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INFORMATION DISCLOSURE STATEMENT BY APPLICANT

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Sheet 1 of 3

Complete if Known	
Application Number	13/456,720
Filing Date	April 26, 2012
First Named Inventor	GUPTA
Group Art Unit	1629
Examiner Name	ANDERSON, James D.
Attorney Docket Number	FR2009/121 - US - CNT

U.S. PATENT DOCUMENTS

Examiner Initials*	Cite No.[1]	U.S. Patent Document Number	Kind Code[2] (if known)	Name of Patentee or Applicant of Cited Document	Date of Publication of Cited Document MM-DD-YYYY	Pages, Columns, Lines, Where Relevant Passages or Relevant Figures Appear
		5,847,170		BOUCHARD et al.	12-08-1998	
		7,241,907		DIDIER, et al.	07-10-2007	
		6,372,780		BOUCHARD, et al.	04-16-2002	
		6,331,635		BOUCHARD, et al.	12-18-2001	
		6,387,946		BOUCHARD, et al.	05-14-2002	
		5,438,072		BOBEE, et al.	08-01-1995	
		2002/0038038		BOUCHARD, et al.	03-28-2002	

FOREIGN PATENT DOCUMENTS

Examiner Initials*	Cite No.[1]	Foreign Patent Document Office[3]	Number[4]	Kind Code[5] (if known)	Name of Patentee or Applicant of Cited Document	Date of Publication of Cited Document MM-DD-YYYY	Pages, Columns, Lines, Where Relevant Passages or Relevant Figures Appear	T[6]
		WO	96/30355		BOUCHARD, et al.	10-03-1996		
		EP	0817779		BOUCHARD, et al.	01-05-2000		
		WO	2005/028462		DIDIER, et al.	03-31-2005		
		EP	2177630		CHAUCHEREAU et al.	04-21-2010		

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Sheet 2 of 3

Complete if Known	
Application Number	13/456,720
Filing Date	April 26, 2012
First Named Inventor	GUPTA
Group Art Unit	1629
Examiner Name	ANDERSON, James D.
Attorney Docket Number	FR2009/121 - US - CNT

OTHER PRIOR ART -- NON PATENT LITERATURE DOCUMENTS

Examiner Initials*	Cite No.[1]	Include name of the author (in CAPITAL LETTERS), title of the article (when appropriate), title of the item (book, magazine, journal, serial, symposium, catalog, etc.), date, page(s), volume-issue number(s), publisher, city and/or country where published.	T[2]
		PARKIN, et al., Global Cancer Statistics, (2002), CA Cancer J. Clin., (2005), Vol. 55, pp. 74-108	
		HELLERSTEDT, et al., The Current State of Hormonal Therapy for Prostate Cancer, CA Cancer J. Clin.,(2002), Vol 52, pp.154-179	
		HORWITZ et al., External Beam Radiation Therapy for Prostate Cancer, CA Cancer J. Clin., (2000), Vol. 50, pp. 349-375	
		PIENTA et al., Advances in Prostate Cancer Chemotherapy: A New Era Begins, CA Cancer J. Clin, (2005), Vol. 55, pp. 300-318,	
		CABRAL, Factors Determining Cellular Mechanisms of Resistance to Antimitotic Drugs, Drug Resistance Updates, (2001), Vol. 4 No. 1, pp. 3-8	
		DUMONTET, et al., Mechanisms of Action of and Resistance to Antitubulin Agents: Microtubule Dynamics, Drug Transport and Cell Death, J. Clin. Onc., (1999), Vol. 17, No. 3, pp. 1061-1070	
		HALEBLIAN, et al., Characterization of Habits and Crystalline Modification of Solids and Their Pharmaceutical Applications, J. Pharm. Sci., (1975), pp. 1269-1288	
		MELZACK, The McGill Pain Questionnaire: Major Properties And Scoring Methods, Pain, Vol. 1, (1975), pp. 277-299	
		BERRY, et al., Quality of Life and Pain in Advanced Stage Prostate Cancer: Results of a Southwest Oncology Group Randomized Trial Comparing Docetaxel and Estramustine to Mitoxantrone and Prednisone, Journal of Clinical Oncology, (2006), Vol. 24, No. 18, pp. 2828-2835	
		FIZAZ, et al., New agents in Metastatic Prostate Cancer, Eur. J. Cancer. 2009, Vol. 45, Supp. 1., pp. 379-380	

Examiner Signature		Date Considered	

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INFORMATION DISCLOSURE STATEMENT BY APPLICANT

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Sheet 3 of 3

Complete if Known	
Application Number	13/456,720
Filing Date	April 26, 2012
First Named Inventor	GUPTA
Group Art Unit	1629
Examiner Name	ANDERSON, James D.
Attorney Docket Number	FR2009/121 - US - CNT

OTHER PRIOR ART -- NON PATENT LITERATURE DOCUMENTS

Examiner Initials*	Cite No.[1]	Include name of the author (in CAPITAL LETTERS), title of the article (when appropriate), title of the item (book, magazine, journal, serial, symposium, catalog, etc.), date, page(s), volume-issue number(s), publisher, city and/or country where published.	T[2]
		http://clinicaltrials.gov/archive/NCT00417079/2006_12_28, View of NCT00417079 on 2006_12_28 - XRP6258 Plus Predinisone Compared to Miitoxantrone Plus Predisone in Hormaone Refractory Metastatic Prostate Cancer (TROPIC). Retrieved on 02/08/2012	
		de BONO, et al., Prednisone Plus Cabazitaxel or Mitoxantrone for Metastatic Castration-Resistant Prostate Cancer Progressing after Docetaxel Treatment: a Randomised Open-Label Trial, Lancet, Vol. 376, No. 9747, (2010), pp. 1147-1154	
		GALSKY, et al., Cabazitaxel, Nature Reviews- Drug Discovery, Vol. 9, (2010), pp. 677-678	
		International Search Report for WO2011/051894 dated May 5, 2011	
		Jevtana Prescribing Information, pp.1-25, June 17, 2010	

Examiner Signature	/James Anderson/	Date Considered	09/10/2013

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Amendment Pursuant to 37 C.F.R. § 1.121

In the Specification:

Please replace the paragraph beginning at page 7, line 1 of the specification with the following rewritten paragraph:

Cabazitaxel may be administered in base form (cf. above formula), or in the form of a hydrate. It may also be a solvate, i.e. a molecular complex characterized by the incorporation of the crystallization solvent into the crystal of the molecule of the active principle (see in this respect page 1276 of *J. Pharm. Sci.* 1975, *64*(8), 1269-1288). In particular, it may be an acetone solvate, and, more particularly, may be the solvate described in WO **2005/028462** ~~**2005/02846**~~. It may be an acetone solvate of cabazitaxel containing between 5% and 8% and preferably between 5% and 7% by weight of acetone (% means content of acetone/content of acetone+cabazitaxel × 100). An average value of the acetone content is 7%, which approximately represents the acetone stoichiometry, which is 6.5% for a solvate containing one molecule of acetone. The procedure described below allows the preparation of an acetone solvate of cabazitaxel:

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L1            1 CABAZITAXEL/CN

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ED   Entered STN:  14 Nov 1996
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     dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-
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cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, 12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, [2aR-[2aα,4β,4aβ,6β,9α(αR*,.beta.S*),11α,12α,12aα,12bα]]-
OTHER NAMES:
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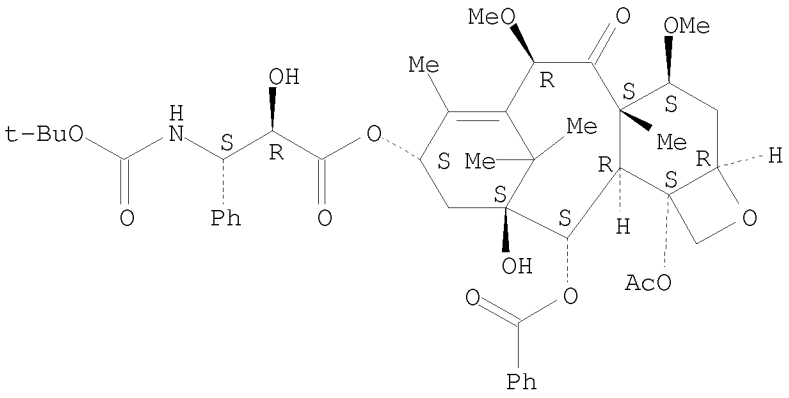
CI COM
SR CA
LC STN Files: ADISINSIGHT, BIOSIS, CA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, EMBASE, IMSPATENTS, IMSRESEARCH, IPA, TOXCENTER, USAN, USPAT2, USPATFULL

Absolute stereochemistry. Rotation (-).



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through 1968.  These records have been re-indexed to match current
BIOSIS indexing.

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             1 183133-96-2/BI
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=> s l8 and py<2011
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L9          10 L8 AND PY<2011

=> file medline; del sel y
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   1.05      28.76

FILE 'MEDLINE' ENTERED AT 10:59:12 ON 10 SEP 2013

 FILE LAST UPDATED: 7 Sep 2013 (20130907/UP).  FILE COVERS 1946 TO DATE.

 MEDLINE(R) is a registered trademark of the U.S. National Library of
 Medicine (NLM).

 The 2013 MeSH Thesaurus is now available.

 The 2013 Medline reload was completed on January 26, 2013.
 See HELP RLOAD for details.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.
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See HELP RANGE before carrying out any RANGE search.

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                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.45      29.21

FILE 'EMBASE' ENTERED AT 10:59:26 ON 10 SEP 2013
Copyright (c) 2013 Elsevier B.V. All rights reserved.

 FILE COVERS: Embase-originated material 1947 to 10 Sep 2013 (20130910/ED)
              Unique MEDLINE content 1948 to present
              Emtree thesaurus last updated September 1, 2013

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

 Effective July 28, 2013, the content in Embase Alert (EMBAL)
 is strictly complementary to that in Embase (EMBASE).  EMBAL
 contains, at any given time, approximately 100,000 novel
 records not yet available in Embase.  Search both databases
 for the most timely and comprehensive results.  See NEWS for
 details.



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             0 1438897-79-0/BI
             0 1443430-50-9/BI
           460 183133-96-2/BI
             0 890654-44-1/BI
        849140 ?NEOPLAS?
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       2252686 ?TUMO?
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L12        678 L4 AND (?NEOPLAS? OR ?CANCER? OR ?TUMO? OR ONCO? OR CARCIN? OR
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L13         80 L12 AND PY<2011

=> s l13 and prostate
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          3880 PROSTATES
        216974 PROSTATE
                  (PROSTATE OR PROSTATES)
L14         59 L13 AND PROSTATE

=> d his

     (FILE 'HOME' ENTERED AT 10:55:45 ON 10 SEP 2013)

     FILE 'REGISTRY' ENTERED AT 10:55:54 ON 10 SEP 2013
L1            1 S CABAZITAXEL/CN
L2           13 S 183133-96-2/CRN
L3           14 S L1 OR L2
                  SELECT CHEM L3 1-
L4                QUE E1-E20

     FILE 'HCAPLUS' ENTERED AT 10:56:37 ON 10 SEP 2013
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L5          245 S L4(L)((PAC OR PKT OR DMA OR BAC OR THU)/RL OR (TREAT? OR CURE
L6          206 S L5 AND (?NEOPLAS? OR ?CANCER? OR ?TUMO? OR ONCO? OR CARCIN? O
L7           42 S L6 AND (AD<20101020 OR PD<20101020 OR PRD<20101020)
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L8          125 S L4 AND (?NEOPLAS? OR ?CANCER? OR ?TUMO? OR ONCO? OR CARCIN? O
L9           10 S L8 AND PY<2011

     FILE 'MEDLINE' ENTERED AT 10:59:12 ON 10 SEP 2013
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L10         236 S L4 AND (?NEOPLAS? OR ?CANCER? OR ?TUMO? OR ONCO? OR CARCIN? O
L11          22 S L10 AND PY<2011

     FILE 'EMBASE' ENTERED AT 10:59:26 ON 10 SEP 2013
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L12         678 S L4 AND (?NEOPLAS? OR ?CANCER? OR ?TUMO? OR ONCO? OR CARCIN? O
L13          80 S L12 AND PY<2011
L14          59 S L13 AND PROSTATE

=> dup rem l7 l9 l11 l14
COST IN U.S. DOLLARS                            SINCE FILE      TOTAL
                                                     ENTRY    SESSION
FULL ESTIMATED COST                                   2.17      31.38

FILE 'HCAPLUS' ENTERED AT 11:00:06 ON 10 SEP 2013
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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FILE 'BIOSIS' ENTERED AT 11:00:06 ON 10 SEP 2013
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FILE 'MEDLINE' ENTERED AT 11:00:06 ON 10 SEP 2013

FILE 'EMBASE' ENTERED AT 11:00:06 ON 10 SEP 2013
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PROCESSING COMPLETED FOR L7
PROCESSING COMPLETED FOR L9
PROCESSING COMPLETED FOR L11
PROCESSING COMPLETED FOR L14
L15         101 DUP REM L7 L9 L11 L14 (32 DUPLICATES REMOVED)

=> d l15 1-101 ibib abs hitstr

L15  ANSWER 1 OF 101  HCAPLUS  COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER:        2013:277129  HCAPLUS
DOCUMENT NUMBER:         158:331494
TITLE:                   Preparation of cyclodextrin-based polymers for
                         therapeutic delivery as antitumor agents
INVENTOR(S):             Crawford, Thomas C.; Fetzer, Oliver S.; Reiter,
                         Lawrence Alan; Wolfgang, Marc
PATENT ASSIGNEE(S):      Cerulean Pharma Inc., USA
SOURCE:                  PCT Int. Appl., 386pp.
                         CODEN: PIXXD2
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  3
PATENT INFORMATION:

     PATENT NO.          KIND  DATE        APPLICATION NO.          DATE
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     WO 2013025337        A1   20130221    WO 2012-US48865          20120730
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             BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE,
             EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS,
             JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY,
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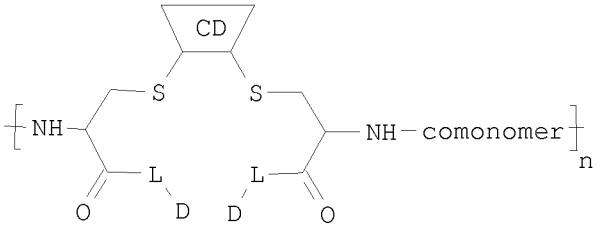
MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM,
PE, PG, PH, PL, PT, QA, RO, RS, RU, RW, SC, SD, SE, SG, SK, SL,
SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC,
VN, ZA, ZM, ZW
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR,
HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS,
SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD,
SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, RU, TJ, TM

US 20120058971 A1 20120308 US 2011-13208703 20110812 <--
PRIORITY APPLN. INFO.: US 2011-13208703 A 20110812
US 2009-61263749 P 20091123 <--
US 2010-61391922 P 20101011 <--
US 2010-953390 A2 20101123

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

GI



I

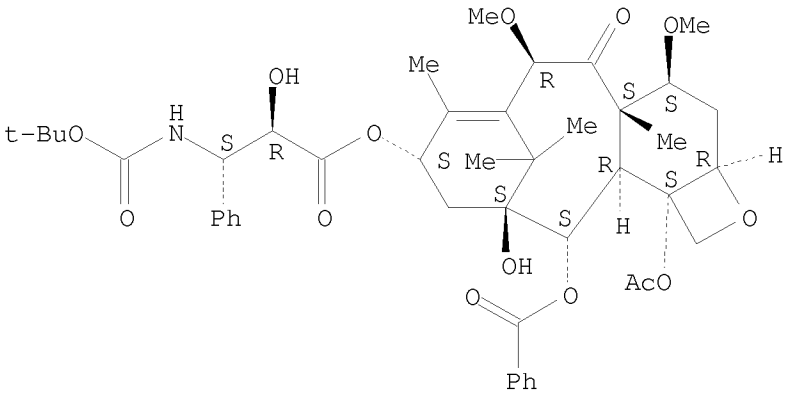
AB Methods and compns. relating to cyclodextrin-based polymers (CDP)-taxane conjugates I, wherein CD ring is cyclodextrin derivative; each L is independently a linker or absent; each D is independently a taxane, OH; each comonomer comprises polyethylene glycol; n is at least 4, provided that the CDP-taxane conjugate comprises at least one taxane selected from docetaxel, larotaxel, and **cabazitaxel**, to a subject in an amount effective to **treat** the **cancer**, are described herein. A method of selecting a subject having **cancer**, for **treating** the subject with a CDP-taxane conjugate was claimed, the method comprising: determining if a subject with **cancer** is at risk for or has diarrhea, proliferative disorder, fluid retention, hepatic impairment, severe neuropathy, from **treatment** with an **anticancer** agent with a CDP-taxane conjugate.

IT **183133-96-2**
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of cyclodextrin-based polymers for **therapeutic** delivery as **antitumor** agents)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 2 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2012:568662 HCAPLUS
DOCUMENT NUMBER: 156:534669
TITLE: Polymer-agent conjugates, particles, compositions, and related methods of use
INVENTOR(S): Crawford, Thomas C.; Eliasof, Scott; Gangal, Geeti; Ng, Pei-Sze; Reiter, Lawrence Alan; Zhang, Jerry
PATENT ASSIGNEE(S): Cerulean Pharma Inc., USA
SOURCE: PCT Int. Appl., 654 pp., Cont. of U.S. Ser. No. 72,297.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2012050899	A1	20120419	WO 2011-US53716	20110928 <--
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RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
US 20110189092	A1	20110804	US 2010-894040	20100929 <--
US 20110268658	A1	20111103	US 2011-13004838	20110111 <--
US 20110262490	A1	20111027	US 2011-13072297	20110325 <--

PRIORITY APPLN. INFO.:

US 2010-894040	A1	20100929 <--
US 2011-13004838	A1	20110111
US 2011-13072297	A1	20110325
US 2009-61164720	P	20090330 <--
US 2009-61164722	P	20090330 <--
US 2009-61164725	P	20090330 <--
US 2009-61164728	P	20090330 <--

US 2009-61164731	P	20090330	<--
US 2009-61164734	P	20090330	<--
US 2009-61262993	P	20091120	<--
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WO 2010-US28770	A1	20100326	<--
US 2010-748637	A2	20100329	<--

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

OTHER SOURCE(S): CASREACT 156:534669

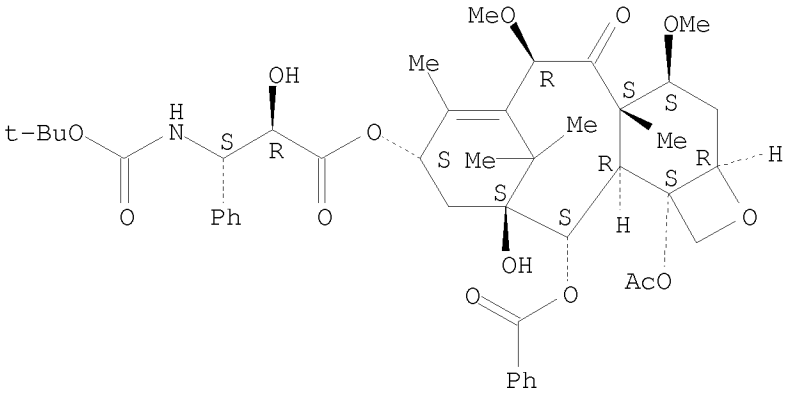
AB Described herein are polymer-agent conjugates and particles, which can be used, for example, in the treatment of **cancer.** Also described herein are mixts., compns. and dosage forms containing the particles, methods of using the particles (e.g., to treat a disorder), kits including the polymer-agent conjugates and particles, methods of making the polymer-agent conjugates and particles, methods of storing the particles and methods of analyzing the particles.

IT **183133-96-2, Cabazitaxel**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(polymer-agent conjugates, particles, compns., and related methods of use)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 3 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN

ACCESSION NUMBER: 2012:265414 HCAPLUS

DOCUMENT NUMBER: 156:357951

TITLE: Circulating biomarkers for disease

INVENTOR(S): Spetzler, David; Holterman, Daniel; Pawlowski, Traci; Kuslich, Christine

PATENT ASSIGNEE(S): Caris Life Sciences Luxembourg Holdings, Luxembourg

SOURCE: PCT Int. Appl., 639pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English

FAMILY ACC. NUM. COUNT: 13

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2012024543	A1	20120223	WO 2011-US48327	20110818 <--
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AU 2011291599	A1	20130307	AU 2011-291599	20110818 <--
EP 2606353	A1	20130626	EP 2011-818814	20110818 <--
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US 2010-61374951	P	20100818 <--
US 2010-61379670	P	20100902 <--
US 2010-61381305	P	20100909 <--
US 2010-61383305	P	20100915 <--
US 2010-61391504	P	20101008 <--
US 2010-61393823	P	20101015 <--
US 2010-61411890	P	20101109
US 2010-61413377	P	20101112
US 2010-61414870	P	20101117
US 2010-61416560	P	20101123
US 2010-61421851	P	20101210
US 2010-61423557	P	20101215
US 2010-61428196	P	20101229
US 2011-61471417	P	20110404
WO 2011-US48327	W	20110818

AB Biomarkers can be assessed for diagnostic, therapy-related or prognostic methods to identify phenotypes, such as a condition or disease, or the stage or progression of a disease. Circulating biomarkers from a bodily fluid can be used in profiling of physiol. states or determining phenotypes. These include nucleic acids, protein, and circulating structures such as vesicles. Biomarkers can be used for theranostic purposes to select candidate treatment regimens for diseases, conditions, disease stages, and stages of a condition, and can also be used to determine treatment efficacy. The biomarkers can be circulating biomarkers, including vesicles and microRNA. Various anti-DLL4 antibodies were tethered to beads and used to capture vesicles in blood samples from subjects with breast **cancer**, colorectal **cancer** or normal subjects. The bead captured vesicles were detected with fluorescently-labeled antibodies against the tetraspanins CD9, CD63, and CD81. The median fluorescence intensity of the captured and labeled vesicles was measured using laser detection. There were noticeable differences both among normals and breast **cancer** plasma as well as between the avs. of normals and **cancer** patients.

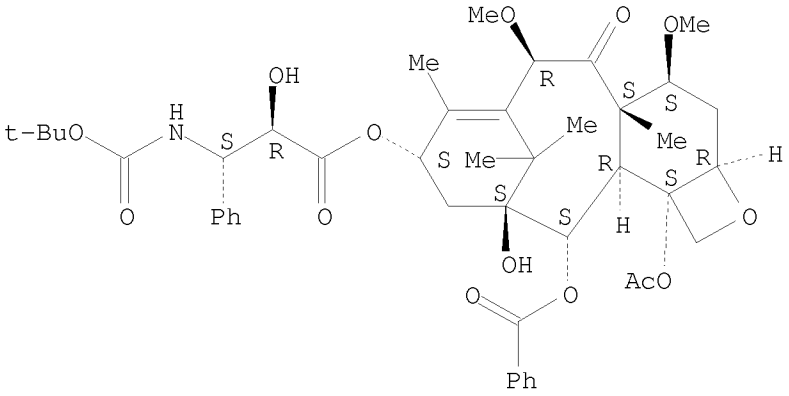
IT **183133-96-2, Cabazitaxel**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); **USES (Uses)**
(DLL4, vesicle, and microRNA circulating biomarkers for disease diagnosis, **therapy**, prognosis, or theranostics)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-

(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).

