Scientific Research Publishing

Journal of Cancer Therapy, 2016, 7, 675-679
http://www.scirp.org/journal/jct
ISSN Online: 2151-1942
ISSN Print: 2151-1934

Molecular Marker of Tumours

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How to cite this paper: Surma, M. (2016) Molecular Marker of Tumours. *Journal of Cancer Therapy*, **7**, 675-679.
http://dx.doi.org/10.4236/jct.2016.710070

Received: June 17, 2016
Accepted: September 19, 2016
Published: September 22, 2016

Abstract

Molecular structure of the marker of tumour is determined by magneto-optical analysis of blood serum. The marker is the laevorotatory enantiomer of alanine. The cancer status of a subject is described by the number of molecules of the laevorotatory alanine enantiomer ${}^{(-)}\rho$ and the effectiveness of therapy is measured by the number of molecules of the dextrorotatory alanine enantiomer ${}^{(+)}\rho$. The values of ${}^{(-)}\rho$ and ${}^{(+)}\rho$ are determined separately for the patient before and after therapy.

Keywords

Cancer, Molecular Marker, Protein Markers

1. Introduction

Molecular structure of the marker of cancer status has not been known so far. The optically active components of human blood are the free laevorotatory alanine enantiomer of the structure of an electric quadrupole and the dextrorotatory enantiomer of the structure of an electric dipole as well as some optically active protein structures. Protein markers provide information on the cancer disease status but with a large margin of error, the information is often unreliable for evaluation of the actual status of disease development and effectiveness of the therapy applied. As shown in paper [1], reliable information is provided by the magnetic field B^2 induced angle of rotation b^{exp} of the polarisation plane of light passing through the serum from the blood of the patient. The human blood serum in a magnetic field B^2 becomes optically birefringent (Magneto-optical Circular Birefringence). This effect, MOCB, measured by the value of the angle of rotation b^{exp} is an indicator informing about cancer status if the serum is optically laevorotatory ($b^{exp} < 0$). The serum from the blood of patients after effective therapy and from healthy people is dextrorotatory ($b^{exp} > 0$).

2. Experiment and Experimental Data

Protein markers have been dominant in use and in literature, however, it should be realised that the procedures of serum denaturation destroys molecular structures of the optically active enantiomers which also provide information on the cancer status or effectiveness of therapy or lack of cancer. The complexity of protein structures is illustrated by the fact of the presence of 20 amino acids in a human cystatin C [2]. The presence of amino acids in different percent contributions in proteins is well known in literature [3]. Analysis of results of b^{exp} parameter of MOCB marker for about 600 samples of blood serum from subjects diagnosed with cancer at different degrees of the tumour development [4]-[6] permits making diagnosis on the basis of this parameter value. Although the structure of the marker is not known yet, on the basis of analysis of $b^{exp} < 0$ it can be concluded that it contains laevorotatory alanine $Ala^{(-)}$. This result means that in these serum samples the contribution of laevorotatory enantiomers dominates over the content of dextrorotatory ones. The laevorotatory enantiomer of alanine is a molecular marker of tumour. This conclusion is supported by: MOCB data analysis [4]-[6], Follicle Receptor protein marker analysis [7] and literature data (tables 3-1 [3]).

According to literature, the alanine enantiomers make about 7.8% of amino acid residues in the patient blood.

The authors of [7] report results of protein marker determination for typical tumours "selectively expressed on the surface of the blood vessels of a wide range of tumours located in the prostate, breast, colon, pancreas, urinary bladder, kidney, lung, liver, stomach, testis and ovary" to support the indications obtained by the protein marker Follicle Receptor. These data are compared with the independent method based on determination of MOCB marker in blood serum from the patients diagnosed with the same tumours: prostate, breast, lung, ovary and kidney. The comparison between the results obtained by the protein marker and the MOCB marker indicates that the values of both markers are determined by the presence of the laevorotatory enantiomer of alanine $Ala^{(-)}$ in the blood serum of cancer patients. In the blood serum from healthy people and patients after successful therapy the dextrorotatory enantiomer $Ala^{(+)}$ is dominant. Results of MOCB marker for a few chemically pure enantiomers have been reported in [8].

The alanine density is 1.42 g/cm^3, and its molecular mass is $M_e = 89.09$ g/mol. Analysis of magneto-optical measurements of blood serum samples implies that the presence of cancer and advancement of its development are correlated with the content of laevorotatory, the electric quadrupole structure, enantiomer of alanine $Ala^{(-)}$.

3. Results

Comparison of the results of MOCB molecular marker and tPSA protein marker are given in **Tables 1-3**.

MOCB, $b^{exp} < 0$, the measurements for the serum from prostate patients (1, 2, 3, 4) indicates the presence of cancer and tPSA > 4 ng/mL indicates the presence of cancer.

Table 1. Prostate.

$b^{exp} < 0$; (1,2,3,4,5): different cancer patients.					
	$^{(-)}\rho = 1.79 \times 10^{22}\ (-b^{exp})$				
	Mocb		tPSA	tPSA	Mocb/tPSA
	$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ ^{(-)}\rho/mm^3$	ng/mL	$10^3\ g/mm^3$	$10^2\ degT^{-2}g^{-1}$
1	−1.36	0.24	6.70	6.70	−0.20
2	−5.93	1.06	5.45	5.45	−1.08
3	−9.49	1.69	5.14	5.14	−1.84
4	−15.43	2.76	6.09	6.09	−2.53
5	−18.07	3.23	3.40	3.40	−5.31
$b^{exp} > 0$; (1a,2a,3a,4a,5a): different recovered patients after effective therapy.					
	$^{(+)}\rho = 1.79 \times 10^{22}\ (b^{exp})$				
	Mocb		tPSA	tPSA	Mocb/tPSA
	$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-8}\ ^{(+)}\rho/mm^3$	ng/mL	$10^3\ g/mm^3$	$10^2\ degT^{-2}g^{-1}$
1a	1.45	0.26	1.87	1.87	0.77
2a	6.64	1.18	7.00	7.00	0.94
3a	9.56	1.71	2.37	2.37	4.03
4a	14.58	2.61	1.53	1.53	9.52
5a	18.63	3.33	2.26	2.26	8.24

Table 2. Prostate cancer patients and after effective therapy.

