mg/kg.

Further, the acute toxicity study of L4 compound was examined in mice and guinea pigs using more than one route. The LD50 values of this compound was determined to be 550 mg/kg intra peritoneal in mice; 730 mg/kg and 460 mg/ kg in guinea pigs using oral route and intra peritoneal respectively. In addition, the genotoxicity of L4 compound was studied in Salmonella typhimurium TA1530 and TA1537 and it was found that L4 compound is not mutagenic up to a dose of 50 µg/ml.

It is evident from Table 4 that these compounds which showed variations in their molecular weights amongst each other showed different activities to the cancer cell lines used in the present study. For example, compound L6 with molecular weight 175 showed the best activity against mammary MAXF MDA-MB-231 cell line only; while compound L9 with molecular weight 183 showed the best activity against Gastric GXA MKN 45 cell line only. Whereas, compound L4 with a molecular weight of 268 exhibited a potential activity against all the cell lines used.

Table 4: Inhibitory Concentration (IC_{50}) [µg/ml] of compounds with different molecular weights in 10 different tumor cell lines.

	Cell Lines & IC_{50} (µg/ml)												
Compounds	BXF RT112	CXF COLO 205	GXA MKN45	HNXF CAL-27	LXFA 526L	LXFL 529L	MAXF MDA-MB-231	MAXF SK-BR-3	PAXF 1658	RXF SN 12C	**Molecular weights**	**Geo.Mean abs.IC_{50}**	**Geo.Mean abs. IC_{70}**
L6	0.178	0.209	0.274	0.137	0.179	0.137	0.089	0.177	0.173	0.183	175	0.167	0.235
L9	0.3	0.108	0.03	0.254	0.234	0.111	0.18	0.215	0.3	0.232	183	0.211	0,273
L2	0.09	0.118	0.171	0.081		0.12	0.05	0.117	0.098	0.121	198	0.102	0,129
L15	0.034	0.037	0.04	0.021	0.034	0.062	0.014	0.033	0.013	0.035	211	0.022	0.029
L13	0.3	0.3	0.3	0.14	0.3	0.135	0.076	0.156	0.113	0.3	213	0.190	0.296
L18	0.094	0.081	0.131	0.052	0.097	0.038	0.028	0.045	0.044	0.087	239	0.063	0.095
L22	0.08	0.068	0.03	0.126	0.068	0.038	0.032	0.041	0.044	0.099	258	0.071	0.130
L4	0.012	0.014	0.028	0.02	0.025	0.021	0.021	0.014	0.019	0.013	268	0.015	0.018

To analyze the results of the current study and to find out if a correlation exists between the molecular weights and the anticancer activity of any given compound, the Newton-Raphson (N-R) method of optimization[25] is adopted as an optimization tool. This technique, which is up to our knowledge is not used before in a study similar to our research, has been considered as an optimization technique in many engineering oriented researches. For instance, the N-R is used for optimization of molecular mechanic calculation.[26] It is also used for optimization of vibration problems[27], and for partial differential correction of digital image correlation.[28]

We have adopted the N-R method because we assumed that it is the most suitable numerical technique method for optimization. Further, achieving an inhibitory concentration value of 0.00001 µg/ml is the optimal result that all cancer research centers are targeting. So the first step in our assumption was to calculate the optimum molecular weight depending on the N-R technique followed by calculation of the molecular weights for all the tested ester compounds (Table 5). These results are plotted in Fig. 2, where the values of the optimized results are very close to the best molecular weights obtained from the experimental results. The only result that showed a deviation is that of the cell line (PAXF SK-BR-3).

Table 5: Results for 10 tumor cell lines showing the best chemical compounds obtained defined by their molecular weights, and the corresponding optimum molecular weight.

	Tumor Cell Lines									
MW	BXF RT112	CXF COLO 205	GXA MKN45	HNXF CAL-27	LXFA 526L	LXFL 529L	MAXF MDA-MB-231	MAXF SK-BR-3	PAXF SK-BR-3	RXF SN12C
Best Molecular Weight (Experimental)	268	268	268	268	268	268	211	268	211	268
Optimum Molecular Weight (N-R method)	268.02	268.03	269.03	268.05	268.2	268.01	210.1	268.03	268.02	268.04

It is evident from figure 2 that there are marginal differences in the values of the molecular weights of these compounds (Experimental) and those obtained using the optimization tool (Optimized), however, such minor differences are not clearly shown in this figure. Therefore, to elucidate more these differences we considered another scale and plotted the results of Table 5 again as shown in figure 3. In fact the range bounded by the dash lines in figure 2 is considered in figure 3.