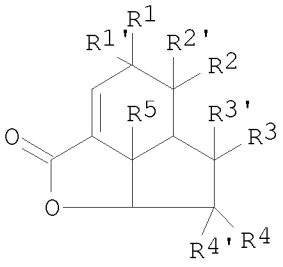


OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

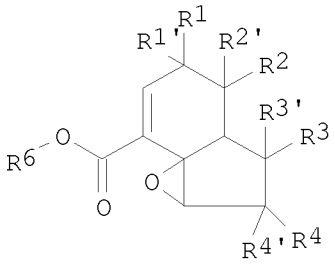
L15 ANSWER 4 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2012:126578 HCAPLUS
DOCUMENT NUMBER: 156:203034
TITLE: Preparation of indeno oxirenes and tricyclic lactones for treatment of **cancer** and other diseases via inhibition of IL-6/STAT or PI3K/NF-κB signaling
INVENTOR(S): Gidloef, Ritha; Johansson, Martin; Sterner, Olov; Munoz, Eduardo
PATENT ASSIGNEE(S): Partners foer Utvecklingsinvesteringar Inom Life Sciences, P.U.L.S. AB, Swed.
SOURCE: PCT Int. Appl., 64pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2012010555	A1	20120126	WO 2011-EP62243	20110718 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CN 103097368	A	20130508	CN 2011-80035500	20110718 <--
EP 2595972	A1	20130529	EP 2011-739028	20110718 <--

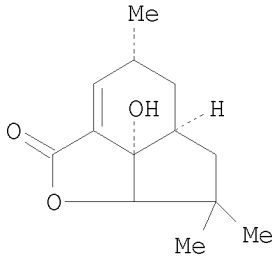
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR
JP 2013531051 T 20130801 JP 2013-520108 20110718 <--
PRIORITY APPLN. INFO.: SE 2010-50815 A 20100719 <--
WO 2011-EP62243 W 20110718
OTHER SOURCE(S): CASREACT 156:203034; MARPAT 156:203034
GI



I



II



III

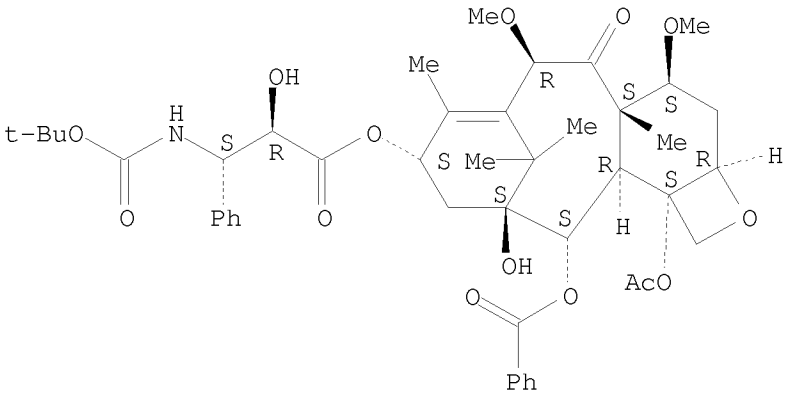
AB The present invention discloses novel compds. (I and II, wherein R1–R4 and R1'–R4' are independently H, C1–5 alkyl, C3–8 carbocycle, etc.; R5 is OH, OC1–5 fluoroalkyl, etc.; R6 is H, C1–5 alkyl, C1–5 fluoroalkyl, and C3–8 carbocycle) useful for the inhibition of IL-6/STAT signaling and/or PI3K/NF-κB signaling in the treatment of associated diseases or conditions, e.g. **cancer**. A pharmaceutical composition comprising such novel compds., its use and a method thereof, is also disclosed. Synthetic procedures for preparing I are exemplified. Example compound III was prepared from the corresponding epoxide, which in turn was prepared from the corresponding tetrahydroindene-4-carboxylate. III had IC50 values of 2.10 and 2.66 μM in STAT3 and NF-κB inhibition assays, resp. III was also tested in a human prostate **cancer** cell proliferation assay.

IT **183133-96-2, Cabazitaxel**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**codrug**; preparation of indeno oxirenes and tricyclic lactones for **treatment** of **cancer** and other diseases via inhibition of IL-6/STAT or PI3K/NF-κB signaling)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 5 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2012:56068 HCAPLUS
DOCUMENT NUMBER: 156:167613
TITLE: Preparation of conjugates of hydroxyalkyl starch with **antitumor** agents
INVENTOR(S): Knoller, Helmut; Heckmann, Dominik; Hacket, Frank; Zander, Norbert; Nocken, Frank
PATENT ASSIGNEE(S): Fresenius Kabi Deutschland GmbH, Germany
SOURCE: PCT Int. Appl., 360pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2012004005	A1	20120112	WO 2011-EP3458	20110711 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 2590677	A1	20130515	EP 2011-731283	20110711 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR				
US 20130184455	A1	20130718	US 2013-13809081	20130327 <--
PRIORITY APPLN. INFO.:			EP 2010-7108 A	20100709 <--
			US 2010-61363112 P	20100709 <--
			WO 2011-EP3458 W	20110711

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): CASREACT 156:167613

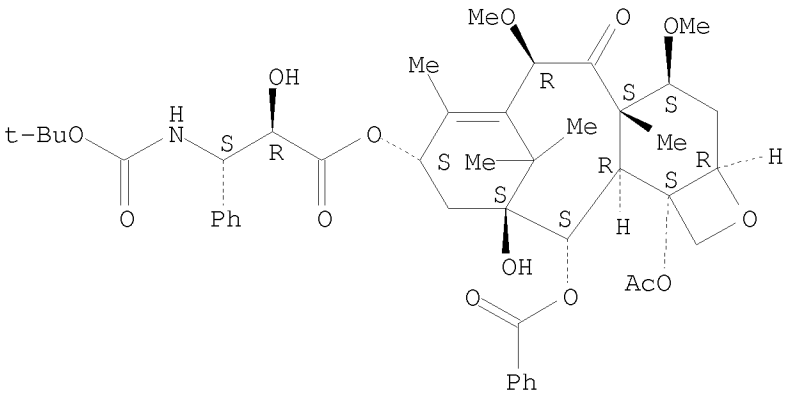
AB The present invention relates to a hydroxyalkyl starch conjugate and a method for preparing it, the hydroxyalkyl starch conjugate comprising a hydroxyalkyl starch derivative and an **antitumor** agent, the **antitumor** agent comprising at least one secondary hydroxyl group, wherein the hydroxyalkyl starch is linked via the secondary hydroxyl group to the **antitumor** agent. The conjugate according to the present invention has a structure according to the following formula, HAS'(-L-M)n wherein M is a residue of the cytotoxic agent, L is a linking moiety, HAS' is the residue of the hydroxyalkyl starch derivative, and n ≥1, and wherein the hydroxyalkyl starch derivative has a mean mol. weight above the renal threshold. A docetaxel derivative was conjugated with hydroxyethyl starch to give a compound and the administration of this conjugate allowed for a more efficient reduction of **tumor** size and/or was less toxic (as indicated by the body weight change) than the administration of non-conjugated docetaxel.

IT **183133-96-2DP, Cabazitaxel**, conjugates with HES derivs.
RL: **PAC (Pharmacological activity)**; SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of conjugates comprising hydroxyalkyl starch and **antitumor** agent)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

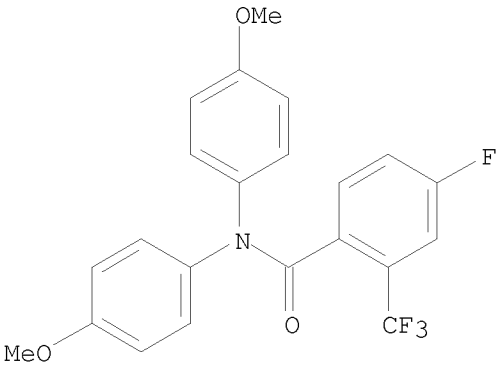
L15 ANSWER 6 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2012:467402 HCAPLUS
DOCUMENT NUMBER: 156:412029
TITLE: Estrogen receptor ligands and methods of use thereof
INVENTOR(S): Dalton, James T.; Steiner, Mitchell S.; Morton, Ronald A.
PATENT ASSIGNEE(S): GTx, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 69pp., Cont.-in-part of PCT/US2010/025032.
CODEN: USXXCO
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 9
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20120077845	A1	20120329	US 2011-13215679	20110823 <--
JP 2008156239	A	20080710	JP 2006-343474	20061220 <--
WO 2010096801	A1	20100826	WO 2010-US25032	20100223 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2011226856	A1	20111013	AU 2011-226856	20110923 <--
AU 2011226856	B2	20120927		
WO 2013043304	A1	20130328	WO 2012-US52141	20120823
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BN, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, QA, RO, RS, RU, RW, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, RU, TJ, TM				
AU 2012312902	A1	20130523	AU 2012-312902	20120823

PRIORITY APPLN. INFO.:	US 2009-61154707	P	20090223 <--
	US 2009-61168983	P	20090414 <--
	US 2009-61261669	P	20091116 <--
	WO 2010-US25032	A2	20100223 <--
	US 2010-61380113	P	20100903 <--
	AU 2006-318400	A3	20061128 <--
	US 2006-604897	T0	20061128 <--
	US 2011-13215679	A	20110823
	WO 2012-US52141	W	20120823

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): CASREACT 156:412029; MARPAT 156:412029
GI



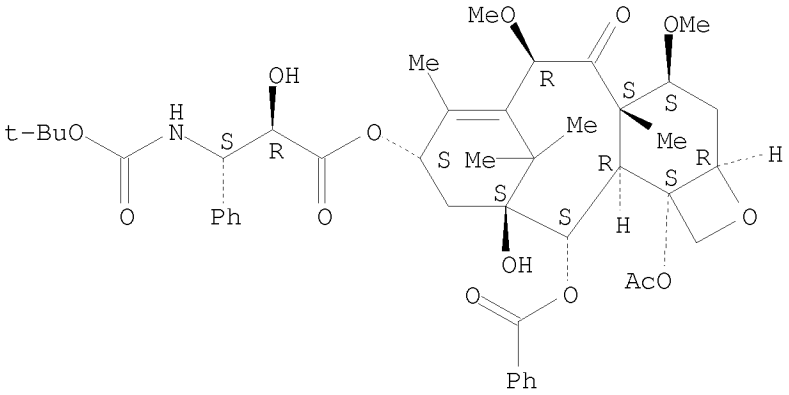
AB The present invention relates to methods for reducing testosterone levels by reduction of LH or independent of LH levels in a male subject and methods of treating, suppressing, reducing the incidence, reducing the severity, or inhibiting prostate **cancer**, advanced prostate **cancer**, and castration-resistant prostate **cancer** (CRPC) and palliative treatment of prostate **cancer**, advanced prostate **cancer** and castration-resistant prostate **cancer** (CRPC). The compds. of this invention suppress free or total testosterone levels to castrate levels which may be used to treat prostate **cancer**, advanced prostate **cancer**, and CRPC without causing bone loss, decreased bone mineral d., increased risk of bone fractures, increased body fat, hot flashes and/or gynecomastia.

IT **183133-96-2, Cabazitaxel**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**treatment** of castration-resistant prostate **cancer** that continues to progress or worsen despite continued **treatment** with addnl. drugs)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 7 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2012:346030 HCAPLUS

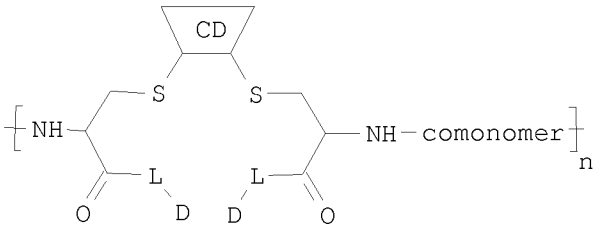
DOCUMENT NUMBER: 156:337543
TITLE: Preparation of cyclodextrin-based polymers for therapeutic delivery as **antitumor** agents
INVENTOR(S): Crawford, Thomas C.; Fetzer, Oliver S.; Reiter, Lawrence Alan; Wolfgang, Marc
PATENT ASSIGNEE(S): Cerulean Pharma Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 181pp., Cont.-in-part of U.S. Ser. No. 953,390.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20120058971	A1	20120308	US 2011-13208703	20110812 <--
US 20110237540	A1	20110929	US 2010-953390	20101123 <--
US 20120225825	A1	20120906	US 2012-13441477	20120406 <--
WO 2013025337	A1	20130221	WO 2012-US48865	20120730

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BN, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, QA, RO, RS, RU, RW, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, RU, TJ, TM

PRIORITY APPLN. INFO.:
US 2009-61263749 P 20091123 <--
US 2010-61391922 P 20101011 <--
US 2010-953390 A2 20101123
US 2011-13208703 A1 20110812

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
GI



I

AB Methods and compns. relating to cyclodextrin-based polymers (CDP)-taxane conjugates I, wherein CD ring is cyclodextrin derivative; each L is independently a linker or absent; each D is independently a taxane, OH; each comonomer comprises polyethylene glycol; n is at least 4, provided that the CDP-taxane conjugate comprises at least one taxane selected from docetaxel, larotaxel, and **cabazitaxel**, to a subject in an amount effective to **treat** the **cancer**, are described herein. A method of selecting a subject having **cancer**, for **treating** the subject with a CDP-taxane conjugate was claimed, the method comprising: determining if a subject with **cancer** is at risk for or has diarrhea, proliferative disorder, fluid

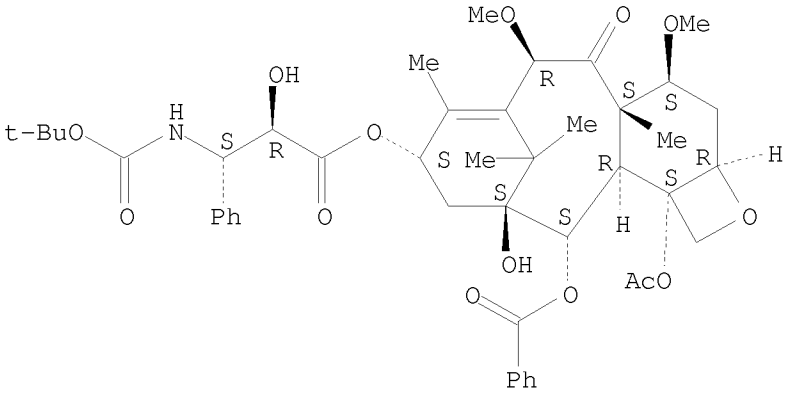
retention, hepatic impairment, severe neuropathy, from **treatment** with an **anticancer** agent with a CDP-taxane conjugate.

IT **183133-96-2**
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of cyclodextrin-based polymers for **therapeutic** delivery as **antitumor** agents)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 8 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:1506136 HCAPLUS
DOCUMENT NUMBER: 156:11747
TITLE: Compositions comprising a CDP-therapeutic agent conjugates and methods for treatment of autoimmune and other diseases
INVENTOR(S): Eliasof, Scott D.
PATENT ASSIGNEE(S): Cerulean Pharma Inc., USA
SOURCE: PCT Int. Appl., 528pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2011146638	A1	20111124	WO 2011-US37025	20110518 <--

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL,

SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

CA 2799202	A1	20111124	CA 2011-2799202		20110518 <--
AU 2011255647	A1	20121115	AU 2011-255647		20110518 <--
IL 222800	A	20121231	IL 2011-222800		20110518 <--
EP 2571525	A1	20130327	EP 2011-784186		20110518 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR					
CN 103037903	A	20130410	CN 2011-80023143		20110518 <--
JP 2013526549	T	20130624	JP 2013-510367		20110518 <--
MX 2012013100	A	20130122	MX 2012-13100		20121109 <--
PRIORITY APPLN. INFO.:			US 2010-61345641	P	20100518 <--
			WO 2011-US37025	W	20110518

OTHER SOURCE(S): CASREACT 156:11747

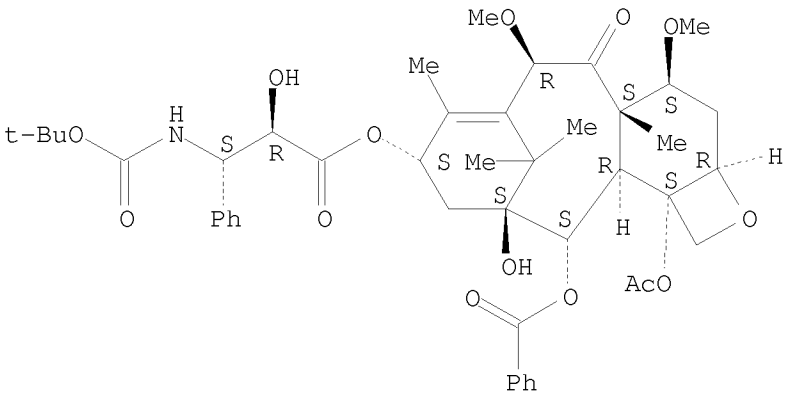
AB Provided are methods relating to the preparation and use of cyclodextrin-based polymer (CDP)-therapeutic agent conjugates for the treatment of autoimmune disease, inflammatory disease, or **cancer**. Also provided are CDP-therapeutic agent conjugates, particles comprising CDP-therapeutic agent conjugates, and compns. comprising CDP-therapeutic agent conjugates.

IT **183133-96-2, Cabazitaxel**
RL: PRPH (Prophetic); RCT (Reactant); **THU (Therapeutic use)**; BIOL (Biological study); RACT (Reactant or reagent); USES (Uses)
(compns. comprising a CDP-**therapeutic** agent conjugates and methods for **treatment** of autoimmune and other diseases)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 9 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN

ACCESSION NUMBER:	2011:1340624 HCAPLUS
DOCUMENT NUMBER:	155:587050
TITLE:	Antigen-presenting cell vaccine for treating solid **tumors**
INVENTOR(S):	Slawin, Kevin; Spencer, David; Lapteva, Natalia
PATENT ASSIGNEE(S):	Bellicum Pharmaceuticals, Inc., USA
SOURCE:	PCT Int. Appl., 267pp. CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
WO 2011130566	A2	20111020	WO 2011-US32572		20110414	<--
WO 2011130566	A3	20111229				
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW						
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM						
CA 2795947	A1	20111020	CA 2011-2795947		20110414	<--
US 20110287038	A1	20111124	US 2011-13087329		20110414	<--
AU 2011239569	A1	20121025	AU 2011-239569		20110414	<--
EP 2558109	A2	20130220	EP 2011-769619		20110414	<--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR						
JP 2013525305	T	20130620	JP 2013-505156		20110414	<--
PRIORITY APPLN. INFO.:			US 2010-61325127	P	20100416	<--
			US 2010-61351760	P	20100604	<--
			US 2011-61442582	P	20110214	
			WO 2011-US32572	W	20110414	

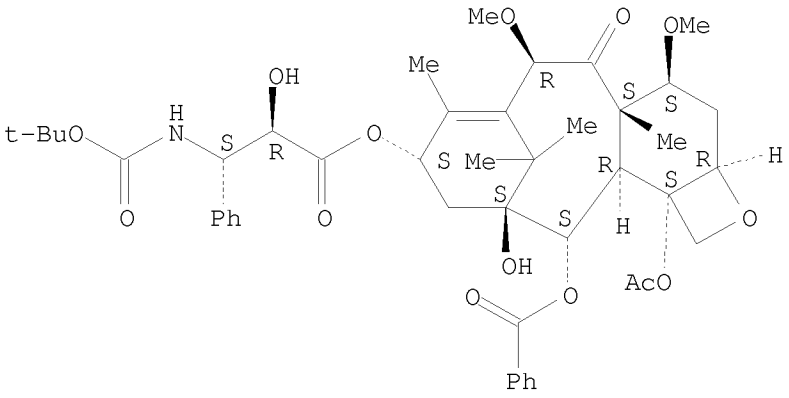
AB The authors disclose transgenic antigen-presenting cell expressing an inducible CD40 protein for activating an antigenic response against a prostate **cancer** antigen, for example prostate specific membrane antigen (PSMA,). The inducible CD40 comprises a membrane targeting region, a multimeric ligand-binding region and a CD40 cytoplasmic polypeptide region lacking the CD40 extracellular domain. In one example, inducible CD40 was constructed from the human CD40 cytoplasmic signaling domain, a myristoylation-targeting domain, and two tandem domains from human FKBP12 which bind the dimerizing drug AP20187.

IT **183133-96-2, Cabazitaxel**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(in combination **therapy** with transgenic CD40 chimera antigen-presenting cell vaccine)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 10 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:1337750 HCAPLUS
DOCUMENT NUMBER: 155:580869
TITLE: Therapeutic agents having reduced toxicity
INVENTOR(S): Mutz, Mitchell W.; Webb, Robert, III; Gestwicki, Jason E.
PATENT ASSIGNEE(S): Amplyx Pharmaceuticals Inc., USA
SOURCE: PCT Int. Appl., 58pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2011130317	A2	20111020	WO 2011-US32175	20110412 <--
WO 2011130317	A3	20120405		

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2010-61323820 P 20100413 <--
US 2010-61324211 P 20100414 <--
OTHER SOURCE(S): CASREACT 155:580869; MARPAT 155:580869

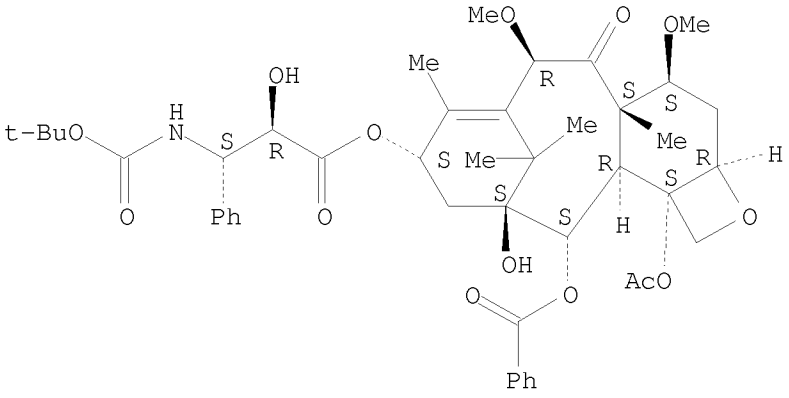
AB Therapeutic hybrid compds. having an active moiety and a toxicity reducing moiety are provided, as are methods of use of such compds., methods of preparation of such compds., and compns. containing such compds. In some embodiments, the hybrid compds. have lower toxicity (such as lower neurotoxicity) compared with the non-hybridized active moiety.