1. Patient P (1); Medical diagnose date 12.05.2003.		
Mocb		
$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ ^{(-)}\rho/mm^3$	tPSA/ ng/mL
−5.93	1.06	5.45
After effective therapy RP1 medical diagnosis dated 28.01.2004.		
Mocb		
$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ ^{(+)}\rho/mm^3$	tPSA/ng/mL
8.85	1.55	5.35
2. Patient P (2); Medical diagnose date 12.05.2003.		
Mocb		
$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ ^{(-)}\rho/mm^3$	tPSA/ ng/mL
−2.22	0.39	5.76
After effective therapy RP2 medical diagnosis dated 10.03.2004.		
Mocb		
$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ ^{(+)}\rho/mm^3$	tPSA/ ng/mL
14.58	2.60	8.00
3. Patient P (3); Medical diagnose date 02.06.2003.		
Mocb		
$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ ^{(-)}\rho/mm^3$	tPSA/ ng/mL
−1.36	0.24	6.70
Mocb		
$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ ^{(+)}\rho/mm^3$	tPSA/ ng/mL
18.63	3.33	2.37

For patients (1, 2, 3, 4) the results obtained by the two methods MOCB and protein markers are fully consistent: $b^{exp} < 0$, tPSA > 4 ng/mL.

Disagreement of Mocb with tPSA data: Table 1 (5), $b^{exp} < 0$, tPSA < 4 ng/mL. The value of the ratio MOCB/tPSA decreases. It means that the content of $Ala^{(-)}$ enantiomers residues in the patients (1, 2, 3, 4, 5) serum increases as their data ($b^{exp} < 0$) decreases.

Development of tumour is manifested in increase in the content of $Ala^{(-)}$ in the blood serum of cancer patient and a negative value of MOCB marker, $b^{exp} < 0$. Effectiveness of therapy is manifested as an increase in the content of $Ala^{(+)}$ in the blood serum and an increase in $b^{exp} > 0$, Table 2 Cancer patients: P (1), P (2), P (3); $b^{exp} < 0$, tPSA > 4 ng/mL, agreement of MOCB with tPSA data.

Recovered patient RP3; $b^{exp} > 0$, tPSA < 4 ng/mL, agreement of MOCB with tPSA data. Recovered patients: RP1, RP2; $b^{exp} > 0$, tPSA > 5 ng/mL, disagreement of MOCB with tPSA data.

Agreement of MOCB with tPSA data: Table 3 (2,5,8), $b^{exp} > 0$, tPSA < 4 ng/mL. Table 3 (1, 3, 4, 6, 7), $b^{exp} > 0$. Marker $Ala^{(+)}$dominant in the blood serum, ${}^{(+)}\rho \neq 0$ and increase as increase $b^{exp} > 0$.

4. Discussion

The authors of [7] analyze the presence of Follicle-Stimulating Hormone Receptor in tumour. All the above mentioned tumours contain the protein of the mass of $M_e \approx$ 87,000 Dalton, which is indicated by the glycosylated FSH receptor. The results correspond to those based on MOCB marker, for example for prostate cancer $M_e \approx$ 30,000 Dalton and on the data from [4] [5] monomers occur in tumours in contrast to dimers whose presence is natural in glycosylated FSH receptor. Their presence results, reported in table 3-3 [3], for the multimers forms of protein, are natural in glycosylated FSH receptor. Their presence detected by the MOCB method suggests that the laevorotatory enantiomer of alanine $Ala^{(-)}$ present in the proteins ([3], tables 3-1), is a marker of tumour, irrespective of its localisation in the organism.

The paper presents arguments supporting the use of alanine enantiomers $Ala^{(-)}$ and a molecular markers informing about the presence and state of development of cancer.

Table 3. Data for healthy subjects.

	Mocb	${}^{(+)}\rho = 1.79 \times 10^{22} b^{exp}$	tPSA
	$10^5\ b^{exp}/degT^{-2}mm^{-1}$	$10^{-18}\ {}^{(+)}\rho/mm^3$	ng/mL
1	1.03	0.18	16.60
2	4.30	0.73	1.48
3	6.64	1.19	7.00
4	8.51	1.52	17.90
5	14.58	2.61	1.53
6	14.63	2.62	8.00
7	18.63	3.33	7.56
8	31.59	5.65	1.18

Alanine enantiomers $Ala^{(-)}$ and $Ala^{(+)}$ are present in the protein markers used in clinical treatment. The agreement and disagreements of MOCB marker and tPSA marker indications are given in Tables 1-3.

Electric quadrupole moment and magnetic dipole moment were measured for optically inactive structures of CH_3Cl [9] and CO_2 [10]. Theoretical analysis of B^2 field induced magneto-optical birefringence in optically active media is given in [11].

Analysis of the results presented in this paper indicates potential of a new method for diagnostics of cancer changes based on determination of the content of optically active alanine enantiomers $Ala^{(-)}$ and $Ala^{(+)}$ in the blood. The measurement of the content of the laevorotatory enantiomer $Ala^{(-)}$ provides reliable information on the presence of cancer changes. The effectiveness cancer therapy can be measured by the ratio of $Ala^{(-)}$/$Ala^{(+)}$.

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