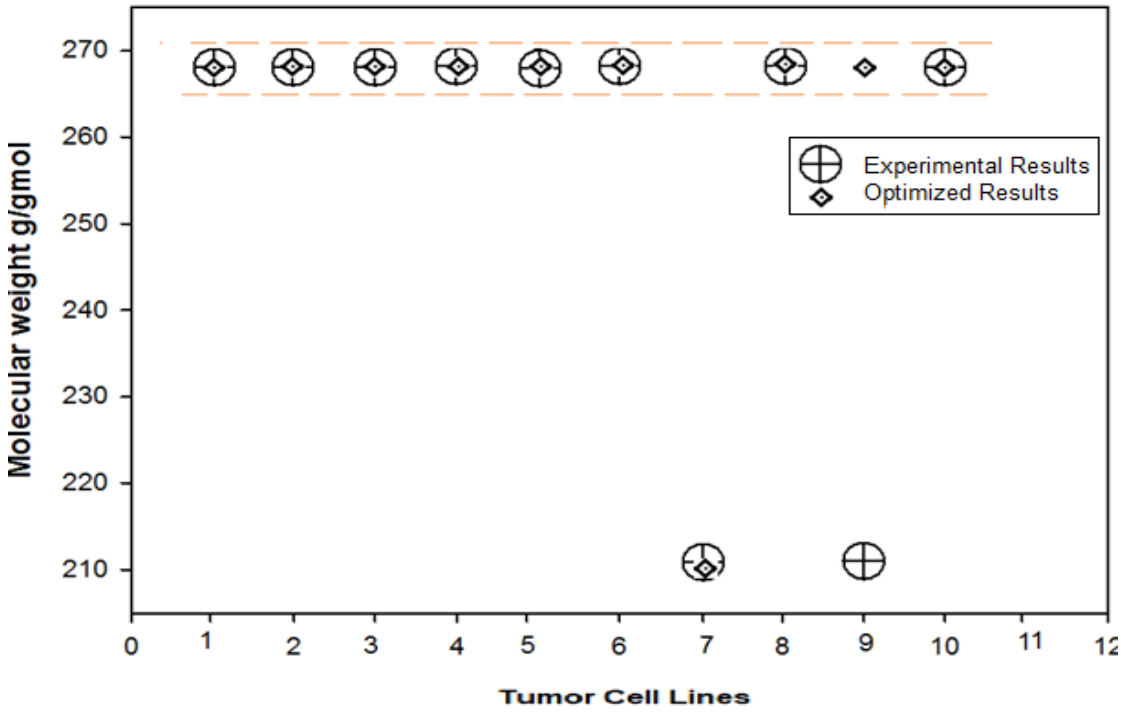


Fig. 2: Results for 10 tumor cell lines and the corresponding optimum molecular weights calculated using a numerical optimization method.

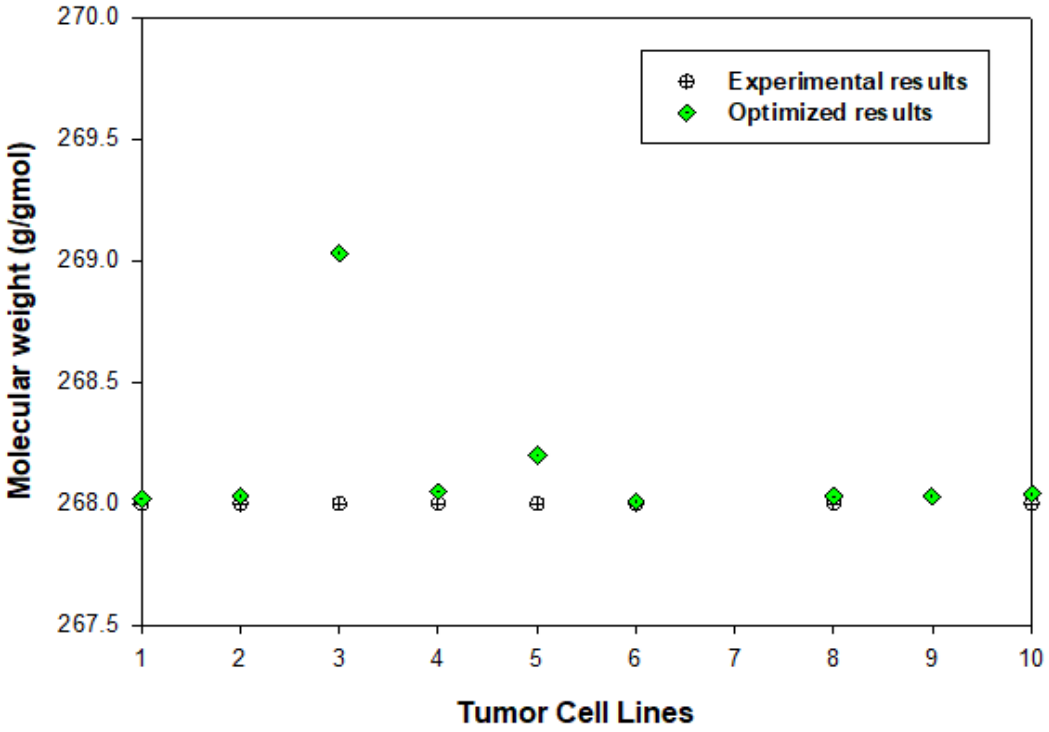


Fig. 3: Results for 10 tumor cell lines and the corresponding numerical results close to the region of optimum molecular weights.