IT **183133-96-2, Cabazitaxel**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**therapeutic** agents having reduced toxicity)

RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-

(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 11 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:1304241 HCAPLUS
DOCUMENT NUMBER: 155:559702
TITLE: Detection of circulating mRNA, microRNA, protein and vesicle markers in disease diagnosis and selection of therapies
INVENTOR(S): Kuslich, Christine; Poste, George; Klass, Michael; Spetzler, David; Pawlowski, Traci
PATENT ASSIGNEE(S): Caris Life Sciences Luxembourg Holdings, Luxembourg
SOURCE: PCT Int. Appl., 584pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 13
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2011127219	A1	20111013	WO 2011-US31479	20110406 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2795776	A1	20111013	CA 2011-2795776	20110406 <--
AU 2011237669	A1	20121101	AU 2011-237669	20110406 <--
IL 222232	A	20121129	IL 2011-222232	20110406 <--
EP 2556172	A1	20130213	EP 2011-766687	20110406 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR				

CN 103025890	A	20130403	CN 2011-80027541		20110406	<--
KR 2013043104	A	20130429	KR 2012-7028831		20110406	<--
JP 2013526852	T	20130627	JP 2013-503934		20110406	<--
PRIORITY APPLN. INFO.:			US 2010-61321392	P	20100406	<--
			US 2010-61321407	P	20100406	<--
			US 2010-61322690	P	20100409	<--
			US 2010-61332174	P	20100506	<--
			US 2010-61334547	P	20100513	<--
			US 2010-61348214	P	20100525	<--
			US 2010-61348685	P	20100526	<--
			US 2010-61354125	P	20100611	<--
			US 2010-61355387	P	20100616	<--
			US 2010-61356974	P	20100621	<--
			US 2010-61357517	P	20100622	<--
			US 2010-61362674	P	20100708	<--
			US 2010-61364785	P	20100715	<--
			US 2010-61370088	P	20100802	<--
			US 2010-61379670	P	20100902	<--
			US 2010-61381305	P	20100909	<--
			US 2010-61383305	P	20100915	<--
			US 2010-61391504	P	20101008	<--
			US 2010-61393823	P	20101015	<--
			US 2010-61411890	P	20101109	
			US 2010-61413377	P	20101112	
			US 2010-61416560	P	20101123	
			WO 2011-US31479	W	20110406	

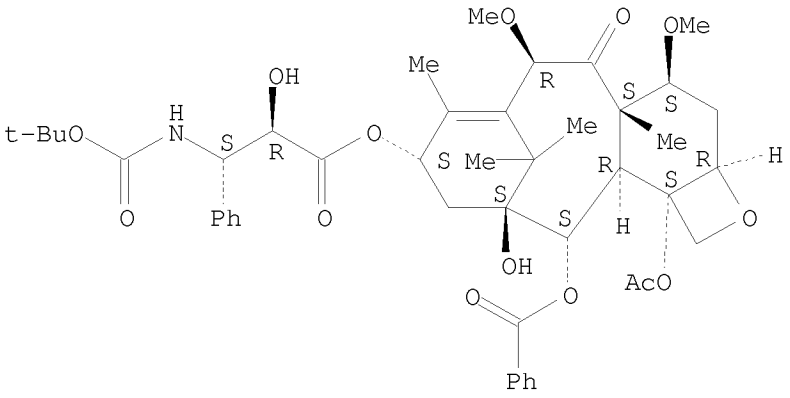
AB Biomarkers can be assessed for diagnostic, therapy-related or prognostic methods to identify phenotypes, such as a condition or disease, or the stage or progression of a disease. Circulating biomarkers from a bodily fluid can be used in profiling of physiol. states or determining phenotypes. These include nucleic acids, protein, and circulating structures such as vesicles. Biomarkers can be used for theranostic purposes to select candidate treatment regimens for diseases, conditions, disease stages, and stages of a condition, and can also be used to determine treatment efficacy. The biomarkers can be circulating biomarkers, including microvesicles and their protein content and microRNAs.

IT **183133-96-2, Cabazitaxel**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses) (selection for **cancer therapy**; detection of circulating mRNA, microRNA, protein and vesicle markers in disease diagnosis)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 12 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:1304315 HCAPLUS
DOCUMENT NUMBER: 155:527762
TITLE: Resistance expression signature-based methods for predicting or monitoring whether a patient affected by a **cancer** is responsive to a treatment with a molecule of the taxoid family
INVENTOR(S): Chauchereau, Anne; Commo, Frederic; Al Nakouzi, Nader
PATENT ASSIGNEE(S): Institut Gustave Roussy, Fr.
SOURCE: PCT Int. Appl., 68pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2011124669	A1	20111013	WO 2011-EP55482	20110408 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
EP 2556166	A1	20130213	EP 2011-713767	20110408 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR				
US 20130130928	A1	20130523	US 2012-13638053	20121204 <--
PRIORITY APPLN. INFO.:			EP 2010-305361 A	20100408 <--
			WO 2011-EP55482 W	20110408

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
AB The invention discloses methods for predicting or monitoring whether a

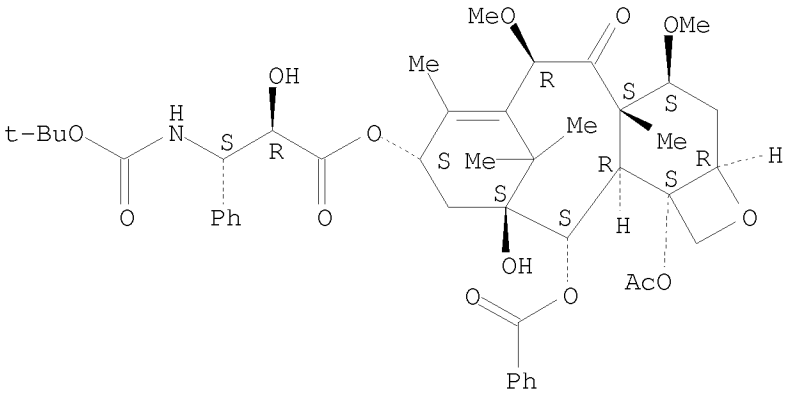
patient affected by a **cancer** is responsive to a treatment with a mol. of the taxoid family based on a resistance expression signature,. It also provides methods for screening or identifying a compound suitable for improving the treatment of a **cancer** with a mol. of the taxoid family or for reducing the resistance development during the treatment of a **cancer** with the mol. of the taxoid family. Taxoid family includes docetaxel, larotaxel, carbazitaxel (XRP6258), BMS-184476, BMS-188797, BMS275183, ortataxel, RPR 109881A, RPR116258, NBT-287, PG-paclitaxel, ABRAXANE, Tesetaxel, IDN5390, Taxoprexin, DHA-paclitaxel and MAC-321.

IT **183133-96-2**, XRP6258
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(signature-based methods for predicting or monitoring taxoid **antitumor** responsiveness)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 13 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:1236993 HCAPLUS
DOCUMENT NUMBER: 155:502024
TITLE: Compositions of glucose transport inhibitors as **antitumor** agents
INVENTOR(S): Chen, Xiaozhuo; Bergmeier, Stephen
PATENT ASSIGNEE(S): Ohio University, USA
SOURCE: PCT Int. Appl., 107pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

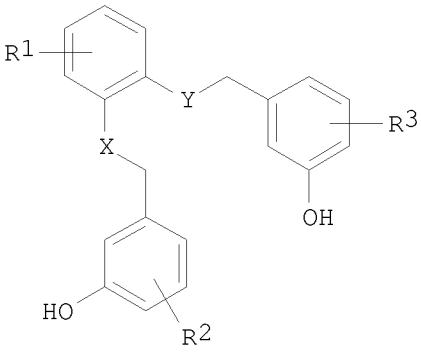
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2011119866	A1	20110929	WO 2011-US29843	20110324 <--

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG,

ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP,
KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA,
MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE,
PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV,
SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR,
HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS,
SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL,
SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

CA 2794266	A1	20110929	CA 2011-2794266		20110324 <--
US 20120121536	A1	20120517	US 2011-13071386		20110324 <--
EP 2549863	A1	20130130	EP 2011-760242		20110324 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR					
CN 103037690	A	20130410	CN 2011-80025712		20110324 <--
PRIORITY APPLN. INFO.:			US 2010-61317062	P	20100324 <--
			WO 2011-US29843	W	20110324

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): CASREACT 155:502024; MARPAT 155:502024
GI



I

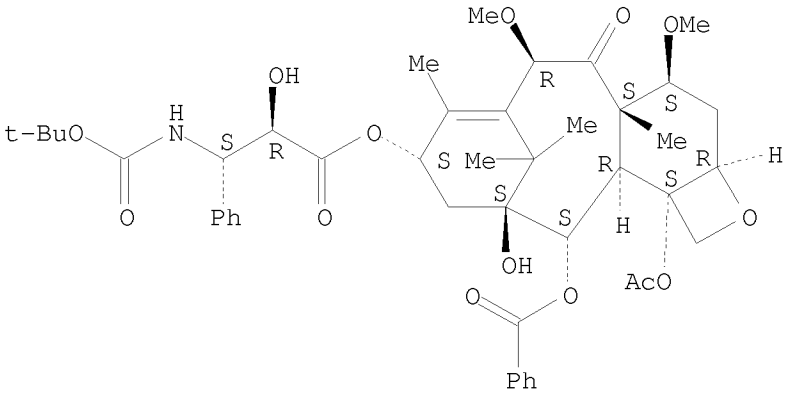
AB Glucose deprivation is an attractive strategy in **cancer** research and treatment. **Cancer** cells upregulate glucose uptake and metabolism for maintaining accelerated growth and proliferation rates. Specifically blocking these processes is likely to provide new insights to the role of glucose transport and metabolism in **tumorigenesis**, as well as in apoptosis. As solid **tumors** outgrow the surrounding vasculature, they encounter microenvironments with a limited supply of nutrients leading to a glucose deprived environment in some regions of the **tumor**. **Cancer** cells living in the glucose deprived environment undergo changes to prevent glucose deprivation-induced apoptosis. Knowing how **cancer** cells evade apoptosis induction is also likely to yield valuable information and knowledge of how to overcome the resistance to apoptosis induction in **cancer** cells. Disclosed herein are novel **anticancer** compds. of Formula I (wherein R1, R2, R3 = independently, H, halo, alkyl, benzyl, amino, nitro, cyano, and alkoxy; X, Y = independently, C, O, N, S) that inhibit basal glucose transport, resulting in **tumor** suppression and new methods for the study of glucose deprivation in animal **cancer** research.

IT **183133-96-2, Cabazitaxel**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(compns. and methods for glucose transport inhibition)

RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-

α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 14 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:653224 HCAPLUS
DOCUMENT NUMBER: 155:12279
TITLE: Preparation of cyclodextrin-based polymers for therapeutic delivery
INVENTOR(S): Wolfgang, Marc; Reiter, Lawrence A.; Crawford, Thomas C.; Fetzer, Oliver S.
PATENT ASSIGNEE(S): Cerulean Pharma Inc., USA
SOURCE: PCT Int. Appl., 341pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2011063421	A1	20110526	WO 2010-US57913	20101123 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2781669	A1	20110526	CA 2010-2781669	20101123 <--
AU 2010321533	A1	20120531	AU 2010-321533	20101123 <--
EP 2503888	A1	20121003	EP 2010-832380	20101123 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO,				

RS, SE, SI, SK, SM, TR

CN 102781237	A	20121114	CN 2010-80052896		20101123 <--
IL 219699	A	20130228	IL 2010-219699		20101123 <--
JP 2013511558	T	20130404	JP 2012-540162		20101123 <--
IN 2012MN01251	A	20120824	IN 2012-MN1251		20120522 <--
MX 2012005987	A	20120625	MX 2012-5987		20120523 <--
PRIORITY APPLN. INFO.:			US 2009-61263749	P	20091123 <--
			US 2010-61391922	P	20101011 <--
			WO 2010-US57913	W	20101123

OTHER SOURCE(S): CASREACT 155:12279

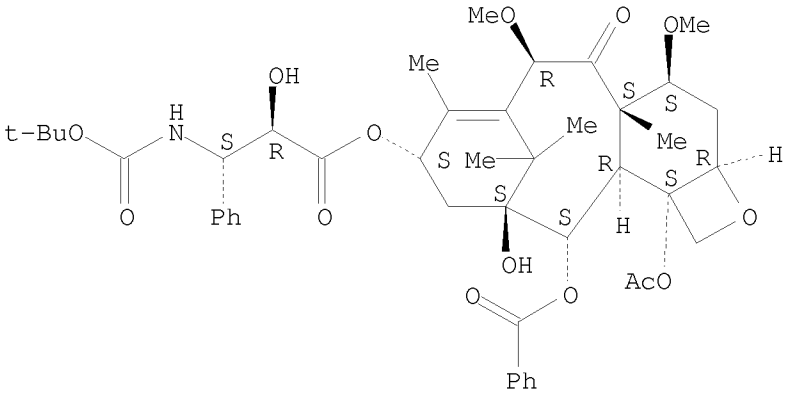
AB Methods and compns. relating to CDP-taxane conjugates are described herein. A method of selecting a subject having **cancer**, for treating the subject with a CDP-taxane conjugate was claimed, the method comprising: determining if a subject with **cancer** is at risk for or has diarrhea, proliferative disorder, fluid retention, hepatic impairment, severe neuropathy, from treatment with an **anticancer** agent with a CDP-taxane conjugate.

IT **183133-96-2, Cabazitaxel**
RL: RCT (Reactant); RACT (Reactant or reagent)
(preparation of cyclodextrin-based polymers for **therapeutic** delivery)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 15 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN

ACCESSION NUMBER: 2011:558134 HCAPLUS

DOCUMENT NUMBER: 154:504191

TITLE: Novel **antitumoral** use of cabazitaxel in metastatic prostate **cancer**

INVENTOR(S): Gupta, Sunil

PATENT ASSIGNEE(S): Aventis Pharma S.A., Fr.

SOURCE: PCT Int. Appl., 39pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2011051894	A1	20110505	WO 2010-IB54866	20101027 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2779009	A1	20110505	CA 2010-2779009	20101027 <--
AU 2010310986	A1	20120614	AU 2010-310986	20101027 <--
KR 2012093986	A	20120823	KR 2012-7013564	20101027 <--
EP 2493466	A1	20120905	EP 2010-782039	20101027 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BA, ME				
CN 102770131	A	20121107	CN 2010-80060052	20101027 <--
IL 219443	A	20130228	IL 2010-219443	20101027 <--
JP 2013509394	T	20130314	JP 2012-535996	20101027 <--
ZA 2012003123	A	20130626	ZA 2012-3123	20101027 <--
AR 78824	A1	20111207	AR 2010-103990	20101029 <--
CR 20120204	A	20120704	CR 2012-204	20120425 <--
US 20120301425	A1	20121129	US 2012-13456720	20120426 <--
MX 2012005030	A	20121205	MX 2012-5030	20120427 <--
IN 2012KN01241	A	20130125	IN 2012-KN1241	20120523 <--

PRIORITY APPLN. INFO.:	US 2009-61256160	P	20091029 <--
	US 2010-61293903	P	20100111 <--
	US 2010-61355834	P	20100617 <--
	US 2010-61355888	P	20100617 <--
	US 2010-61369929	P	20100802 <--
	US 2010-61383933	P	20100917 <--
	US 2010-61389969	P	20101005 <--
	WO 2010-IB54866	W	20101027

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention relates to an **antitumoral pharmaceutical** formulation comprising **cabazitaxel**, which may be in base form or in the form of a hydrate or a solvate, in combination with prednisone or prednisolone, for its use as a medicament in the **treatment** of prostate **cancer**, particularly metastatic prostate **cancer**, especially for patients who are not catered for by a taxane-based **treatment**.

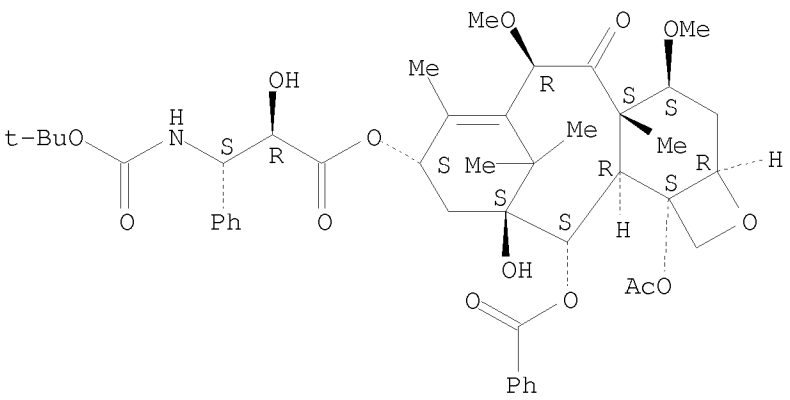
IT **183133-96-2**, **Cabazitaxel**
RL: ADV (Adverse effect, including toxicity); **PAC (Pharmacological activity)**; **PKT (Pharmacokinetics)**; **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(novel **antitumoral** use of **cabazitaxel** in metastatic prostate **cancer**)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX

NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 16 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:1407866 HCAPLUS
DOCUMENT NUMBER: 155:616725
TITLE: Polymer-agent conjugates, particles, compositions, and related methods of use
INVENTOR(S): Crawford, Thomas C.; Eliasof, Scott; Gangal, Geeti; Ng, Pei-Sze; Reiter, Lawrence Alan
PATENT ASSIGNEE(S): Cerulean Pharma Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 251pp., Cont.-in-part of U.S. Ser. No. 894,040.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20110268658	A1	20111103	US 2011-13004838	20110111 <--
CA 2756072	A1	20101014	CA 2010-2756072	20100326 <--
WO 2010117668	A1	20101014	WO 2010-US28770	20100326 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2010234916	A1	20111013	AU 2010-234916	20100326 <--
EP 2413901	A1	20120208	EP 2010-762122	20100326 <--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE,				

SI, SK, SM, TR

CN 102378626	A	20120314	CN 2010-80014516	20100326	<--
JP 2012522055	T	20120920	JP 2012-503526	20100326	<--
IL 215123	A	20130131	IL 2010-215123	20100326	<--
US 20100247668	A1	20100930	US 2010-748637	20100329	<--
US 20110189092	A1	20110804	US 2010-894040	20100929	<--
US 20110262490	A1	20111027	US 2011-13072297	20110325	<--
IN 2011KN03873	A	20120518	IN 2011-KN3873	20110919	<--
WO 2012050899	A1	20120419	WO 2011-US53716	20110928	<--

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RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

MX 2011010390	A	20111214	MX 2011-10390	20110930	<--

PRIORITY APPLN. INFO.:

US 2009-61164720	P	20090330	<--
US 2009-61164722	P	20090330	<--
US 2009-61164725	P	20090330	<--
US 2009-61164728	P	20090330	<--
US 2009-61164731	P	20090330	<--
US 2009-61164734	P	20090330	<--
US 2009-61262993	P	20091120	<--
US 2009-61262994	P	20091120	<--
WO 2010-US28770	A1	20100326	<--
US 2010-748637	A2	20100329	<--
US 2010-894040	A2	20100929	<--
US 2011-13004838	A2	20110111	
US 2011-13072297	A1	20110325	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

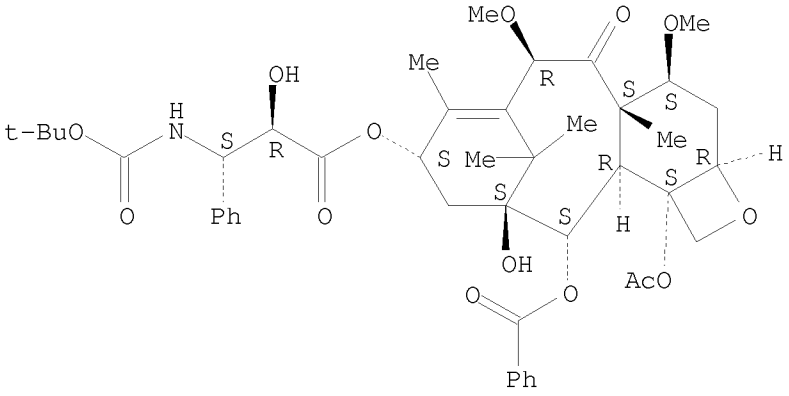
AB Described herein are polymer-agent conjugates and particles, which can be used, for example, in the treatment of **cancer**. Also described herein are mixts., compns. and dosage forms containing the particles, methods of using the particles (e.g., to treat a disorder), kits including the polymer-agent conjugates and particles, methods of making the polymer-agent conjugates and particles, methods of storing the particles and methods of analyzing the particles.

IT **183133-96-2DP**, reaction products with aminoisophthalic acid and glycolic acid-lactic acid copolymer
RL: IMF (Industrial manufacture); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(polymer-agent conjugates, particles, compns., and related methods of use)

RN 183133-96-2 HCAPLUS

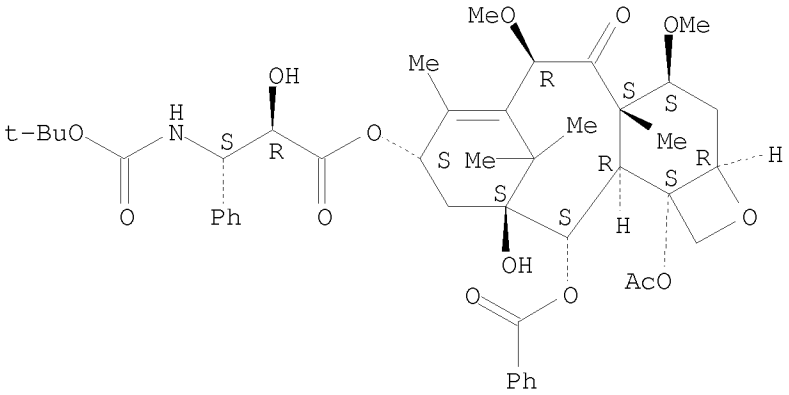
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



IT **183133-96-2D**, conjugates with polymers
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(polymer-agent conjugates, particles, compns., and related methods of use)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

L15 ANSWER 17 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:1369690 HCAPLUS
DOCUMENT NUMBER: 155:624719
TITLE: Biodegradable hydrophobic polymer-drug conjugates and compositions containing particles containing these conjugates for treatment of diseases
INVENTOR(S): Zhang, Jerry; Ng, Pei-Sze
PATENT ASSIGNEE(S): Cerulean Pharma Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 265pp., Cont.-in-part of U.S. Ser. No. 4,838.
CODEN: USXXCO
DOCUMENT TYPE: Patent

LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE	
US 20110262490	A1	20111027	US 2011-13072297		20110325	<--
CA 2756072	A1	20101014	CA 2010-2756072		20100326	<--
WO 2010117668	A1	20101014	WO 2010-US28770		20100326	<--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW						
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM						
AU 2010234916	A1	20111013	AU 2010-234916		20100326	<--
EP 2413901	A1	20120208	EP 2010-762122		20100326	<--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR						
CN 102378626	A	20120314	CN 2010-80014516		20100326	<--
JP 2012522055	T	20120920	JP 2012-503526		20100326	<--
IL 215123	A	20130131	IL 2010-215123		20100326	<--
US 20100247668	A1	20100930	US 2010-748637		20100329	<--
US 20110189092	A1	20110804	US 2010-894040		20100929	<--
US 20110268658	A1	20111103	US 2011-13004838		20110111	<--
IN 2011KN03873	A	20120518	IN 2011-KN3873		20110919	<--
WO 2012050899	A1	20120419	WO 2011-US53716		20110928	<--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, QA, RO, RS, RU, RW, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW						
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM						
MX 2011010390	A	20111214	MX 2011-10390		20110930	<--
PRIORITY APPLN. INFO.:			US 2009-61164720	P	20090330	<--
			US 2009-61164722	P	20090330	<--
			US 2009-61164725	P	20090330	<--
			US 2009-61164728	P	20090330	<--
			US 2009-61164731	P	20090330	<--
			US 2009-61164734	P	20090330	<--
			US 2009-61262993	P	20091120	<--
			US 2009-61262994	P	20091120	<--
			WO 2010-US28770	A1	20100326	<--
			US 2010-748637	A2	20100329	<--
			US 2010-894040	A2	20100929	<--
			US 2011-13004838	A2	20110111	
			US 2011-13072297	A1	20110325	

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB Compns. exhibiting extended blood stability and sustained drug release contain particles containing biodegradable hydrophobic polymer-drug conjugates, hydrophobic-hydrophilic polymers, and surfactants and cyclooligosaccharides as stabilizers/lyoprotectants. A typical composition was prepared by dissolving 600 mg glycolic acid-lactic acid copolymer acetate amide with docetaxel 2-β-alanine glycolate and 400 mg glycolic acid-lactic acid copolymer ester with polyethylene glycol monomethyl ether in Me2CO, mixing the resulting 1.0% solution in 1:10 with 0.5% (w/v) polyvinyl alc. surfactant, removing the Me2CO, washing the resulting nanoparticles with 10 vols. water, concentrating, adjusting the resulting nanoparticle solution to 10% sucrose, and lyophilizing.

IT **1345729-91-0P**
RL: IMF (Industrial manufacture); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(compns. containing oligosaccharides and particulates containing biodegradable hydrophobic polymer-**drug** conjugates, surfactants, and hydrophilic-hydrophobic polymers for **treatment** of diseases)

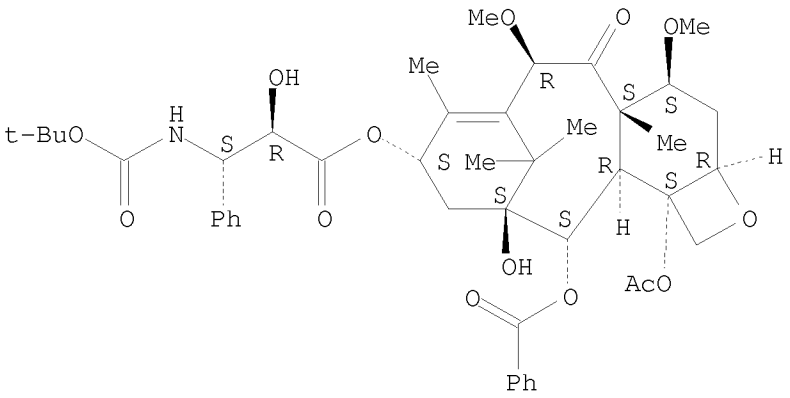
RN 1345729-91-0 HCAPLUS

CN Propanoic acid, 2-hydroxy-, polymer with 2-hydroxyacetic acid, (1R,2S)-1-[[[(2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl]oxy]carbonyl]-2-[[(1,1-dimethylethoxy)carbonyl]amino]-2-phenylethyl ester, acetate (ester) (CA INDEX NAME)

CM 1

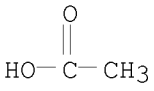
CRN 183133-96-2
CMF C45 H57 N O14

Absolute stereochemistry. Rotation (-).



CM 2

CRN 64-19-7
CMF C2 H4 O2

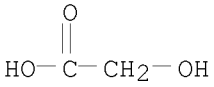


CM 3

CRN 34346-01-5
CMF (C3 H6 O3 . C2 H4 O3)x
CCI PMS

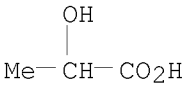
CM 4

CRN 79-14-1
CMF C2 H4 O3



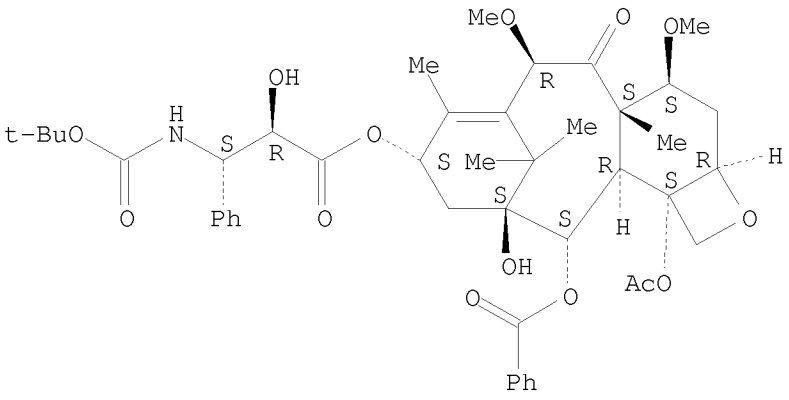
CM 5

CRN 50-21-5
CMF C3 H6 O3



IT **183133-96-2, Cabazitaxel**
RL: RCT (Reactant); RACT (Reactant or reagent)
(compns. containing oligosaccharides and particulates containing biodegradable
hydrophobic polymer-**drug** conjugates, surfactants, and
hydrophilic-hydrophobic polymers for **treatment** of diseases)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 18 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:974818 HCAPLUS
DOCUMENT NUMBER: 155:303002
TITLE: Polymer-agent conjugates, particles, compositions, and related methods of use
INVENTOR(S): Eliasof, Scott; Crawford, Thomas C.; Gangal, Geeti; Reiter, Lawrence Alan; Ng, Pei-Sze
PATENT ASSIGNEE(S): Cerulean Pharma Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 239pp., Cont.-in-part of U.S. Ser. No. 748,637.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 6
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20110189092	A1	20110804	US 2010-894040	20100929 <--
CA 2756072	A1	20101014	CA 2010-2756072	20100326 <--
WO 2010117668	A1	20101014	WO 2010-US28770	20100326 <--
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RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2010234916	A1	20111013	AU 2010-234916	20100326 <--
EP 2413901	A1	20120208	EP 2010-762122	20100326 <--
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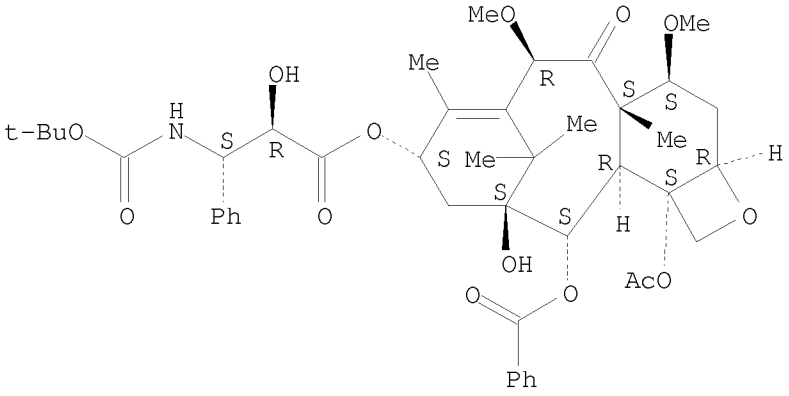
CN 102378626	A	20120314	CN 2010-80014516	20100326 <--
JP 2012522055	T	20120920	JP 2012-503526	20100326 <--
IL 215123	A	20130131	IL 2010-215123	20100326 <--
US 20100247668	A1	20100930	US 2010-748637	20100329 <--
US 20110268658	A1	20111103	US 2011-13004838	20110111 <--
US 20110262490	A1	20111027	US 2011-13072297	20110325 <--
IN 2011KN03873	A	20120518	IN 2011-KN3873	20110919 <--
WO 2012050899	A1	20120419	WO 2011-US53716	20110928 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, QA, RO, RS, RU, RW, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, RW, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
MX 2011010390	A	20111214	MX 2011-10390	20110930 <--
US 20120282306	A1	20121108	US 2012-13548143	20120712 <--

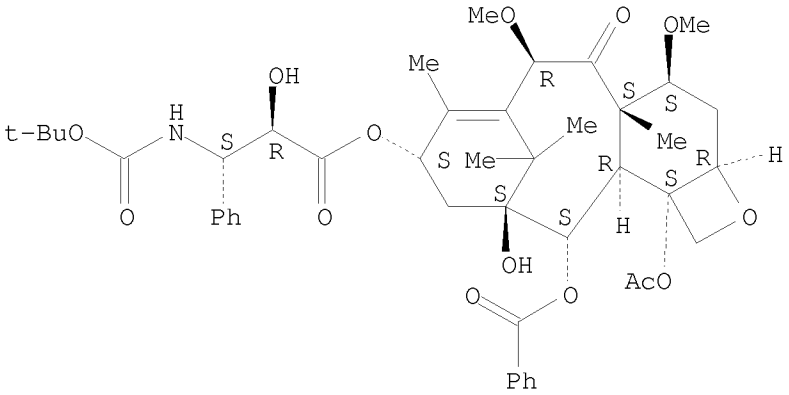
US 20130011445 A1 20130110 US 2012-13548108 20120712 <--
PRIORITY APPLN. INFO.: US 2009-61164720 P 20090330 <--
US 2009-61164722 P 20090330 <--
US 2009-61164725 P 20090330 <--
US 2009-61164728 P 20090330 <--
US 2009-61164731 P 20090330 <--
US 2009-61164734 P 20090330 <--
US 2009-61262993 P 20091120 <--
US 2009-61262994 P 20091120 <--
WO 2010-US28770 A1 20100326 <--
US 2010-748637 A2 20100329 <--
US 2010-894040 A2 20100929 <--
US 2011-13004838 A2 20110111
US 2011-13004848 A1 20110111
US 2011-13072297 A1 20110325
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): CASREACT 155:303002
AB A particle comprises: (a) a plurality of hydrophobic polymer-agent conjugates, wherein (i) each hydrophobic polymer-agent conjugate of the plurality comprises a hydrophobic polymer attached to an agent, (ii) the hydrophobic polymer attached to the agent can be a homopolymer or a polymer made up of more than one kind of monomeric subunit, (iii) the hydrophobic polymer attached to the agent has a weight average mol. weight of about 4-15 kD, (iv) the agent is about 1-30% of the particle and (v) the plurality of hydrophobic polymer-agent conjugates is about 25-80% of the particle; (b) a plurality of hydrophilic-hydrophobic polymers, wherein i) each of the hydrophilic-hydrophobic polymers of the plurality comprises a hydrophilic portion attached to a hydrophobic portion, (ii) the hydrophilic portion has a weight average mol. weight of about 1-6 kD, and (iii) the plurality of hydrophilic-hydrophobic polymers is about 5-30% of the particle; and (c) a surfactant, wherein the surfactant is about 15-35% of the particle; and wherein: the diameter of the particle is less than about 200 nm. The polymer-agent conjugates and particles can be used, for example, in the treatment of **cancer**. Also described herein are mixts., compns. and dosage forms containing the particles, methods of using the particles (e.g., to treat a disorder), kits including the polymer-agent conjugates and particles, methods of making the polymer-agent conjugates and particles, methods of storing the particles and methods of analyzing the particles.
IT **183133-96-2DP**, reaction products with aminoisophthalic acid and glycolic acid-lactic acid copolymer
RL: IMF (Industrial manufacture); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(polymer-agent conjugates, particles, compns., and related methods of use)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



IT **183133-96-2D, Cabazitaxel**, conjugates with polymers
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(polymer-agent conjugates, particles, compns., and related methods of use)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 19 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:563558 HCAPLUS
DOCUMENT NUMBER: 154:513570
TITLE: Process for preparation of taxane derivatives
INVENTOR(S): Gurjar, Mukund K.; Sonawane, Swapnil P.; Patil, Pankaj S.; Mehta, Samit S.
PATENT ASSIGNEE(S): Emcure Pharmaceuticals Limited, India
SOURCE: U.S. Pat. Appl. Publ., 7pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

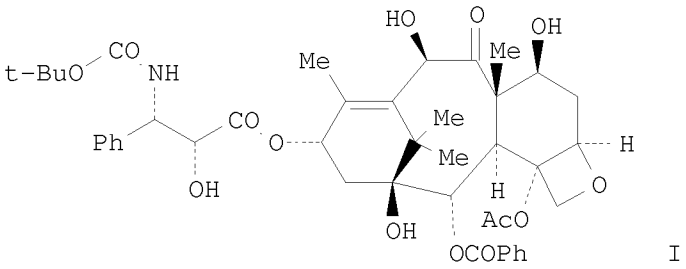
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20110105598	A1	20110505	US 2010-917823	20101102 <--
EP 2330100	A1	20110608	EP 2010-14181	20101102 <--

R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, RS, SE, SI, SK, SM, TR, BA, ME

PRIORITY APPLN. INFO.: IN 2009-MU2559 A 20091104 <--

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

GI



I

AB Processes for the preparation of taxane derivs. with improved purity and enhanced stability were disclosed. The taxane derivs. prepared according to the disclosed processes described are useful for the preparation of pharmaceutical compns. Thus, docetaxel trihydrate was prepared with 70-90% yield from crude docedtaxel (I) using potassium aluminum sulfate in EtOH/cyclohexane/DM water/EtOAc.

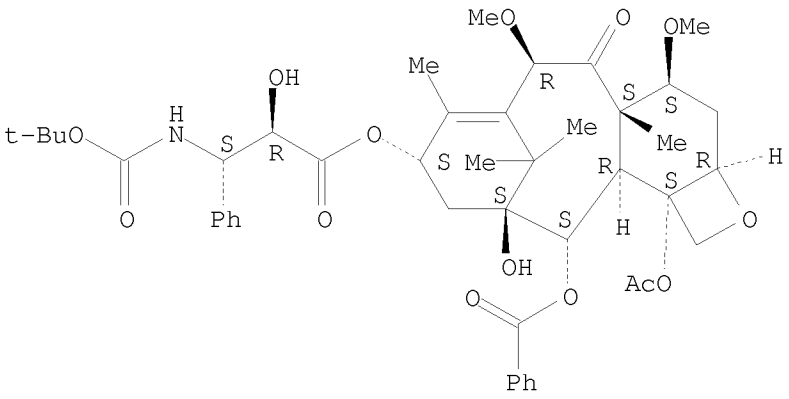
IT **183133-96-2, Cabazitaxel**

RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses) (claimed compound; processes for the preparation of taxane derivs. with improved purity and enhanced stability for **pharmaceutical** compns.)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD

(1 CITINGS)

L15 ANSWER 20 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:550734 HCAPLUS
DOCUMENT NUMBER: 154:477276
TITLE: Novel **antitumoral** use of cabazitaxel
INVENTOR(S): Gupta, Sunil
PATENT ASSIGNEE(S): Aventis Pharma S.A., Fr.
SOURCE: Can. Pat. Appl., 28pp.
CODEN: CPXXEB
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

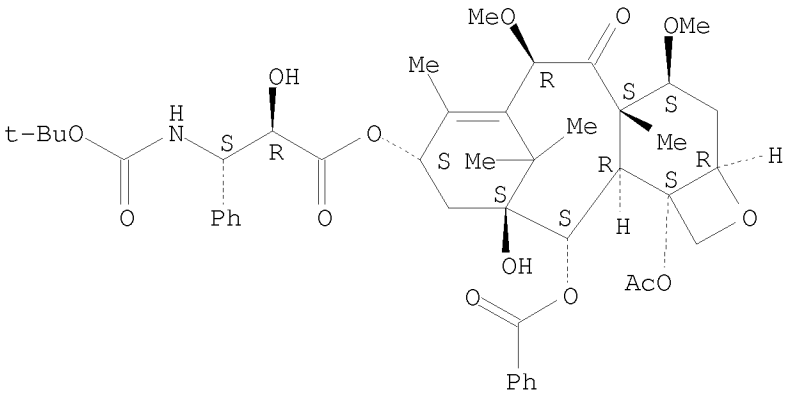
PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
CA 2708489	A1	20110429	CA 2010-2708489		20100623 <--
PRIORITY APPLN. INFO.:			US 2009-61256160	P	20091029 <--
			US 2010-61293903	P	20100111 <--
			US 2010-61355834	P	20100617 <--

AB The invention relates to an **antitumoral pharmaceutical** combination comprising **cabazitaxel** of formula:(see above formula)and prednisone or prednisolone, these two agents possibly being in base form, or in the form of a hydrate or a solvate, intended for **treating** metastatic prostate **cancer**, especially for patients who are not catered for by a taxane-based **treatment**.

IT **183133-96-2, Cabazitaxel**
RL: ADV (Adverse effect, including toxicity); **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(novel **antitumoral** use of **cabazitaxel**)

RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 21 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2010:1405568 HCAPLUS
DOCUMENT NUMBER: 153:596018

TITLE: **Antitumor** combination comprising cabazitaxel and capecitabine
INVENTOR(S): Magherini, Emmanuelle
PATENT ASSIGNEE(S): Sanofi-Aventis, Fr.
SOURCE: PCT Int. Appl., 25pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2010128258	A1	20101111	WO 2010-FR50873	20100506 <--
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RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
FR 2945211	A1	20101112	FR 2009-2189	20090506 <--
FR 2945212	A1	20101112	FR 2009-2264	20090511 <--
FR 2945212	B1	20110701		
CA 2761079	A1	20101111	CA 2010-2761079	20100506 <--
AU 2010244253	A1	20111124	AU 2010-244253	20100506 <--
KR 2012008069	A	20120125	KR 2011-7029019	20100506 <--
EP 2427187	A1	20120314	EP 2010-727466	20100506 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BA, ME, RS				
CN 102458392	A	20120516	CN 2010-80030429	20100506 <--
JP 2012526089	T	20121025	JP 2012-509077	20100506 <--
IL 216063	A	20130131	IL 2010-216063	20100506 <--
US 20120115806	A1	20120510	US 2011-13289250	20111104 <--
MX 2011011765	A	20120601	MX 2011-11765	20111104 <--

PRIORITY APPLN. INFO.:	FR 2009-2189	A	20090506	<--
	FR 2009-2264	A	20090511	<--
	WO 2010-FR50873	W	20100506	<--

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention relates to a **pharmaceutical antitumor** combination including **cabazitaxel** and capecitabine, wherein both of said **antitumor** agents may be in the form of a base, in the form of a **pharmaceutically** acceptable acid salt or in the form of a hydrate or solvate, intended for the **treatment** of metastatic breast **cancer** in patients progressing after a previous **treatment** with anthracyclines and taxanes. Efficacy of a combination of **cabazitaxel** and capecitabine in the **treatment** of patients suffering from breast **cancer** was described.

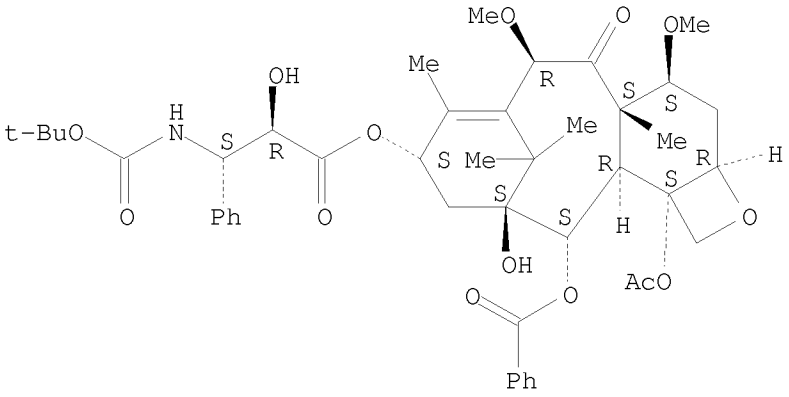
IT **183133-96-2, Cabazitaxel**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**antitumor** combination comprising **cabazitaxel** and capecitabine)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-

dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 22 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2010:437266 HCAPLUS
DOCUMENT NUMBER: 152:446754
TITLE: Resistance expression signature-based methods for predicting or monitoring whether a patient affected by a **cancer** is responsive to a treatment with a molecule of the taxoid family
INVENTOR(S): Chauchereau, Anne; Al Nakouzi, Nader
PATENT ASSIGNEE(S): Institut Gustave Roussy, Fr.; INSERM (Institut National de la Sante et de la Recherche Medicale)
SOURCE: PCT Int. Appl., 73pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2010037859	A2	20100408	WO 2009-EP62851	20091002 <--
WO 2010037859	A3	20100603		

W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA

EP 2177630	A1	20100421	EP 2008-305634	20081002 <--

R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU,

IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI,
SK, TR, AL, BA, MK, RS
EP 2356256 A2 20110817 EP 2009-783708 20091002 <--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU,
IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE,
SI, SK, SM, TR
US 20110177970 A1 20110721 US 2011-13121975 20110331 <--
PRIORITY APPLN. INFO.: EP 2008-305634 A 20081002 <--
WO 2009-EP62851 W 20091002 <--
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

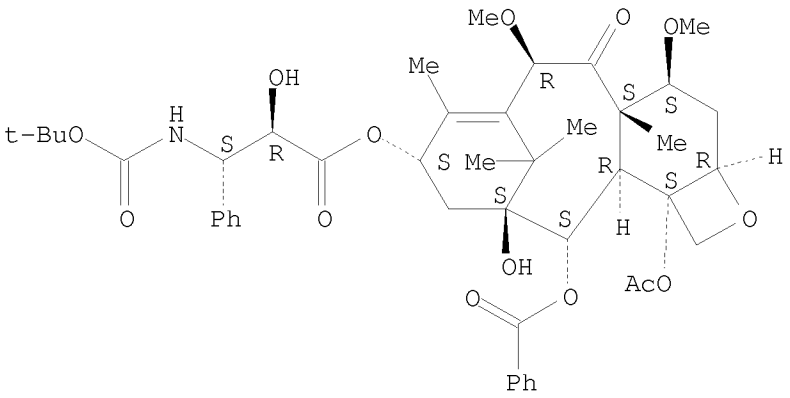
AB The invention discloses in vitro methods for predicting or monitoring whether a patient affected by a **cancer** is responsive to a treatment with a mol. of the taxoid family based on a resistance expression signature, as well as kits for performing the methods and methods for screening or identifying a compound suitable for improving the treatment of a **cancer** with a mol. of the taxoid family or for reducing the resistance development during the treatment of a **cancer** with the mol. of the taxoid family.

IT **183133-96-2**
RL: BSU (Biological study, unclassified); **PAC (Pharmacological activity)**; **THU (Therapeutic use)**; BIOL (Biological study);
USES (Uses)
(signature-based methods for predicting or monitoring taxoid **antitumor** responsiveness)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 23 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2010:1127861 HCAPLUS
DOCUMENT NUMBER: 153:440825
TITLE: Surface topographies for non-toxic bioadhesion control
INVENTOR(S): Brennan, Anthony B.; Long, Christopher James; Bagan, Joseph W.; Schumacher, James Frederick; Spiecker, Mark M.
PATENT ASSIGNEE(S): University of Florida, USA
SOURCE: U.S. Pat. Appl. Publ., 64pp., Cont.-in-part of U.S. Ser. No. 567,103.
CODEN: USXXCO

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 20100226943	A1	20100909	US 2009-550870	20090831
US 20050178286	A1	20050818	US 2004-780424	20040217
US 7650848	B2	20100126	US 2006-567103	20061205
PRIORITY APPLN. INFO.:			US 2004-780424 A2	20040217
			US 2005-202532 A2	20050812
			US 2006-567103 A2	20061205

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention relates to articles and related devices and systems having surface topog. and/or surface elastic properties for providing non-toxic bioadhesion control. An article includes a first plurality of spaced features arranged in a plurality of groupings including repeat units. The spaced features within a grouping are spaced apart at an average distance of about 1 nm to about 500 μm, each feature having a surface that is substantially parallel to a surface on a neighboring feature separated from its neighboring feature. The groupings of features are arranged with respect to one another so as to define a tortuous pathway. The plurality of spaced features provide the article with an engineered roughness index of about 5 to about 20.

OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (4 CITINGS)

L15 ANSWER 24 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2010:1403888 HCAPLUS
DOCUMENT NUMBER: 153:596016
TITLE: **Antitumor** combination comprising cabazitaxel and capecitabine
PATENT ASSIGNEE(S): Sanofi Aventis, Fr.
SOURCE: Fr. Demande, 11pp.
CODEN: FRXXBL
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
FR 2945211	A1	20101112	FR 2009-2189	20090506 <--
FR 2945212	A1	20101112	FR 2009-2264	20090511 <--
FR 2945212	B1	20110701		
CA 2761079	A1	20101111	CA 2010-2761079	20100506 <--
WO 2010128258	A1	20101111	WO 2010-FR50873	20100506 <--
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TH, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LR, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
AU 2010244253	A1	20111124	AU 2010-244253	20100506 <--
KR 2012008069	A	20120125	KR 2011-7029019	20100506 <--

EP 2427187		A1	20120314	EP 2010-727466		20100506 <--
R: AL, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BA, ME, RS						
CN 102458392		A	20120516	CN 2010-80030429		20100506 <--
JP 2012526089		T	20121025	JP 2012-509077		20100506 <--
ZA 2011008109		A	20130130	ZA 2011-8109		20100506 <--
IL 216063		A	20130131	IL 2010-216063		20100506 <--
US 20120115806		A1	20120510	US 2011-13289250		20111104 <--
MX 2011011765		A	20120601	MX 2011-11765		20111104 <--
PRIORITY APPLN. INFO.:				FR 2009-2189	A	20090506 <--
				FR 2009-2264	A	20090511 <--
				WO 2010-FR50873	W	20100506 <--

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

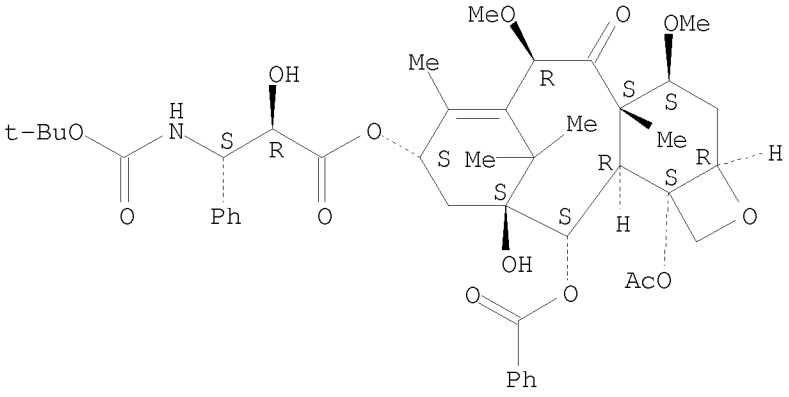
AB The invention relates to a **pharmaceutical antitumor** combination including **cabazitaxel** and capecitabine, wherein both of said **antitumor** agents may be in the form of a base, in the form of a **pharmaceutically** acceptable acid salt or in the form of a hydrate or solvate, intended for the **treatment** of metastatic breast **cancer** in patients progressing after a previous **treatment** with anthracyclines and taxanes. Efficacy of a combination of **cabazitaxel** and capecitabine in the **treatment** of patients suffering from breast **cancer** was described.

IT **183133-96-2**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**antitumor** combination comprising **cabazitaxel** and capecitabine)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 25 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN

ACCESSION NUMBER:	2010:498376 HCAPLUS
DOCUMENT NUMBER:	152:493436
TITLE:	Gene expression markers for prediction of the response of a **tumor** to taxane therapy
INVENTOR(S):	Chauchereau, Anne; Al Nakouzi, Nader
PATENT ASSIGNEE(S):	Institut Gustave Roussy, Fr.
SOURCE:	Eur. Pat. Appl., 59pp.

CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 2177630	A1	20100421	EP 2008-305634	20081002 <--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MT, NL, NO, PL, PT, RO, SE, SI, SK, TR, AL, BA, MK, RS				
WO 2010037859	A2	20100408	WO 2009-EP62851	20091002 <--
WO 2010037859	A3	20100603		
W: AE, AG, AL, AM, AO, AT, AU, AZ, BA, BB, BG, BH, BR, BW, BY, BZ, CA, CH, CL, CN, CO, CR, CU, CZ, DE, DK, DM, DO, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LY, MA, MD, ME, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PE, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, ST, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AP, EA, EP, OA				
EP 2356256	A2	20110817	EP 2009-783708	20091002 <--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HR, HU, IE, IS, IT, LI, LT, LU, LV, MC, MK, MT, NL, NO, PL, PT, RO, SE, SI, SK, SM, TR				
US 20110177970	A1	20110721	US 2011-13121975	20110331 <--

PRIORITY APPLN. INFO.: EP 2008-305634 A 20081002 <--
WO 2009-EP62851 W 20091002 <--

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

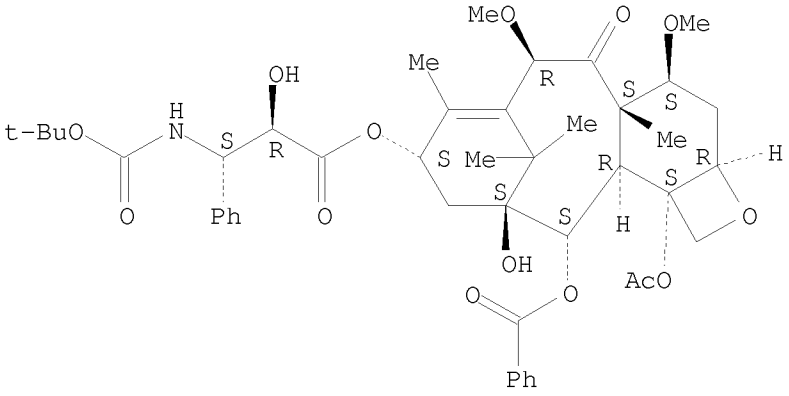
AB Gene expression markers that can be used to predict the response of a **tumor** to therapy with taxanes are described. These markers can also be used to monitor the effectiveness of the therapy. Markers that show increased expression in a favorable response and markers that show decreased expression are identified. There are 300 genes that are members of known biochem. networks and anal. of expression of one or two members of each network is sufficient to assess the response to the therapy. Changes in expression can also be used in the early identification of the development of resistance to these drugs.

IT **183133-96-2**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(as **antitumor** agent, predicting **tumor** response to;
gene expression markers for prediction of response of **tumor**
to taxane **therapy**)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 2 THERE ARE 2 CAPLUS RECORDS THAT CITE THIS RECORD (2 CITINGS)
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 26 OF 101 BIOSIS COPYRIGHT (c) 2013 The Thomson Corporation on STN
ACCESSION NUMBER: 2010:609402 BIOSIS
DOCUMENT NUMBER: PREV201000609402
TITLE: Challenges for the Development of New Agents in Prostate **Cancer.**
AUTHOR(S): Alva, Ajjai S.; Bradley, Deborah A.; Hussain, Maha [Reprint Author]
CORPORATE SOURCE: Univ Michigan, Ctr Comprehens Canc, Ann Arbor, MI 48109 USA mahahuss@umich.edu
SOURCE: Figg, WD [Editor]; Chau, CH [Editor]; Small, EJ [Editor]. (**2010**) pp. 389-397. Drug Management of Prostate Cancer. Publisher: SPRINGER, 233 SPRING STREET, NEW YORK, NY 10013, UNITED STATES.
ISBN: 978-1-60327-831-7(H).
DOCUMENT TYPE: Book; (Book Chapter)
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Nov 2010
Last Updated on STN: 2 May 2012
AB Development of novel therapeutic agents in advanced prostate **cancer** presents particular challenges due to multiple factors including advanced age at diagnosis with competing causes of death and difficulties in assessing responses in bone (the predominant site of metastatic disease). Over the last 2 decades, few non hormonal drugs have met the regulatory requirements for approval in advanced prostate **cancer.** These include mitoxantrone (1996), zoledronic acid (2002) and docetaxel (2004). Sipuleucel-T, an autologous cell based vaccine and **cabazitaxel** were approved in 2010. Despite these breakthroughs, the general landscape for new and effective treatments in prostate **cancer** remains challenging. The aim of this review is to discuss the specific obstacles in the development of novel agents in prostate **cancer** and potential strategies to overcome them.

L15 ANSWER 27 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2011109972 EMBASE
TITLE: Clinical **cancer** advances 2010: Annual report on progress against **cancer** from the american society of clinical **oncology.**

AUTHOR: Kris, Mark G.; Benowitz, Steven I.; Adams, Sylvia; Diller, Lisa; Ganz, Patricia; Kahlenberg, Morton S.; Le, Quynh-Thu; Markman, Maurie; Masters, Greg A.; Newman, Lisa; Obel, Jennifer C.; Seidman, Andrew D.; Smith, Sonali M.; Vogelzang, Nicholas; Petrelli, Nicholas J.
CORPORATE SOURCE: American Society of Clinical Oncology, 2318 Mill Road, Alexandria, VA 22314, United States. steven.benowitz@asco.org
AUTHOR: Benowitz, S. I., Dr. (correspondence)
CORPORATE SOURCE: American Society of Clinical Oncology, 2318 Mill Road, Alexandria, VA 22314, United States. steven.benowitz@asco.org
SOURCE: Journal of Clinical Oncology, (20 Dec 2010) Vol. 28, No. 36, pp. 5327-5347.
Refs: 57
ISSN: 0732-183X; E-ISSN: 1527-7755 CODEN: JCONDN
PUBLISHER: American Society of Clinical Oncology, 330 John Carlyle Street, Suite 300, Alexandria, VA 22314, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 7 Mar 2011
Last Updated on Embase: 7 Mar 2011

L15 ANSWER 28 OF 101 BIOSIS COPYRIGHT (c) 2013 The Thomson Corporation on STN DUPLICATE 1
ACCESSION NUMBER: 2011:155349 BIOSIS
DOCUMENT NUMBER: PREV201100155349
TITLE: Actualities in prostate **cancer** in ASCO annual meeting 2010.
Original Title: Actualites dans le **cancer** de la prostate lors de l'Asco 2010.
AUTHOR(S): Pouessel, D. [Reprint Author]; Culine, S.
CORPORATE SOURCE: Hop Henri Mondor, Med Oncol Serv, F-94010 Creteil, France damien.pouessel@hmn.aphp.fr
SOURCE: Bulletin du Cancer (Montrouge), (**DEC 2010**) Vol. 97, No. 12, pp. 1563-1572.
CODEN: BUCABS. ISSN: 0007-4551. E-ISSN: 1769-6917.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: French
ENTRY DATE: Entered STN: 23 Mar 2011
Last Updated on STN: 23 Mar 2011

AB In urologic **oncology**, prostate **cancer** represented, even this year, a wide part during the ASCO 2010 meeting. In the non metastatic diseases, two phase III trials confirmed the benefit of radiotherapy combined with hormonotherapy in locally advanced stage. For patients with metastatic hormonoresistant **cancer**, two randomized trials will probably change the daily practice in the next months. On the one hand, denosumab versus zoledronate decreased significantly the risk of skeletal-related events in bone metastases. On the other hand, compared with mitoxantrone, **cabazitaxel** in docetaxel pretreated patients improved overall survival. On the contrary, docetaxel in monotherapy remains the standard of care in first line chemotherapy in castration refractory metastatic prostate **cancer**. Indeed, in two trials, combination of bevacizumab or calcitriol with docetaxel showed no benefit for patients with more toxicities. Finally, docetaxel-based chemotherapy was again evaluated in two other situations: biological recurrence, and hormono-sensitive metastatic stage. Preliminary results of tolerance were presented this year. No doubt that communications during future ASCO meetings would reported definitive

results of efficiency of these phase III studies.

L15 ANSWER 29 OF 101 MEDLINE ® on STN
ACCESSION NUMBER: 2010994252 MEDLINE
DOCUMENT NUMBER: PubMed ID: 20868208
TITLE: Horizon scanning for novel therapeutics for the treatment of prostate **cancer**.
AUTHOR: Bianchini Diletta; Zivi Andrea; Sandhu Shahneen; de Bono Johann S
CORPORATE SOURCE: The Institute of Cancer Research, Royal Marsden NHS Foundation Trust, Section of Medicine, Drug Development Unit, Downs Road, Sutton, Surrey SM2 5PT, UK.
SOURCE: Expert opinion on investigational drugs, **(2010 Dec)** Vol. 19, No. 12, pp. 1487-502. Electronic Publication Date: 24 Sep 2010
Journal code: 9434197. E-ISSN: 1744-7658. L-ISSN: 1354-3784.
DIGITAL OBJECT IDENTIFIER: 10.1517/13543784.2010.514261
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RETRACTED PUBLICATION)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201109
ENTRY DATE: Entered STN: 26 Nov 2010
Last Updated on STN: 30 Sep 2011
Entered Medline: 29 Sep 2011
AB IMPORTANCE OF THE FIELD: Treatment options for patients with advanced prostate **cancer** (PCa) remain limited. Improved understanding of the underlying molecular drivers of prostate **cancer** pathogenesis, progression and resistance development has provided the fundamental basis for rational targeted drug design.

AREAS COVERED IN THIS REVIEW: This review will discuss the most recent developments in the field of prostate **cancer** therapies including key findings such as the identification of ETS gene rearrangements, the dissection of prostate **cancer** molecular heterogeneity and the discovery that castration-resistant prostate **cancer** (CRPC) remains androgen-driven despite the androgen-depleted milieu, thus making androgen receptor signaling a continued focus of molecularly targeted treatments. A multitude of new molecularly targeted agents are in clinical development and are highly likely to change the current treatment paradigm.

WHAT THE READER WILL GAIN: This review will outline the current clinical development of molecular targeted treatments in CRPC.

TAKE HOME MESSAGE: Unraveling the complex molecular biology that underpins this heterogeneous disease may pave the way to personalized therapy with a wide range of rationally targeted agents and combination treatments. In conclusion, we can predict that the rational clinical development of new targeted drugs will improve the outcome of men with prostate **cancer** in the years ahead.

L15 ANSWER 30 OF 101 MEDLINE ® on STN
ACCESSION NUMBER: 2011171242 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21294476
TITLE: Castration-refractory prostate **cancer**: new therapies, new questions.
AUTHOR: Appleman Leonard J
CORPORATE SOURCE: Division of Hematology-Oncology, Department of Medicine University of Pittsburgh, Pittsburgh, Pennsylvania, USA.

SOURCE: Oncology (Williston Park, N.Y.), **(2010 Dec)** Vol. 24, No. 14, pp. 1318-9, 1326.
Journal code: 8712059. ISSN: 0890-9091. L-ISSN: 0890-9091.
PUB. COUNTRY: United States
DOCUMENT TYPE: Commentary
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201102
ENTRY DATE: Entered STN: 8 Feb 2011
Last Updated on STN: 25 Feb 2011
Entered Medline: 24 Feb 2011

L15 ANSWER 31 OF 101 MEDLINE ® on STN
ACCESSION NUMBER: 2011171241 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21294475
TITLE: A renaissance in the medical treatment of advanced prostate **cancer**.
AUTHOR: Rove Kyle O; Flaig Thomas W
CORPORATE SOURCE: Division of Urology, University of Colorado Denver School of Medicine, Aurora, Colorado 80045, USA.
SOURCE: Oncology (Williston Park, N.Y.), **(2010 Dec)** Vol. 24, No. 14, pp. 1308-13, 1318.
Journal code: 8712059. ISSN: 0890-9091. L-ISSN: 0890-9091.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201102
ENTRY DATE: Entered STN: 8 Feb 2011
Last Updated on STN: 25 Feb 2011
Entered Medline: 24 Feb 2011
AB Prostate **cancer** will be diagnosed in one of six men during their lifetimes, and a small portion of these will progress after primary and salvage therapies. For many years, there were few treatment options for these patients after routine hormonal maneuvers, and standard of care since the early 2000s has consisted primarily of docetaxel, which improved survival over the previous first-line therapy mitoxantrone. In recent years, however, new therapies have begun to emerge to treat this devastating form of prostate **cancer**. This review examines the mechanisms behind these therapeutics and the key trials seeking to validate their clinical use.

L15 ANSWER 32 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 2
ACCESSION NUMBER: 2011:228149 HCAPLUS
DOCUMENT NUMBER: 155:58672
TITLE: **Cabazitaxel**: Filling one of the gaps in the **treatment** of prostate **cancer**
AUTHOR(S): Figg, William D., II; Figg, William D., Sr.
CORPORATE SOURCE: National Cancer Institute, National Institutes of Health (USA), Bethesda, MD, USA
SOURCE: Cancer Biology & Therapy (**2010**), 10(12), 1233-1234
CODEN: CBTAAO; ISSN: 1538-4047
PUBLISHER: Landes Bioscience
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Prostate **cancer** is the second most frequently diagnosed **cancer** in men and the fifth most common **cancer** overall. Definitive **therapy** (surgery or radiation) is highly effective, but if the **tumor** escapes the gland, **treatment** options are limited. For this population

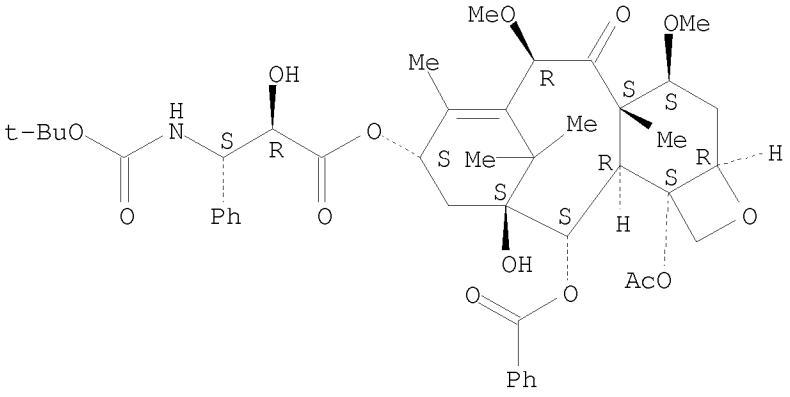
of patients, and androgen suppression is the cornerstone of initial **therapy**. Furthermore, progression to castration-resistant prostate **cancer** (CRPC) is inevitable. The current front-line **treatment** for patients with CRPC is the **chemotherapeutic** agent docetaxel (administered every 3 wk). Until now, it is the only agent that has been shown prolong survival in CRPC. The approval trial for docetaxel found a median overall survival of 19.2 mo for patients receiving docetaxel plus prednisone compared to 16.3 mo for patients receiving mitoxantrone plus prednisone. Mitoxantrone plus prednisone is often utilized for its palliative benefits, but two randomized trials failed to demonstrate a survival advantage. Cabazitaxcl is a tubulin-binding taxane (a partially synthesized derivative 10-deacetylbaccatin III, the major natural taxoid derived from Pacific yew tree), which showed activity in docetaxel-resistant **cancer** cell lines. It was selected for clin. development primarily due poor affinity for the ATP-dependent **drug** efflux pump P-glycoprotein (P-gp; also known as ABCB1). **Cabazitaxel** resulted in a 2.4 mo improvement in overall survival. **Cabazitaxel** provides an important addition to our **treatment** armamentarium for **treating** metastatic CRPC in the post-docetaxel setting. The one question left unanswered by the trial above is the definition of docetaxel resistance.

IT **183133-96-2, Cabazitaxel**
RL: ADV (Adverse effect, including toxicity); **PAC (Pharmacological activity)**; **THU (Therapeutic use)**; BIOL (Biological study);
USES (Uses)
(**cabazitaxel** - filling one of gaps in **treatment** of prostate **cancer**)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 15 THERE ARE 15 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 33 OF 101 MEDLINE ® on STN DUPLICATE 3
ACCESSION NUMBER: 2010694352 MEDLINE
DOCUMENT NUMBER: PubMed ID: 20651307
TITLE: New taxane treats advanced prostate **cancer**.
AUTHOR: Thompson Cheryl A
SOURCE: American journal of health-system pharmacy : AJHP :

official journal of the American Society of Health-System Pharmacists, **(2010 Aug)** Vol. 67, No. 15, pp. 1220. Journal code: 9503023. E-ISSN: 1535-2900. L-ISSN: 1079-2082.
DIGITAL OBJECT IDENTIFIER: 10.2146/news100052
PUB. COUNTRY: United States
DOCUMENT TYPE: News Announcement
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201011
ENTRY DATE: Entered STN: 24 Jul 2010
Last Updated on STN: 17 Nov 2010
Entered Medline: 16 Nov 2010

L15 ANSWER 34 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 2010:1238015 HCAPLUS
DOCUMENT NUMBER: 155:29782
TITLE: Prednisone plus **cabazitaxel** or mitoxantrone for metastatic castration-resistant prostate **cancer** progressing after docetaxel **treatment**: a randomised open-label trial
AUTHOR(S): de Bono, Johann Sebastian; Oudard, Stephane; Ozguroglu, Mustafa; Hansen, Steinbjorn; Machiels, Jean-Pascal; Kocak, Ivo; Gravis, Gwenaelle; Bodrogi, Istvan; Mackenzie, Mary J.; Shen, Liji; Roessner, Martin; Gupta, Sunil; Sartor, A. Oliver
CORPORATE SOURCE: Royal Marsden NHS Foundation Trust and The Institute of Cancer Research, Sutton, UK
SOURCE: Lancet (**2010**), 376(9747), 1147-1154
CODEN: LANCAO; ISSN: 0140-6736
PUBLISHER: Elsevier Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Cabazitaxel** is a novel tubulin-binding taxane **drug** with **antitumor** activity in docetaxel-resistant **cancers**. We aimed to compare the efficacy and safety of **cabazitaxel** plus prednisone with those of mitoxantrone plus prednisone in men with metastatic castration-resistant prostate **cancer** with progressive disease after docetaxel-based **treatment**. We undertook an open-label randomised phase 3 trial in men with metastatic castration-resistant prostate **cancer** who had received previous hormone **therapy**, but whose disease had progressed during or after **treatment** with a docetaxel-containing regimen. Participants were **treated** with 10 mg oral prednisone daily, and were randomly assigned to receive either 12 mg/m2 mitoxantrone i.v. over 15-30 min or 25 mg/m2 **cabazitaxel** i.v. over 1 h every 3 wk. The random allocation schedule was computer-generated; patients and **treating** physicians were not masked to **treatment** allocation, but the study team was masked to the data anal. The primary endpoint was overall survival. Secondary endpoints included progression-free survival and safety. Anal. was by intention to **treat**. This study is registered at ClinicalTrials.gov, NCT00417079. 755 Men were allocated to **treatment** groups (377 mitoxantrone, 378 **cabazitaxel**) and were included in the intention-to-**treat** anal. At the cutoff for the final anal. (Sept 25, 2009), median survival was 15.1 mo (95% CI 14.1-16.3) in the **cabazitaxel** group and 12.7 mo (11.6-13.7) in the mitoxantrone group. The hazard ratio for death of men **treated** with **cabazitaxel** compared with those taking mitoxantrone was 0.70 (95% CI 0.59-0.83, $p < 0.0001$). Median progression-free survival was 2.8 mo (95% CI 2.4-3.0) in the **cabazitaxel** group and 1.4 mo (1.4-1.7) in the mitoxantrone group (HR 0.74, 0.64-0.86, $p < 0.0001$). The most common clin. significant grade 3 or higher adverse events were neutropenia (**cabazitaxel**, 303 [82%] patients vs mitoxantrone, 215 [58%]) and diarrhoea (23 [6%] vs one [< 1%]). 28 (8%) patients in the **cabazitaxel**

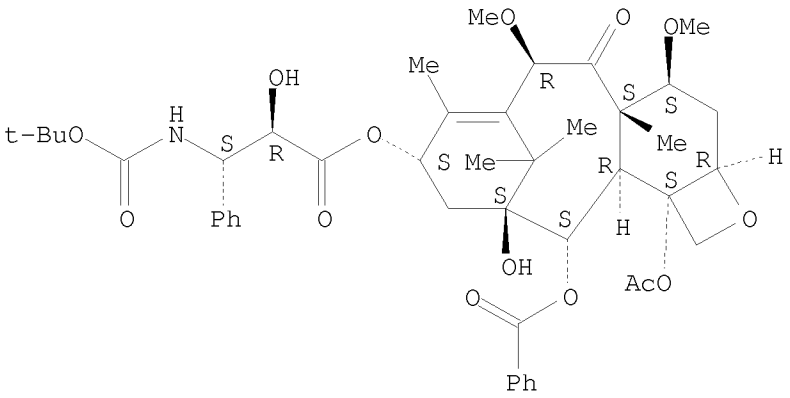
group and five (1%) in the mitoxantrone group had febrile neutropenia. **Treatment** with **cabazitaxel** plus prednisone has important clin. **antitumor** activity, improving overall survival in patients with metastatic castration-resistant prostate **cancer** whose disease has progressed during or after docetaxel-based **therapy**. Funding: Sanofi-Aventis.