CONCLUSION

We would like to highlight that, from our point of view, the marginal difference in the results obtained from applying the mathematical model with those obtained experimentally is a considerable finding. It is basically demonstrating a consistency and clear correlation between the experimental part represented by the anticancer activity and the results of numerical analysis. Therefore, initially we can postulate that this application can be used as a tool to design new anticancer products. However, additional studies are required to confirm this correlation.

Guarantor
The corresponding author is a guarantor of submission.

Conflict of Interest
Authors declare no conflict of interest.

REFERENCES

1. (Accessed on May 3, 2018). http://www.who.int/mediacentre/factsheets/fs297/en/ .Google Scholar.
2. https:// www.cancer, gov/about-cancer/understanding/statistics (Accessed on May3, 2018). Google Scholar.
3. MS, Kinch, Drug Discover Today, 2014; 19(12): 1831. doi:10.1016/j.drudis.2014.08.007 {PubMed} {Cross Ref}.
4. MV, Blagosklonny, Cell Cycle., 2004; 3(8): 1035 {PubMed}.
5. GC, Winkler, EL, Barle, G.Galati, MV, Kluwe, Reg. Toxicol Pharmacol, 2014; 70(1): 46. doi: 10.1016/j.yrtph.2014.06.012.
6. BA, Baldo, NH, Pham, Cancer Metastasis Rev., 2013; 32(3-4): 723. doi:10.1007/s 10555-013-9447-3{PubMed} (Cross Ref}.
7. K, Masui, B.Gini, J.Wykosky, C, Zanca, PS, Mischel, FB, Furnari, WK, Cavenee, Carcinogenesis, 2013; 34(4): 725. doi:10.1093/carcin/bgt086.(PMC free article){PubMed}(Cross Ref)
8. S, Meyers, A, Baker, Nat Biotechnol, 2001; 19: 727.
9. LJ, Rashan, TAK-Al-Allaf. The 37th IUPAC Congress, Berlin (14-19 August 1999).
10. BJ, Bradbury, J, Baumgold, R, Paek, U, Kammula, J, Zimmet, KA, Jacogbson. J. Med. Chem., 1991; 34(3): 1073.
11. EJ, Trybulski, RH, Mangano, A, Rusinko. J. Med. Chem., 1990; 33(12): 3190.
12. VH, Senthy, DK, Hyslop, Neuropharmacology, 1990; 29(2): 185.
13. JJ, Waite, DJ, Jenden, Pharmacol. Exp. Ther., 1989; 248(1): 111.
14. B, Ringdahl, Eur. J. Pharmacol, 1987; 140(1): 13.
15. R, Amstutz, B, Ringdahl, B, Karlen, M, Roch, DJ, Jenden, J. Med. Chem., 1985; 28(12): 1760.
16. N, Hymowitz, HE, Brezenott, Pharmacol. Biochem. Behav, 1984; 21(1): 109.
17. B, Resul, B, Ringdahl, U, Hacksell, U, Svensson, R, Dahlbom, Acta Pharm. Suec., 1979; 16(4): 225.
18. A, Lindquist, R, Dahlbom, Dent. Assist., 1970; 39(10): 527.
19. AG, Mahsumov, A, Safloev, AF, Trifonova, SH Abdullaev, MA, Korshunov, FN, Bondaruk, USSR Patent; SU 24867, 12.11.1976.
20. AMA, Al-Rawi, AH.KZuthier, FT.Abachi, A.Spectrochimica Acta., 1987; 43A: 1121.
21. B.JL, Neumeyer, UV.Moyer, JA, Richman, FJ, Rosenberg, DG, Teiger. J. Med. Chem., 1967; 10(4): 615.
22. WA, Dengler, J, Schulte, DP, Berger, R, Mertelsmann, HH, Fiebig. Anti-Cancer Drugs, 1995; 6(522).
23. J-H, Zhang, TDY, Chung, KR, Oldenburg. J. Biomol. Screen, 1999; 4(6773).
24. JM.Vanditti, RA, Wasley, J, Plowman. Adv. Pharmacol. Chemother, 1984; 20: 1.
25. JL, Litchfield, F, Willcoxon, J. Pharmacol. Exp. Ther., 1949; 96.
26. The Newton's Raphson Method, http.math.ubc.ca/-ante/math104/newtonmethod.pdf (Accessed3/12/2018).
27. R, Hilderbrandt, Applications of the Newton-Raphson optimization technique to molecular mechanics calculations, Computers and Chemistry, 1977; 1: 179-186.
28. Irvine, Application of the Newton-Raphson Method to Vibration Problems, 2003. http://citeseerx.ist.psu.edu/viewdoc/download?rep=rep1&type=pdf&doi=10.1.1.216.6997
29. H. Bruck, M. Sutton, S.R. McNeill, W.H. Peters, Digital image correlation using Newton-Raphson method of partial differential correction, Experimental Mechanics, January 1989; 29(3): 261-267.