IT **183133-96-2, Cabazitaxel**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(prednisone plus **cabazitaxel** compared to mitoxantrone showed **antitumor** activity and improved overall survival in patient with metastatic castration-resistant prostate **cancer** with progressive disease after docetaxel-based **treatment**)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 233 THERE ARE 233 CAPLUS RECORDS THAT CITE THIS RECORD (235 CITINGS)
REFERENCE COUNT: 20 THERE ARE 20 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 35 OF 101 MEDLINE ® on STN DUPLICATE 5
ACCESSION NUMBER: 2010869331 MEDLINE
DOCUMENT NUMBER: PubMed ID: 20888974
TITLE: **Cabazitaxel** in prostate **cancer**: stretching a string.
AUTHOR: Dorff Tanya B; Quinn David I
CORPORATE SOURCE: University of Southern California, Division of Oncology, Kenneth J Norris Comprehensive Cancer Center, Los Angeles, CA 90033, USA.
SOURCE: Lancet, **(2010 Oct 2)** Vol. 376, No. 9747, pp. 1119-20. Journal code: 2985213R. E-ISSN: 1474-547X. L-ISSN: 0140-6736.
DIGITAL OBJECT IDENTIFIER: 10.1016/S0140-6736(10)61510-3
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Commentary
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 201010

ENTRY DATE: Entered STN: 5 Oct 2010
Last Updated on STN: 20 Oct 2010
Entered Medline: 19 Oct 2010
OS.CITING REF COUNT: 1 There are 1 MEDLINE records that cite this record

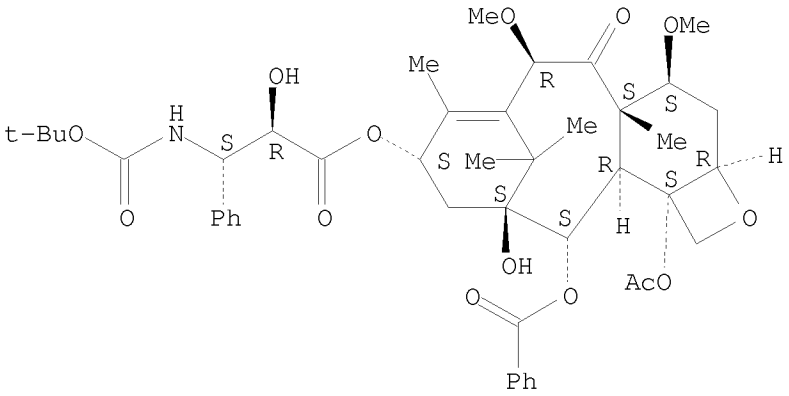
L15 ANSWER 36 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010556791 EMBASE
TITLE: **Cancer**: The revolution has begun.
SOURCE: The Lancet, (2 Oct 2010) Vol. 376, No. 9747, pp. 1117.
ISSN: 0140-6736 CODEN: LANCAO
PUBLISHER: Elsevier Limited, 32 Jamestown Road, London, NW1 7BY, United Kingdom.
PUBLISHER IDENT.: S 0140-6736(10)61518-8
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 016 Cancer
022 Human Genetics
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 20 Oct 2010
Last Updated on Embase: 20 Oct 2010

L15 ANSWER 37 OF 101 MEDLINE ® on STN DUPLICATE 6
ACCESSION NUMBER: 2010531683 MEDLINE
DOCUMENT NUMBER: PubMed ID: 20564864
TITLE: [New drugs at the horizon for men with prostate **cancer**]. Quels nouveaux medicaments a l'horizon pour te traitement du **cancer** avance de la prostate?.
AUTHOR: Shabafrouz Keyvan; Bauer Jean; Berthold Dominik R
CORPORATE SOURCE: Centre pluridisciplinaire d'oncologie CHUV, 1011 Lausanne.
SOURCE: Revue medicale suisse, **(2010 May 26)** Vol. 6, No. 250, pp. 1057-8, 1060-1.
Journal code: 101219148. ISSN: 1660-9379. L-ISSN: 1660-9379.
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201007
ENTRY DATE: Entered STN: 23 Jun 2010
Last Updated on STN: 23 Jul 2010
Entered Medline: 22 Jul 2010
AB Despite major progress in the understanding of biological mechanisms underlying metastatic prostate **cancer**, the treatment of men with advanced prostate **cancer** remains challenging. Several randomized controlled trials with promising or positive results are underway or just released. Here we discuss new treatments which might be used in clinic in the near future: hormonal treatments (Abiraterone and MDV3100), a new chemotherapy (**Cabazitaxel**), a cellular vaccine (Sipuleucel-T), anti-angiogenic drugs (Bevacizumab, Aflibercept), a new radioactive treatment (Alpharadin) and a new bone-protective agent (Deno-sumab).

L15 ANSWER 38 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 7
ACCESSION NUMBER: 2010:932635 HCAPLUS
DOCUMENT NUMBER: 154:350769
TITLE: Castration-resistant prostate **cancer**: current and emerging treatment strategies
AUTHOR(S): Di Lorenzo, Giuseppe; Buonerba, Carlo; Autorino, Riccardo; de Placido, Sabino; Sternberg, Cora N.
CORPORATE SOURCE: Cattedra di Oncologia Medica, Dipartimento di

Endocrinologia e Oncologia Molecolare e Clinica, Universita degli Studi Federico II, Naples, Italy
SOURCE: Drugs (**2010**), 70(8), 983-1000
CODEN: DRUGAY; ISSN: 0012-6667
PUBLISHER: Adis Data Information BV
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. Until very recently, docetaxel was the only approved agent in castration-resistant prostate **cancer** (CRPC) and other effective **therapeutic** options are urgently needed. In recent years, several new agents with promising activity and a favorable toxicity profile have been developed and clin. investigated in the fields of hormonal, cytotoxic, targeted and immune **therapy**. In particular, recent results from two large phase III trials of sipuleucel-T and **cabazitaxel** show that these two agents significantly prolong overall survival in CRPC. Indeed, sipuleucel-T has recently been approved by the US FDA for the **treatment** of CRPC. Many other **pharmaceuticals**, which are presented in this review, have been investigated recently or are being investigated in phase III trials and might prove to be effective in the future. Reviewed articles are discussed in light of the innovations in study design brought by the Prostate **Cancer** Clin. Trials Working Group (PCWG2), which updated the Prostate-Specific Antigen Working Group (PCWG1) guidelines, in order to allow better identification of potentially active drugs in clin. trials.
IT **183133-96-2, Cabazitaxel**
RL: BSU (Biological study, unclassified); **PAC (Pharmacological activity)**; **THU (Therapeutic use)**; BIOL (Biological study);
USES (Uses)
(current and emerging **treatment** strategies for castration-resistant prostate **cancer**)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



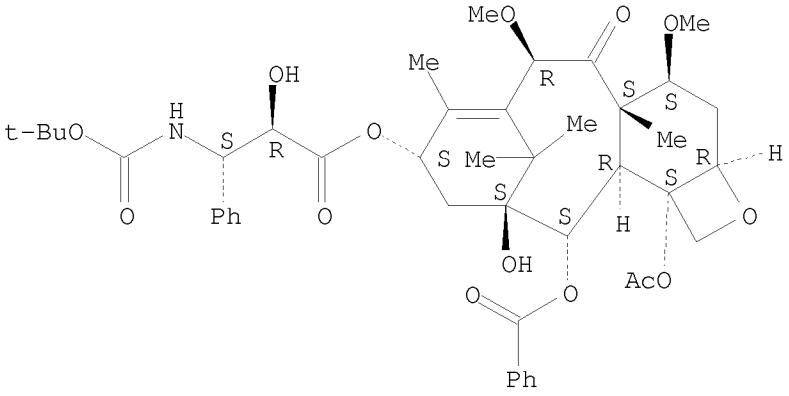
OS.CITING REF COUNT: 21 THERE ARE 21 CAPLUS RECORDS THAT CITE THIS RECORD (21 CITINGS)
REFERENCE COUNT: 68 THERE ARE 68 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 39 OF 101 EMBASE

reserved on STN
ACCESSION NUMBER: 2010502416 EMBASE
TITLE: How does sipuleucel-T alter our clinical practice?.
AUTHOR: George, Daniel (correspondence)
CORPORATE SOURCE: Divisions of Medical Oncology and Urology, Duke University Medical Center, Durham, NC, United States. daniel.george@duke.edu
SOURCE: BJU International, (October 2010) Vol. 106, No. 7, pp. 945-946.
Refs: 7
ISSN: 1464-4096; E-ISSN: 1464-410X CODEN: BJINFO
PUBLISHER: Blackwell Publishing Ltd, 9600 Garsington Road, Oxford, OX4 2XG, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 016 Cancer
017 Public Health, Social Medicine and Epidemiology
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 30 Sep 2010
Last Updated on Embase: 30 Sep 2010

L15 ANSWER 40 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2011:462676 HCAPLUS
DOCUMENT NUMBER: 155:647736
TITLE: Cabazitaxel: ipilimumab
AUTHOR(S): Knauth, M. Alan; Waddell, J. Aubrey; Solimando, Dominic A., Jr
CORPORATE SOURCE: Blount Memorial Hospital, Maryville, TN, USA
SOURCE: Hospital Pharmacy (**2010**), 45(11), 828, 830, 833-835
CODEN: HOPHAZ; ISSN: 0018-5787
PUBLISHER: Thomas Land Publishers, Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. The complexity of **cancer** chemotherapy requires pharmacists be familiar with the complicated regimens and highly toxic agents used. This column reviews various issues related to preparation, dispensing, and administration of **antineoplastic** therapy, and the agents, both com. available and investigational, used to treat malignant diseases.
IT **183133-96-2, Cabazitaxel**
RL: BSU (Biological study, unclassified); **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study);
USES (Uses)
(understanding various issues related to preparation, dispensing and administration of **antineoplastic cabazitaxel** and ipilimumab **therapy** may reduce complexity of **chemotherapy** used in **cancer**)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



REFERENCE COUNT: 14 THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 41 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2010644171 EMBASE

TITLE: Novel therapeutic strategies following docetaxel-based chemotherapy in castration-resistant **prostate cancer**.

AUTHOR: Fujimoto, Naohiro (correspondence); Matsumoto, Tetsuro

CORPORATE SOURCE: Department of Urology, School of Medicine, University of Occupational and Environmental Health, 1-1 Iseigaoka, Yahatanishi, Kitakyushu, 807-8555, Japan. n-fuji@med.uoeh-u.ac.jp

AUTHOR: Shiota, Masaki

CORPORATE SOURCE: Graduate School of Medical Sciences, Kyushu University, Fukuoka, Japan.

AUTHOR: Kubo, Tatsuhiko

CORPORATE SOURCE: Department of Public Health, University of Occupational and Environmental Health, Kitakyushu, Japan.

SOURCE: Expert Review of Clinical Pharmacology, (November 2010) Vol. 3, No. 6, pp. 785-795.
Refs: 66
ISSN: 1751-2433

PUBLISHER: Expert Reviews Ltd., 2 Albert Place, London, N3 1QB, United Kingdom.

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles

FILE SEGMENT:ClinicalTrials.gov

CLINICAL TRIAL NO.: NCT00003084; NCT00082693; NCT00097431; NCT00124566; NCT00414388; NCT00474383; NCT00485303; NCT00639690; NCT00676650

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered Embase: 10 Dec 2010
Last Updated on Embase: 10 Dec 2010

AB Prolonged survival of patients with castration-resistant **prostate cancer** has been demonstrated following treatment with a combination of docetaxel and prednisone. This combination has, therefore, become the standard first-line chemotherapy for castration-resistant **prostate cancer**. Median survival, however, does not exceed 20 months and there

are currently no approved second-line treatments for patients who progress after docetaxel treatment. The development of effective and safe treatment strategies is urgently required. Several clinical trials are currently evaluating the use of cytotoxic, antiandrogenic and molecular targeting agents. Preclinical studies are identifying the mechanisms responsible for docetaxel resistance and means of enhancing docetaxel activity. The results of these studies will provide the basis for rationally designed therapeutic approaches. This article summarizes the results of recent preclinical and clinical studies and discusses future perspectives. © 2010 Expert Reviews Ltd.

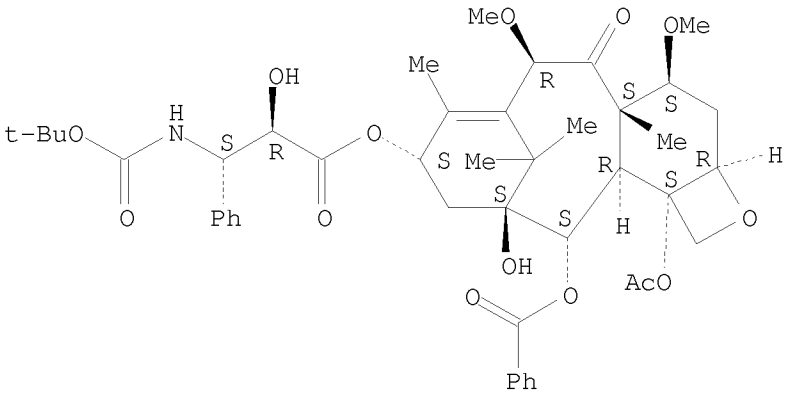
L15 ANSWER 42 OF 101 MEDLINE ® on STN DUPLICATE 8
ACCESSION NUMBER: 2010965562 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21071329
TITLE: New drugs: Sipuleucel-T, **cabazitaxel**, and collagenase clostridium histolyticum.
AUTHOR: Hussar Daniel A; Daniels W Layton
CORPORATE SOURCE: Philadelphia College of Pharmacy, University of the Sciences in Philadelphia, USA.
SOURCE: Journal of the American Pharmacists Association : JAPhA, **(2010 Nov-Dec)** Vol. 50, No. 6, pp. 772-5.
Journal code: 101176252. E-ISSN: 1544-3450. L-ISSN: 1086-5802.
DIGITAL OBJECT IDENTIFIER: 10.1331/JAPhA.2010.10542
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201103
ENTRY DATE: Entered STN: 13 Nov 2010
Last Updated on STN: 2 Mar 2011
Entered Medline: 1 Mar 2011

L15 ANSWER 43 OF 101 BIOSIS COPYRIGHT (c) 2013 The Thomson Corporation on STN DUPLICATE 9
ACCESSION NUMBER: 2010:191457 BIOSIS
DOCUMENT NUMBER: PREV201000191457
TITLE: Role of targeted therapy in the treatment of advanced prostate **cancer**.
AUTHOR(S): Fizazi, Karim; Sternberg, Cora N.; Fitzpatrick, John M. [Reprint Author]; Watson, R. William; Tabesh, Majid
CORPORATE SOURCE: Mater Misericordiae Univ Hosp, Dept Surg, Dublin 7, Ireland jfitzpatrick@mater.ie
SOURCE: BJU International, (**MAR 2010**) Vol. 105, No. 6, pp. 748-767.
ISSN: 1464-4096. E-ISSN: 1464-410X.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Apr 2010
Last Updated on STN: 7 Apr 2010
AB Over the past decade, the treatment of advanced prostate **cancer** has developed significantly, and perhaps the most dramatic shift came in 2004 with the demonstration that docetaxel-based chemotherapy significantly improved overall survival in patients with castration-resistant prostate **cancer**. This led to a significant expansion of the role of chemotherapy in the management of prostate **cancer**. In addition, there is now considerable progress being made in the development of more effective antiandrogens, cytochrome P17 inhibitors, novel chemotherapy regimens, targeted therapies, and immunotherapies that can complement existing therapies and may soon become integrated into the treatment paradigm. Progress in our understanding of molecular signalling pathways that play

an important role in prostate **cancer** has stimulated the investigation of targeted therapies, including antiangiogenic agents, bone-targeted agents, and specific inhibitors of key signalling molecules and chaperone proteins. For the most part, targeted agents are being combined with chemotherapy, similar to the approach taken in other solid **tumours**. Various therapeutic vaccine strategies also appear to have potential in the treatment of advanced prostate **cancer**. However, the development of new approaches to the treatment of prostate **cancer** presents many challenges that will demand collaboration and consensus building with respect to biomarkers for patient selection, clinical endpoints, and trial designs.

L15 ANSWER 44 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 10
ACCESSION NUMBER: 2011:1223824 HCAPLUS
DOCUMENT NUMBER: 156:604568
TITLE: Cabazitaxel, a new taxane with favorable properties
AUTHOR(S): Bouchet, B. P.; Galmarini, C. M.
CORPORATE SOURCE: ISPB, IFR62, INSERM U590, Centre Leon Berard, Universite Lyon 1, Lyon, Fr.
SOURCE: Drugs of Today (**2010**), 46(10), 735-742
CODEN: MDACAP; ISSN: 1699-3993
PUBLISHER: Prous Science
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. **Cabazitaxel** is a new taxane characterized by convenient administration, a favorable **pharmacokinetic** and safety profile and a decreased propensity for P-glycoprotein (Pgp)-mediated **drug** resistance. In preclin. studies **cabazitaxel** inhibited cell growth in a wide range of human **cancer** cell lines, including **tumor** models expressing Pgp. Phase I clin. trials established that the **cabazitaxel** side effect profile is similar to that reported for taxanes, with neutropenia and neuropathy being the most commonly reported toxicities. Further clin. studies have revealed that **cabazitaxel** is clin. active in women with taxane-resistant metastatic breast **cancer** and in men with metastatic castration-resistant prostate **cancer** previously **treated** with docetaxel. The TROPIC phase III trial concluded that, compared to mitoxantrone/prednisone, the combination **cabazitaxel**/prednisone conferred a statistically significantly longer overall survival in patients after **treatment** with a docetaxel-containing regimen, providing the basis for its FDA approval in 2010.
IT **183133-96-2, Cabazitaxel**
RL: BSU (Biological study, unclassified); **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**cabazitaxel**, a new taxane with favorable properties)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

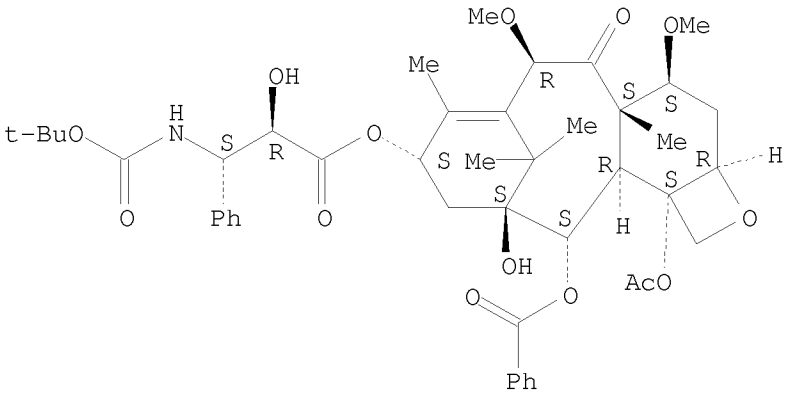
Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 8 THERE ARE 8 CAPLUS RECORDS THAT CITE THIS RECORD (8 CITINGS)
REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 45 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 11
ACCESSION NUMBER: 2010:1101273 HCAPLUS
DOCUMENT NUMBER: 154:350810
TITLE: Cabazitaxel
AUTHOR(S): Galsky, Matthew D.; Dritselis, Argyris; Kirkpatrick, Peter; Oh, William K.
CORPORATE SOURCE: Mount Sinai School of Medicine, New York, NY, 10029-6574, USA
SOURCE: Nature Reviews Drug Discovery (**2010**), 9(9), 677-678
CODEN: NRDDAG; ISSN: 1474-1776
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review. In June 2010, the taxane **anticancer drug cabazitaxel** (Jevtana; Sanofi-Aventis), in combination with prednisone, was approved by the US Food and **Drug** Administration (FDA) for the **treatment** of patients with hormone-refractory metastatic prostate **cancer** who had been previously **treated** with a regimen containing the taxane docetaxel.
IT **183133-96-2, Cabazitaxel**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(Jevtana; Jevtana plus prednisone may be effective in **treatment** of patient with hormone-refractory metastatic prostate **cancer** previously **treated** with docetaxel)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 26 THERE ARE 26 CAPLUS RECORDS THAT CITE THIS RECORD (26 CITINGS)
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 46 OF 101 MEDLINE ® on STN
ACCESSION NUMBER: 2011483480 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21542197
TITLE: Improved survival in second-line advanced prostate **cancer** treated with **cabazitaxel**.
AUTHOR: Anonymous
SOURCE: Nature reviews. Clinical oncology, **(2010 Dec)** Vol. 7, No. 12, pp. 671.
Journal code: 101500077. E-ISSN: 1759-4782. L-ISSN: 1759-4774.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Commentary
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; PUBMED-NOT-MEDLINE
ENTRY MONTH: 201105
ENTRY DATE: Entered STN: 5 May 2011
Last Updated on STN: 5 May 2011
Entered Medline: 4 May 2011

L15 ANSWER 47 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010659662 EMBASE
TITLE: Chemotherapy: Improved survival in second-line advanced **prostate cancer** treated with **cabazitaxel**.
AUTHOR: Richards, Lisa (correspondence)
SOURCE: Nature Reviews Clinical Oncology, (December 2010) Vol. 7, No. 12, pp. 671.
Refs: 1
ISSN: 1759-4774; E-ISSN: 1759-4782
PUBLISHER: Nature Publishing Group, Houndmills, Basingstoke, Hampshire, RG21 6XS, United Kingdom.
PUBLISHER IDENT.: NRCLINONC2010177
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 6 Jun 2013

Last Updated on Embase: 6 Jun 2013

L15 ANSWER 48 OF 101 MEDLINE ® on STN DUPLICATE 12
ACCESSION NUMBER: 2011016107 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21124035
TITLE: Castration refractory prostate **cancer**: cinderella finally comes to the ball.
AUTHOR: Chowdhury Simon; Harper Peter; Powles Thomas
SOURCE: Onkologie, **(2010)** Vol. 33, No. 12, pp. 655-6. Electronic Publication Date: 29 Nov 2010
Journal code: 7808556. E-ISSN: 1423-0240. L-ISSN: 0378-584X.
DIGITAL OBJECT IDENTIFIER: 10.1159/000322635
PUB. COUNTRY: Switzerland
DOCUMENT TYPE: Editorial
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201105
ENTRY DATE: Entered STN: 7 Dec 2010
Last Updated on STN: 5 May 2011
Entered Medline: 4 May 2011

L15 ANSWER 49 OF 101 MEDLINE ® on STN DUPLICATE 13
ACCESSION NUMBER: 2011071947 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21188774
TITLE: Prostate **cancer**: **Cabazitaxel** boosts post-docetaxel survival.
AUTHOR: Richards Lisa
SOURCE: Nature reviews. Urology, **(2010 Dec)** Vol. 7, No. 12, pp. 645.
Journal code: 101500082. E-ISSN: 1759-4820. L-ISSN: 1759-4812.
DIGITAL OBJECT IDENTIFIER: 10.1038/nrurol.2010.200
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Commentary
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: NONMEDLINE; PUBMED-NOT-MEDLINE
ENTRY MONTH: 201104
ENTRY DATE: Entered STN: 29 Dec 2010
Last Updated on STN: 6 Apr 2011
Entered Medline: 5 Apr 2011

L15 ANSWER 50 OF 101 BIOSIS COPYRIGHT (c) 2013 The Thomson Corporation on STN
ACCESSION NUMBER: 2010:555904 BIOSIS
DOCUMENT NUMBER: PREV201000555904
TITLE: Docetaxel Rechallenge in Castration-Resistant Prostate **Cancer**: Scientific Legitimacy of Common Clinical Practice.
AUTHOR(S): Buonerba, Carlo; Palmieri, Giovannella; Di Lorenzo, Giuseppe [Reprint Author]
CORPORATE SOURCE: Univ Naples Federico 2, Dipartimento Endocrinol and Oncol Mol and Clin, Cattedra Oncol Med, Naples, Italy
giuseppedilorenzoncol@hotmail.com
SOURCE: European Urology, (**OCT 2010**) Vol. 58, No. 4, pp. 636-637.
CODEN: EUURAV. ISSN: 0302-2838.
DOCUMENT TYPE: Letter
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Oct 2010
Last Updated on STN: 20 Oct 2010

L15 ANSWER 51 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights

reserved on STN
ACCESSION NUMBER: 2011024187 EMBASE
TITLE: **Cabazitaxel** and prednisone as second-line therapy of metastatic, castration-resistant **prostate cancer**.
AUTHOR: Jana, Bagi R. P.
CORPORATE SOURCE: University of Central Florida College of Medicine, Orlando, FL, United States.
AUTHOR: Jana, B. R. P. (correspondence)
CORPORATE SOURCE: University of Central Florida College of Medicine, Orlando, FL, United States.
SOURCE: Community Oncology, (December 2010) Vol. 7, No. 12, pp. 540-542.
Refs: 1
ISSN: 1548-5315
PUBLISHER: Elsevier Oncology, 46 Green Street, 2nd Floor, Huntington, NY 11743, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 20 Jan 2011
Last Updated on Embase: 20 Jan 2011
AB A novel taxane improves progression-free and overall survival of patients with metastatic, hormone-refractory **prostate cancer** after failure of docetaxel therapy. © 2010 Elsevier Inc.

L15 ANSWER 52 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010493195 EMBASE
TITLE: Therapeutic targeting of the **prostate cancer** microenvironment.
AUTHOR: Karlou, Maria; Tzelepi, Vassiliki; Efstathiou, Eleni (correspondence)
CORPORATE SOURCE: Department of Genitourinary Medical Oncology, David H. Koch Center for Applied Research of Genitourinary Cancers, University of Texas, P. O. Box 301439, Houston, TX 77230-1439, United States. eefstathiou@mdanderson.org
SOURCE: Nature Reviews Urology, (September 2010) Vol. 7, No. 9, pp. 494-509.
Refs: 196
ISSN: 1759-4812; E-ISSN: 1759-4820
PUBLISHER: Nature Publishing Group, Houndmills, Basingstoke, Hampshire, RG21 6XS, United Kingdom.
PUBLISHER IDENT.: NRUROL.2010.134
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
016 Cancer
022 Human Genetics
028 Urology and Nephrology
037 Drug Literature Index
FILE SEGMENT:ClinicalTrials.gov
CLINICAL TRIAL NO.: NCT00072930; NCT00103337; NCT00121238; NCT00321646; NCT00348998; NCT00349557; NCT00439270; NCT00473746; NCT00510718; NCT00513071; NCT00537381; NCT00544440; NCT00570700; NCT00607724; NCT00631527; NCT00831792; NCT00959946; NCT00974311; NCT01023061; NCT01163084
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 29 Sep 2010
Last Updated on Embase: 29 Sep 2010

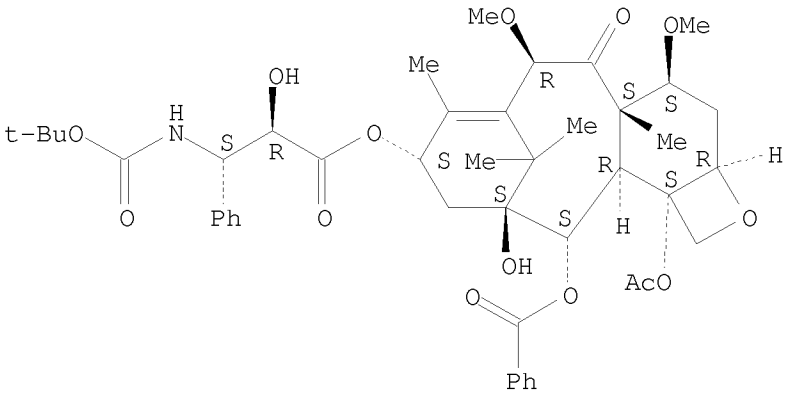
AB Solid **tumors** can be thought of as multicellular 'organs' that consist of a variety of cells as well as a scaffold of noncellular matrix. Stromal–epithelial crosstalk is integral to **prostate cancer** progression and metastasis, and androgen signaling is an important component of this crosstalk at both the primary and metastatic sites. **Intratumoral** production of androgen is an important mechanism of castration resistance and has been the focus of novel therapeutic approaches with promising results. Various other pathways are important for stromal–epithelial crosstalk and represent attractive candidate therapeutic targets. Hedgehog signaling has been associated with **tumor** progression, growth and survival, while Src family kinases have been implicated in **tumor** progression and in regulation of **cancer** cell migration. Fibroblast growth factors and transforming growth factor β signaling regulate cell proliferation, apoptosis and angiogenesis in the **prostate cancer** microenvironment. Integrins mediate communication between the cell and the extracellular matrix, enhancing growth, migration, invasion and metastasis of **cancer** cells. The contribution of stromal–epithelial crosstalk to **prostate cancer** initiation and progression provides the impetus for combinatorial microenvironment-targeting strategies. © 2010 Macmillan Publishers Limited. All rights reserved.

ACCESSION NUMBER: 2010637442 EMBASE
TITLE: Polysorbate 80 hypersensitivity reactions: A renewed call to action.
AUTHOR: Norris, LeAnn B.; Qureshi, Zaina P.; Brandon Bookstaver, P.; Bennett, Charles L.
CORPORATE SOURCE: South Carolina Center of Economic Excellence for Medication Safety and Efficacy, The Southern Network on Adverse Reactions (SONAR), South Carolina College of Pharmacy, University of South Carolina, CLS 314C, 715 Sumter Street, Columbia, SC 29208, United States. norris@sccp.sc.edu
AUTHOR: Raisch, Dennis W.
CORPORATE SOURCE: Pharmacoeconomics, Epidemiology, Public Policy and Outcomes Research (PEPPOR), College of Pharmacy, University of New Mexico, Albuquerque, NM, United States.
AUTHOR: Sartor, Oliver
CORPORATE SOURCE: Tulane Cancer Center, Tulane University, New Orleans, LA, United States.
AUTHOR: Chen, Hao; Chen, Fei
CORPORATE SOURCE: eHealthMe, Madison, WI, United States.
AUTHOR: Bennett, Charles L.
CORPORATE SOURCE: Hollings Cancer Center, Medical University of South Carolina, Charleston, SC, United States.
AUTHOR: Norris, L. B. (correspondence)
CORPORATE SOURCE: South Carolina Center of Economic Excellence for Medication Safety and Efficacy, The Southern Network on Adverse Reactions (SONAR), South Carolina College of Pharmacy, University of South Carolina, CLS 314C, 715 Sumter Street, Columbia, SC 29208, United States. norris@sccp.sc.edu
SOURCE: Community Oncology, (September 2010) Vol. 7, No. 9, pp. 425-428.
Refs: 7
ISSN: 1548-5315
PUBLISHER: Elsevier Oncology, 46 Green Street, 2nd Floor, Huntington, NY 11743, United States.
COUNTRY: United States

DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LANGUAGE: English
ENTRY DATE: Entered Embase: 7 Dec 2010
Last Updated on Embase: 7 Dec 2010

L15 ANSWER 54 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 14
ACCESSION NUMBER: 2011:1532 HCAPLUS
DOCUMENT NUMBER: 154:124221
TITLE: Critical appraisal of cabazitaxel in the management of advanced prostate **cancer**
AUTHOR(S): Pal, Sumanta Kumar; Twardowski, Przemyslaw; Sartor, Oliver
CORPORATE SOURCE: Division of Genitourinary Malignancies, Department of Medical Oncology and Experimental Therapeutics, City of Hope Comprehensive Cancer Center, Los Angeles, CA, USA
SOURCE: Clinical Interventions in Aging (**2010**), 5, 395-402
CODEN: CIALBC; ISSN: 1178-1998
URL: http://www.dovepress.com/getfile.php?fileID=8270
PUBLISHER: Dove Medical Press Ltd.
DOCUMENT TYPE: Journal; General Review; (online computer file)
LANGUAGE: English
AB A review. Docetaxel remains a cornerstone of **therapy** for the patient with metastatic castration-resistant prostate **cancer** (CRPC). However, the landscape of CRPC **therapy** is changing rapidly - recently, data from the phase III TROPIC study revealed a survival advantage with the novel taxane **cabazitaxel**/prednisone (compared with mitoxantrone/prednisone) in a cohort of 755 men with docetaxel-refractory metastatic CRPC. Interestingly, **cabazitaxel** bears substantial structural similiarity to docetaxel but appears to be mechanistically distinct. In preclin. studies, the agent has **antitumor** activity in a variety of docetaxel-refractory in vitro and in vivo models. Subsequent to phase I testing in advanced solid **tumors** (where neutropenia was identified as a dose-limiting toxicity), the agent was assessed in a phase II trial in advanced, taxane-refractory breast **cancer** and in the aforementioned phase III TROPIC study. This review describes in detail the preclin. and clin. development of **cabazitaxel**.
IT **183133-96-2, Cabazitaxel**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**cabazitaxel** may be effective for **treatment** of patient with taxane-refractory metastatic prostate **cancer**)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 7 THERE ARE 7 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
REFERENCE COUNT: 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 55 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010637447 EMBASE
TITLE: The conundrum of sipuleucel-T for **prostate cancer**.
AUTHOR: Knopf, Kevin B.
CORPORATE SOURCE: California Pacific Medical Center, San Francisco, CA, United States.
AUTHOR: Knopf, K. B. (correspondence)
CORPORATE SOURCE: California Pacific Medical Center, San Francisco, CA, United States.
SOURCE: Community Oncology, (August 2010) Vol. 7, No. 8, pp. 343-344.
Refs: 3
ISSN: 1548-5315
PUBLISHER: Elsevier Oncology, 46 Green Street, 2nd Floor, Huntington, NY 11743, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
028 Urology and Nephrology
036 Health Policy, Economics and Management
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 2 Dec 2010
Last Updated on Embase: 2 Dec 2010

L15 ANSWER 56 OF 101 BIOSIS COPYRIGHT (c) 2013 The Thomson Corporation on STN DUPLICATE 15
ACCESSION NUMBER: 2010:403164 BIOSIS
DOCUMENT NUMBER: PREV201000403164
TITLE: Castration-Refractory Prostate **Cancer**: New Drugs in the Pipeline.
AUTHOR(S): Schrijvers, Dirk [Reprint Author]; Van Erps, Peter; Cortvriend, Jim
CORPORATE SOURCE: Ziekenhuisnetwerk Antwerpen Middelheim, Dept Hematooncol, Lindendreef 1, B-2020 Antwerp, Belgium
dirk.schrijvers@zna.be
SOURCE: Advances in Therapy, (**MAY 2010**) Vol. 27, No. 5, pp. 285-296.

ISSN: 0741-238X. E-ISSN: 1865-8652.
DOCUMENT TYPE: Article
General Review; (Literature Review)
LANGUAGE: English
ENTRY DATE: Entered STN: 14 Jul 2010
Last Updated on STN: 28 Jul 2010

AB The standard treatment for patients with castration-refractory prostate **cancer** (CRPC) is the combination docetaxel-prednisone, if the patient can support chemotherapy. Several new treatments have been tested in chemotherapy-naive or docetaxel-pretreated patients with CRPC. Some of these treatments have shown activity in first-line and second-line treatment. In this review, an update is given of new treatment studies performed in patients with CRPC.

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ACCESSION NUMBER: 0050297302 EMBASE
TITLE: **Cabazitaxel** plus prednisone/prednisolone significantly increases overall survival compared to mitoxantrone plus prednisone/ prednisolone in patients with metastatic castration-resistant **prostate cancer** (mCRPC) previously treated with docetaxel: Final results with updated overall survival of a multinational phase III trial (tropic).
AUTHOR: Oudard, S.M. (correspondence)
CORPORATE SOURCE: Medical Oncology Hopital Europeen Georges Pompidou, Paris, France.
AUTHOR: De Bono, J.S.
CORPORATE SOURCE: Royal Marsden National Health Service Foundation Trust, Institute of Cancer Research, Sutton, United Kingdom.
AUTHOR: Ozguroglu, M.
CORPORATE SOURCE: Istanbul University, Istanbul, Turkey.
AUTHOR: Hansen, S.
CORPORATE SOURCE: Odense University Hospital, Odense, Denmark.
AUTHOR: Machiels, J.
CORPORATE SOURCE: Oncology Medical, Cliniques Universitaires Saint-Luc, UniversiteCatholique de Louvain, Brussels, Belgium.
AUTHOR: Shen, L.; Gupta, S.
CORPORATE SOURCE: Sanofi-Aventis, Malvern, United States.
AUTHOR: Sartor, A.O.
CORPORATE SOURCE: Tulane University, New Orleans, United States.
SOURCE: Annals of Oncology, (October 2010) Vol. 21, Supp. SUPPL. 8, pp. viii272. Abstract Number: 871PD.
Meeting Info: 35th ESMO Congress. Milan, Italy. 08 Oct 2010-12 Oct 2010
ISSN: 0923-7534
PUBLISHER: Oxford University Press.
DOCUMENT TYPE: Journal; Conference; (Conference Abstract)
FILE SEGMENT: CONF
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: Sep 2012
Last Updated on Embase: Sep 2012

AB Background: Treatment of mCRPC following docetaxel (D) therapy failure due to progressive disease (PD) is an unmet medical need. TROPIC trial evaluated the efficacy and safety of a novel chemotherapy **cabazitaxel** (Cbz) in men with mCRPC previously treated with D. Methods: Men with mCRPC, ECOG PS 0-2, and adequate organ function who had prior hormone therapy, chemotherapy, and radiotherapy, but had PD during or after D (cumulative dose ≥225 mg/m 2) were randomized to 10 mg/day of prednisone/ prednisolone with either mitoxantrone 12 mg/m 2 (MP) or Cbz 25 mg/m 2 (CbzP), both administered 3-weekly. The primary endpoint was overall survival (OS). Secondary endpoints: progression-free survival;

response; time to progression (TTP) for **tumor**, PSA, pain; and safety. The study had 90% power to detect a 25% lower hazard rate for death in the CbzP group after 511 events (2-sided α= 0.05). Results: From Jan 2007 to Oct 2008, 755 men (median age 68 yr) were randomized. Patients' characteristics were well balanced. Median prior D dose was 577 mg/m2 for CbzP and 529 mg/m 2 for MP. Median follow-up was 13.7 months in the study. Median number of cycles was 6 for CbzP and 4 for MP. In the primary ITT analysis, the CbzP group had a statistically significantly longer OS 15.1 months compared with 12.7 with MP (HR 0.72; 95% CI, 0.61 - 0.84; P <0.0001). Multivariate analysis of OS after adjusting prognostic factors was in favor of CbzP. PFS, response rates, and TTP (by RECIST and PSA) also statistically significantly favored CbzP. Despite longer treatment with CbzP no worsening in ECOG PS was seen. Present Pain Intensity score improved in 21% of men in CbzP vs. 18% in MP arm. Analgesic use between the two treatment groups was comparable. Most frequent Grade 3/4 toxicity was neutropenia (82% CbzP; 58% MP); rates of febrile neutropenia were 7.5% and 1.3%, respectively. Conclusions: Men with mCRPC progressing after D benefit from CbzP treatment with longer OS, PFS, TTP by **tumor** assessments and PSA, and higher response rates.

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ACCESSION NUMBER: 2010242830 EMBASE
TITLE: Update on castrate-resistant **prostate cancer**: 2010.
AUTHOR: Lassi, Kiran (correspondence); Dawson, Nancy A.
CORPORATE SOURCE: Lombardi Comprehensive Cancer Center, Georgetown University Hospital, Washington, DC, United States. kiranlassi@yahoo.com
AUTHOR: Lassi, Kiran (correspondence)
CORPORATE SOURCE: 3800 Reservoir Road NW, Washington, DC 20007, United States. kiranlassi@yahoo.com
SOURCE: Current Opinion in Oncology, (May 2010) Vol. 22, No. 3, pp. 263-267.
Refs: 35
ISSN: 1040-8746 CODEN: CUOOE8
PUBLISHER: Lippincott Williams and Wilkins, 530 Walnut Street, Philadelphia, PA 19106-3621, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 14 May 2010
Last Updated on Embase: 14 May 2010

AB PURPOSE OF REVIEW: **Prostate cancer** remains a medical dilemma and a major cause of morbidity and mortality in many western countries. It represents the most common **cancer** in US men, with an estimated 192 280 new cases diagnosed in 2009. The median survival for men with metastatic castrate-resistant **prostate cancer** is 1-2 years, with improvements in survival seen primarily with docetaxel-based therapies. The purpose of this article is to discuss developments of novel agents in the field of metastatic castration-resistant **prostate cancer** (CRPC), including new cytotoxic agents, immune-based therapies, circulating **tumor** markers and targeting agents. RECENT FINDINGS: During this past year, several promising approaches yielded disappointing results in the phase III setting (GVAX); nonetheless, expectations for other agents (Abiraterone, MDV3100, Zibotentan, immunotherapy agents) still remain high. SUMMARY: Systemic therapy options are limited in CRPC and survival benefit remains

to be seen with the new therapies. Circulating **tumor** cells continue to provide important prognostic information and will likely become an important aspect of future clinical decision-making. © Lippincott Williams & Wilkins 2010.

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ACCESSION NUMBER: 2010155572 EMBASE
TITLE: The role of docetaxel based therapy for **prostate cancer** in the era of targeted medicine: Review article.
AUTHOR: Sonpavde, Guru
CORPORATE SOURCE: Texas Oncology, United States.
AUTHOR: Sonpavde, Guru
CORPORATE SOURCE: Veterans Affairs Medical Center, Baylor College of Medicine, Houston, TX, United States.
AUTHOR: Sternberg, Cora N
CORPORATE SOURCE: San Camillo and Forlanini Hospitals, Nuovi Padiglione IV, Circonvallazione Gianicolense 87, 00152 Rome, Italy. csternberg@scamilloforlanini.rm.it
AUTHOR: Sternberg, C. N. (correspondence)
CORPORATE SOURCE: San Camillo and Forlanini Hospitals, Nuovi Padiglione IV, Circonvallazione Gianicolense 87, 00152 Rome, Italy. csternberg@scamilloforlanini.rm.it
SOURCE: International Journal of Urology, (March 2010) Vol. 17, No. 3, pp. 228-240.
Refs: 97
ISSN: 0919-8172; E-ISSN: 1442-2042 CODEN: IJURF3
PUBLISHER: Blackwell Publishing, 550 Swanston Street, Carlton South, VIC 3053, Australia.
COUNTRY: Australia
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 038 Adverse Reactions Titles
037 Drug Literature Index
030 Clinical and Experimental Pharmacology
028 Urology and Nephrology
023 Nuclear Medicine
016 Cancer
006 Internal Medicine
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 20 Apr 2010
Last Updated on Embase: 20 Apr 2010

AB Docetaxel based chemotherapy has been shown to modestly extend life, relieve pain and improve the quality of life in patients with metastatic castration-resistant **prostate cancer**. Current trials are attempting to build on the backbone of docetaxel by combining it with novel biological agents. Trials are also investigating the role of docetaxel for earlier stages of **prostate cancer**. No standard second-line systemic therapy exists and such patients are candidates for clinical trials. The increased understanding of the mechanisms of progressive castration-resistant **prostate cancer** is being translated into an increasing pipeline of novel therapies. © 2010 The Japanese Urological Association.

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ACCESSION NUMBER: 2011081076 EMBASE
TITLE: [Research in therapeutic targets in **prostate cancer**]. Lineas de investigacion y nuevas dianas terapeuticas en **cancer** de prostata.
AUTHOR: Cassinello Espinosa, Javier (correspondence); Holgado Martin, E.; Mohedano Mohedano, N.

CORPORATE SOURCE: Servicio de Oncologia Medica, Hospital Universitario de Guadalajara, C/ Donantes de Sangre, s/n, 19002 Guadalajara, Spain. jacaes@sescam.jccm.es
AUTHOR: Gonzalez De Alba Baamonde, A.
CORPORATE SOURCE: Servicio de Oncologia Medica, Hospital Universitario Son Dureta, Palma de Mallorca, Spain.
SOURCE: Revisiones en Cancer, (2010) Vol. 24, No. 4, pp. 191-199.
Refs: 42
ISSN: 0213-8573 CODEN: RECAER
PUBLISHER: ARAN Ediciones S.A., Castello 128 - 1o, Madrid, 28006, Spain.
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 006 Internal Medicine
016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: Spanish; Castilian
SUMMARY LANGUAGE: English; Spanish; Castilian
ENTRY DATE: Entered Embase: 23 Feb 2011
Last Updated on Embase: 23 Feb 2011
AB **Prostate cancer** is one of the most common **cancers** in men in USA and in European countries. Although hormone-based androgen deprivation therapy is the main therapeutic approach in advanced disease and results in rapid responses, all patients eventually develop progressive castration-resistant disease state. The median survival in patients with castration-resistant **prostate cancer** (CRPC) is about 1-2 years, mostly seen with docetaxel-based regimens. **Cabazitaxel**, a new semi synthetic taxane, emerges as a new standard in second-line chemotherapy in docetaxel-resistant CRPC patients with survival benefit. Despite this major advanced, novel approaches including important molecular targeted therapy continue to be investigated in an attempt to further improve survival: androgen biosynthesis inhibitor abiraterone acetate, androgen receptor antagonist MDV3100, endothelin receptor antagonist atrasentan, dasatinib as SRC inhibitor, antiangiogenics agents as bezacizumab, sunitinib or sorafenib, oligonucleotid antisense against clusterin such as custirsen, the immune-based strategies including dendritic cell vaccines such as sipuleucel and monoclonal antibodies against **tumor** antigens as ipilumumab. Finally, circulating **tumor** cells (CTC) might be useful as an intermediate endpoint of survival in the future. Copyright © 2010 Aran Ediciones, S. L.

ACCESSION NUMBER: 2011202822 EMBASE
TITLE: Practice changing data and new developments in the management of **prostate cancer** - ASCO 2010.
AUTHOR: De Santis, Maria (correspondence); Bachner, M.
CORPORATE SOURCE: Kaiser Franz Josef Hospital, ACR-ITR VIEnna/CEADDP, LBI-ACR VIEnna-CTO, Vienna, Austria. maria.desantis@wienkav.at
AUTHOR: De Santis, Maria (correspondence)
CORPORATE SOURCE: 3rd Medical Department, Center for Oncology and Hematology, Kaiser Franz Josef Spital, SMZ Sud, Kundratstrasse 3, 1100 Wien, Austria. maria.desantis@wienkav.at
SOURCE: Memo - Magazine of European Medical Oncology, (December 2010) Vol. 3, No. 4, pp. 164-166.
Refs: 13
ISSN: 1865-5041; E-ISSN: 1865-5076
PUBLISHER: Springer Wien, Sachsenplatz 4-6, P.O. Box 89, Vienna, A-1201, Austria.
COUNTRY: Austria
DOCUMENT TYPE: Journal; General Review; (Review)

FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 28 Apr 2011
Last Updated on Embase: 28 Apr 2011

AB In the 2010 ASCO meeting valuable information and even practice-changing results for the medical treatment of castration resistant **prostate cancer** (CRPC) were presented. Focus was put on bone-targeted treatment, chemotherapy for second line use, combinations of standard chemotherapy with different new drugs and data on a novel antiangiogenic compounds. Denosumab was shown to be superior to zoledronic acid in the treatment of patients with bone metastases of CRPC. **Cabazitaxel** improved survival in CRPC patients who progressed during or after docetaxel. This substance might become the first standard treatment for second line use. There are still concerns about the dose and toxicity. Both new drugs still need to be approved but will change our practice in the management of bone metastases and in the second line setting and enlarge significantly our small armamentarium of medical treatment of CRPC. So far, there is no proven benefit in adding any drug to standard docetaxel. This fact was confirmed by final data analyses of the combination of docetaxel with bevacizumab or calcitriol. Tasquinimod is a promising novel anti-angiogenic compound that delayed disease progression significantly in asymptomatic CRPC patients. A phase III-trial will reveal the true value of this compound. In conclusion, in the 2010 ASCO meeting, valuable new information concerning the medical treatment of CRPC was conveyed which might indeed change our clinical practice. © Springer-Verlag 2010.

ACCESSION NUMBER: 2010591556 EMBASE
TITLE: New approaches in hormone-resistant **prostate cancer**.
AUTHOR: Lassi, Kiran (correspondence); Dawson, Nancy A.
CORPORATE SOURCE: Lombardi Comprehensive Cancer Center, Georgetown University Hospital, Washington, DC, United States. kiran.lassi@gmail.com
AUTHOR: Lassi, Kiran (correspondence)
CORPORATE SOURCE: 3800 Reservoir Road NW, Washington, DC 20007, United States. kiran.lassi@gmail.com
SOURCE: Cancer and Chemotherapy Reviews, (July-September 2010) Vol. 5, No. 3, pp. 127-132.
Refs: 45
ISSN: 1885-740X
PUBLISHER: Publicaciones Permanyer, Mallorca 310, Barcelona, E-08037, Spain.
COUNTRY: Spain
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 12 Nov 2010
Last Updated on Embase: 12 Nov 2010

AB **Prostate cancer** remains the leading cause of **cancer** in men in the United States, with an estimated incidence of 192,280 cases and 27,360 deaths in 2009. Despite advances in hormonal and chemotherapeutic agents, the median survival for men with metastatic castrate-resistant **prostate cancer** is one to two years. This article is an update on novel

approaches under investigation in castration-resistant **prostate cancer**. © Permanyer Publications 2010.

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ACCESSION NUMBER: 2010619655 EMBASE
TITLE: Current management of castrate-resistant **prostate cancer**.
AUTHOR: Hotte, Sebastien J.
CORPORATE SOURCE: Department of Oncology, McMaster University, Juravinski Cancer Centre, 699 Concession Street, Hamilton, ON L8V 5C2, Canada. sebastien.hotte@jcc.hhsc.ca
AUTHOR: Saad, F.
CORPORATE SOURCE: Departments of Surgery and Urology, Centre Hospitalier de l'Universite de Montreal, Montreal, QC, Canada.
AUTHOR: Hotte, S. J. (correspondence)
CORPORATE SOURCE: Department of Oncology, McMaster University, Juravinski Cancer Centre, 699 Concession Street, Hamilton, ON L8V 5C2, Canada. sebastien.hotte@jcc.hhsc.ca
SOURCE: Current Oncology, (2010) Vol. 17, No. SUPPL. 2, pp. s72-s79.
Refs: 45
ISSN: 1198-0052 CODEN: CUONF6
PUBLISHER: Multimed Inc., 66 Martin Street, Milton, ONT L9T 2R2, Canada.
COUNTRY: Canada
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
033 Orthopedic Surgery
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 23 Nov 2010
Last Updated on Embase: 23 Nov 2010

AB **Prostate cancer** (PCa) is the most frequently diagnosed **cancer** in North America. Castrate-resistant pca presents a spectrum of disease ranging from rising PSA levels in the absence of metastases or symptoms and despite androgen-deprivation therapy, to metastases and significant debilitation from **cancer** symptoms. Castrate-resistant PCa is usually suspected in patients with new symptoms on androgen deprivation therapy, with a rising PSa, or with new evidence of disease on bone scans or computed tomography scans. Institution of treatment and the choice of systemic or local therapy depend on a number of factors. This review discusses the various currently available treatments for patients with castrate-resistant PCa, from secondary hormonal manipulations to options for postdocetaxel systemic therapy. © 2010 Multimed Inc.

L15 ANSWER 64 OF 101 MEDLINE ® on STN

ACCESSION NUMBER: 2010796036 MEDLINE
DOCUMENT NUMBER: PubMed ID: 20814400
TITLE: New treatments for metastic prostate **cancer**.
AUTHOR: Anonymous
SOURCE: The Medical letter on drugs and therapeutics, **(2010 Sep 6)** Vol. 52, No. 1346, pp. 69-70.
Journal code: 2985240R. E-ISSN: 1523-2859. L-ISSN: 0025-732X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 201010
ENTRY DATE: Entered STN: 4 Sep 2010
Last Updated on STN: 14 Oct 2010
Entered Medline: 13 Oct 2010
AB The FDA has approved 2 new treatments for castration-resistant (formerly called hormone-refractory) prostate **cancer**. Sipuleucel-T (Provenge - Dendreon) s the first immunotherapy approved for treatment of prostate **cancer**. **Cabazitaxel** (Jevtana - Sanofi-Aventis) is approved for second-line treatment of metastatic castration-resistant prostate **cancer** previously treated with docetaxel (Taxotere).

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ACCESSION NUMBER: 0020814400 EMBASE
COPYRIGHT: MEDLINE® is the source for the citation and abstract of this record.
TITLE: New treatments for metastic **prostate cancer**..
SOURCE: The Medical letter on drugs and therapeutics, (6 Sep 2010) Vol. 52, No. 1346, pp. 69-70.
E-ISSN: 1523-2859
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: MEDLINE
LANGUAGE: English
ENTRY DATE: Entered Embase: 21 Oct 2010
Last Updated on Embase: 21 Oct 2010
AB The FDA has approved 2 new treatments for castration-resistant (formerly called hormone-refractory) **prostate cancer**. Sipuleucel-T (Provenge - Dendreon) s the first immunotherapy approved for treatment of **prostate cancer**. **Cabazitaxel** (Jevtana - Sanofi-Aventis) is approved for second-line treatment of metastatic castration-resistant **prostate cancer** previously treated with docetaxel (Taxotere).

L15 ANSWER 66 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010651969 EMBASE
TITLE: [Advanced stage **prostate carcinoma**: Extended survival with new taxane].
Fortgeschrittenes prostatakarzinom: Verlangertes Uberleben mit neuem taxan.
AUTHOR: Jungmayr, Petra, Dr. (correspondence)
SOURCE: Deutsche Apotheker Zeitung, (11 Nov 2010) Vol. 150, No. 45, pp. 54-55.
ISSN: 0011-9857 CODEN: DAZEA2
PUBLISHER: Deutscher Apotheker Verlag, Birkenwaldstr.44,, Stuttgart, 70191, Germany.
COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 006 Internal Medicine
016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: German
ENTRY DATE: Entered Embase: 16 Dec 2010
Last Updated on Embase: 16 Dec 2010

L15 ANSWER 67 OF 101 MEDLINE ® on STN DUPLICATE 16
ACCESSION NUMBER: 2011153811 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21275327
TITLE: **Cabazitaxel**, a taxane for men with hormone-refractory metastatic prostate **cancer**.

AUTHOR: Wilkes Gail M
CORPORATE SOURCE: Boston Medical Center, Boston, Massachusetts, USA.
SOURCE: Oncology (Williston Park, N.Y.), **(2010 Oct)** Vol. 24, No. 10 Suppl, pp. 46-8.
Journal code: 8712059. ISSN: 0890-9091. L-ISSN: 0890-9091.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201102
ENTRY DATE: Entered STN: 1 Feb 2011
Last Updated on STN: 24 Feb 2011
Entered Medline: 23 Feb 2011
OS.CITING REF COUNT: 1 There are 1 MEDLINE records that cite this record

L15 ANSWER 68 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010646994 EMBASE
TITLE: Horizon scanning for novel therapeutics for the treatment of **prostate cancer**.
AUTHOR: Bianchini, D.; Zivi, A.; Sandhu, S.; de Bono, J.S., Dr. (correspondence)
CORPORATE SOURCE: The Royal Marsden Hospital, The Institute of Cancer Research, Sutton, United Kingdom. johann.de-bono@icr.ac.uk
AUTHOR: de Bono, J.S., Dr. (correspondence)
CORPORATE SOURCE: Centre for Cancer Therapeutics, Institute for Cancer Research, Royal Marsden Hospital, Downs Road, Sutton, Surrey SM2 5PT, United Kingdom. johann.de-bono@icr.ac.uk
SOURCE: Annals of Oncology, (October 2010) Vol. 21, No. SUPPL. 7, pp. vii43-vii55.
Refs: 152
ISSN: 0923-7534; E-ISSN: 1569-8041 CODEN: ANONE2
PUBLISHER: Oxford University Press, Great Clarendon Street, Oxford, OX2 6DP, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 8 Dec 2010
Last Updated on Embase: 8 Dec 2010

AB Treatment options for patients with advanced **prostate cancer** (PCa) remain limited. Improved understanding of the underlying molecular drivers of PCa pathogenesis, progression and resistance development has provided the fundamental basis for rational targeted drug design. Key findings in recent years include the identification of ETS gene rearrangements, the dissection of PCa molecular heterogeneity and the discovery that castration-resistant **prostate cancer** (CRPC) remains androgen driven despite the androgen-depleted milieu, thus making androgen receptor (AR) signaling a continued focus of molecularly targeted treatments. AR ligand-independent activation of tyrosine kinase prosurvival signaling cascades and angiogenesis have also been implicated in disease progression. A multitude of new molecularly targeted agents that abrogate AR signaling, inhibit the mitogenic and prosurvival signal transduction pathways, perturb the **tumor**-bone microenvironment, impair **tumor** vasculature, facilitate immune modulation and induce apoptosis are in clinical development and are highly likely to change the current treatment paradigm. It is clear that the success of these molecular

targeted therapies hinges in part on optimal patient selection based on the molecular disease profile and an improved understanding of the mechanistic basis of acquired resistance. This review outlines the current clinical development of molecular targeted treatments in CRPC, with particular emphasis on agents that are in the later stages of clinical development, and details the challenges and future direction of developing these **antitumor** agents.

L15 ANSWER 69 OF 101 EMBASE
ACCESSION NUMBER: 2010454193 EMBASE
TITLE: [FDA approval: New taxane in hormone-refractory **prostate carcinoma**].
FDA-zulassung: Neues taxan bei hormonrefraktarem prostatakarzinom.
AUTHOR: Kusnick, Carolina, Dr. (correspondence)
SOURCE: Deutsche Apotheker Zeitung, (5 Aug 2010) Vol. 150, No. 31, pp. 41-42.
ISSN: 0011-9857 CODEN: DAZEA2
PUBLISHER: Deutscher Apotheker Verlag, Birkenwaldstr.44,, Stuttgart, 70191, Germany.
COUNTRY: Germany
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
036 Health Policy, Economics and Management
037 Drug Literature Index
LANGUAGE: German
ENTRY DATE: Entered Embase: 7 Sep 2010
Last Updated on Embase: 7 Sep 2010

L15 ANSWER 70 OF 101 MEDLINE ® on STN DUPLICATE 17
ACCESSION NUMBER: 2011092742 MEDLINE
DOCUMENT NUMBER: PubMed ID: 21208852
TITLE: Improving outcomes with recent advances in chemotherapy for castrate-resistant prostate **cancer**.
AUTHOR: Sartor Oliver; Halstead Michael; Katz Leah
CORPORATE SOURCE: Tulane Medical School New Orleans, LA, USA. osartor@tulane.edu
SOURCE: Clinical genitourinary cancer, **(2010 Dec 1)** Vol. 8, No. 1, pp. 23-8.
Journal code: 101260955. E-ISSN: 1938-0682. L-ISSN: 1558-7673.
DIGITAL OBJECT IDENTIFIER: 10.3816/CGC.2010.n.004
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 201106
ENTRY DATE: Entered STN: 7 Jan 2011
Last Updated on STN: 16 Jun 2011
Entered Medline: 15 Jun 2011
OS.CITING REF COUNT: 2 There are 2 MEDLINE records that cite this record
AB The FDA's approval of docetaxel in 2004 created a clear mandate for clinical researchers to create new therapies effective for metastatic castrate-resistant prostate **cancer** (mCRPC) patients with progression post-docetaxel. In 2010, the first trial to prolong survival in this setting was announced using a **cabazitaxel**, a novel taxane. This

therapeutic agent was specifically designed to have activity in model systems resistant to conventional taxanes. After phase I testing, with observation of clinically significant activity in patients with advanced **cancers**, **cabazitaxel**/prednisone was tested in a large phase III trial enrolling patients with mCRPC who had disease progression despite prior docetaxel therapy. This phase III trial TROPIC (Treatment of Hormone-Refractory Metastatic Prostate **Cancer** Previously Treated With a Taxotere-Containing Regimen), using mitoxantrone/prednisone as a control arm, demonstrated a significant improvement in survival (the primary endpoint). Both subset analyses and secondary endpoints (response rates and time to progression) were supportive of the survival findings. This trial was pivotal for the FDA's approval of the new drug application for **cabazitaxel**. This review focuses on both the pre-clinical and clinical development of **cabazitaxel**.

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ACCESSION NUMBER: 2010277862 EMBASE
TITLE: **Cabazitaxel** prolongs survival in late metastatic CRPC: Commentary.
AUTHOR: Armstrong, Andrew J.
SOURCE: Oncology Report, (2010) No. MARCH-APRIL, pp. 22. ISSN: 1548-5323; E-ISSN: 1548-5323
PUBLISHER: Elsevier Oncology, 46 Green Street, 2nd Floor, Huntington, NY 11743, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered Embase: 14 Jun 2010
Last Updated on Embase: 14 Jun 2010

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ACCESSION NUMBER: 2010277863 EMBASE
TITLE: **Cabazitaxel** prolongs survival in late metastatic CRPC.
AUTHOR: MacNeil, Jane Salodof
SOURCE: Oncology Report, (2010) No. MARCH-APRIL, pp. 22. ISSN: 1548-5323; E-ISSN: 1548-5323
PUBLISHER: Elsevier Oncology, 46 Green Street, 2nd Floor, Huntington, NY 11743, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; (Short Survey)
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered Embase: 14 Jun 2010
Last Updated on Embase: 14 Jun 2010

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ACCESSION NUMBER: 2011592883 EMBASE
TITLE: Cytotoxic chemotherapy for castration resistant **prostate cancer**: 2010 and beyond.
AUTHOR: Seng, Sonia M.; Tsao, Che-Kai; Galsky, Matthew D.; Oh, William K. (correspondence)
CORPORATE SOURCE: Mount Sinai School of Medicine/Tisch Cancer Institute, New York, NY, United States. William.oh@mssm.edu

SOURCE: Drug Discovery Today: Therapeutic Strategies, (Summer 2010) Vol. 7, No. 1-2, pp. 17-22.
Refs: 60
E-ISSN: 1740-6773
PUBLISHER: Elsevier Ltd, Langford Lane, Kidlington, Oxford, OX5 1GB, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 006 Internal Medicine
016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
FILE SEGMENT:ClinicalTrials.gov
CLINICAL TRIAL NO.: NCT00110214; NCT00134056; NCT00519285; NCT00617669; NCT00744497; NCT00917748; NCT00988208; NCT01083615; NCT01188187
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 10 Nov 2011
Last Updated on Embase: 10 Nov 2011

AB **Prostate cancer** is the second most common **cancer** and the fifth most common cause of **cancer** deaths in men worldwide. Before docetaxel chemotherapy, no single agent demonstrated a survival advantage for men with metastatic castration resistant **prostate cancer** (mCRPC). However, 2010 has proven to be a landmark year for **prostate cancer** therapy, with the results of several phase III trials demonstrating improvements in survival with novel therapies. In this review, we examine promising front-line docetaxel combination therapies for chemotherapy naive patients and novel cytotoxic chemotherapies for patients with progressive disease. We will also highlight a clinical trial utilizing a genome based approach to evaluate the efficacy of a fourth generation platinum, satraplatin, in patients with sporadic BRCA deficient **prostate cancers**. © 2011 Elsevier Ltd. All rights reserved.

ACCESSION NUMBER: 2011019900 EMBASE
TITLE: Advanced **prostate cancer** 2010: What a year!.
AUTHOR: Sartor, Oliver (correspondence); Vogelzang, Nicholas
CORPORATE SOURCE: Tulane Cancer Center, New Orleans, LA, United States.
AUTHOR: Sartor, Oliver (correspondence); Vogelzang, Nicholas
CORPORATE SOURCE: Comprehensive Cancer Centers of Nevada, US Oncology Research, Las Vegas, NV, United States.
SOURCE: Clinical Genitourinary Cancer, (1 Dec 2010) Vol. 8, No. 1, pp. 8-9.
Refs: 4
ISSN: 1558-7673; E-ISSN: 1938-0682
PUBLISHER: Cancer Information Group, LP, 3500 Maple Avenue, Suite 750, Dallas, TX 75219, United States.
PUBLISHER IDENT.: G034T3035422253N
COUNTRY: United States
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 006 Internal Medicine
016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered Embase: 27 Jan 2011
Last Updated on Embase: 27 Jan 2011

reserved on STN
ACCESSION NUMBER: 2010527713 EMBASE
TITLE: **Cabazitaxel** gets the go-ahead for advanced **prostate cancer**.
AUTHOR: Ault, Alicia (correspondence)
SOURCE: Oncology Report, (July-August 2010) No. JULY-AUGUST, pp. 2.
ISSN: 1548-5323; E-ISSN: 1548-5323
PUBLISHER: Elsevier Oncology, 46 Green Street, 2nd Floor, Huntington, NY 11743, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 19 Oct 2010
Last Updated on Embase: 19 Oct 2010

L15 ANSWER 76 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010527714 EMBASE
TITLE: **Cabazitaxel** gets the go-ahead for advanced **prostate cancer**: Comment.
AUTHOR: Stadler, Walter M.
SOURCE: Oncology Report, (July-August 2010) No. JULY-AUGUST, pp. 2.
ISSN: 1548-5323; E-ISSN: 1548-5323
PUBLISHER: Elsevier Oncology, 46 Green Street, 2nd Floor, Huntington, NY 11743, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 19 Oct 2010
Last Updated on Embase: 19 Oct 2010

L15 ANSWER 77 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2011592880 EMBASE
TITLE: New frontiers in therapy for metastatic **prostate cancer**.
AUTHOR: Gulley, James L. (correspondence)
CORPORATE SOURCE: Laboratory of Tumor Immunology and Biology and Medical Oncology Branch, National Cancer Institute, Center for Cancer Research, 10 Center Dr. 13N208, MSC-1750, Bethesda, MD 20892, United States. gulleyj@mail.nih.gov
SOURCE: Drug Discovery Today: Therapeutic Strategies, (Summer 2010) Vol. 7, No. 1-2, pp. 1-3.
Refs: 21
E-ISSN: 1740-6773
PUBLISHER: Elsevier Ltd, Langford Lane, Kidlington, Oxford, OX5 1GB, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 016 Cancer
026 Immunology, Serology and Transplantation
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 10 Nov 2011
Last Updated on Embase: 10 Nov 2011

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ACCESSION NUMBER: 0050253336 EMBASE

TITLE: **Cabazitaxel** or mitoxantrone with prednisone in patients with metastatic castration-resistant **prostate cancer** (mCRPC) previously treated with docetaxel: Final results of a multinational phase III trial (TROPIC).

AUTHOR: De Bono, J.S. (correspondence); Oudard, S.; Ozguroglu, M.; Hansen, S.; Machiels, J.H.; Shen, L.; Matthews, P.; Sartor, A.O.

SOURCE: Journal of Clinical Oncology, (20 May 2010) Vol. 28, No. 15, Supp. SUPPL. 1. Abstract Number: 4508.
Meeting Info: 2010 Annual Meeting of the American Society of Clinical Oncology, ASCO. Chicago, IL, United States. 04 Jun 2010-08 Jun 2010
ISSN: 0732-183X

PUBLISHER: American Society of Clinical Oncology.

DOCUMENT TYPE: Journal; Conference; (Conference Abstract)

FILE SEGMENT: CONF

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered Embase: Sep 2012
Last Updated on Embase: Sep 2012

AB Background: The treatment of mCRPC following docetaxel (D) therapy failure due to progressive disease (PD) or toxicity is an unmet medical need. TROPIC evaluated the efficacy and safety of the novel taxane **cabazitaxel** (Cbz) in men with mCRPC previously treated with D. Methods: Men with mCRPC, ECOG PS 0-2, and adequate organ function who had prior hormone therapy, chemotherapy, and radiotherapy, but had PD during or after D (cumulative dose ≥225 mg/m2) were randomized to 10 mg/day of prednisone with either mitoxantrone 12 mg/m2 (MP) or Cbz 25 mg/m2 (CbzP), both administered 3-weekly. The primary endpoint was overall survival (OS). Secondary endpoints were progression- free survival (PFS- composite of **tumor**, PSA, or pain progression; or death); response; time to progression (TTP) for **tumor**, PSA, pain; and safety. The study had 90% power to detect a 25% lower hazard rate for death in the CbzP group after 511 events (2-sided α= 0.05). Results: From Jan 2007 to Oct 2008, 755 men (median age 68 yr; 84% white) were randomized. Patients' characteristics were well balanced. Median prior D dose was 576 mg/m2 for CbzP and 529 mg/m2 for MP. Median follow-up was 12.8 mos. Median number of cycles was 6 for CbzP and 4 for MP. In the primary ITT analysis, the CbzP group had a statistically significantly longer OS compared with MP ($p<0.0001$). PFS, response rates, and TTP (by RECIST and PSA) also statistically significantly favored CbzP. Subgroup analyses by risk factors and a multivariate analysis showed that OS outcomes were consistent and robust in favor of CbzP. Most frequent Gr 3/4 toxicity was neutropenia (81.7% CbzP; 58.0% MP); rates of febrile neutropenia were 7.5% and 1.3%, respectively. Conclusions: Men with mCRPC progressing after D benefit from CbzP treatment with longer OS, TTP by **tumor** assessments and PSA, and higher response rates. (Table presented).

L15 ANSWER 79 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2011079773 EMBASE

TITLE: Castration-refractory **prostate cancer**: New therapies, new questions.

AUTHOR: Appleman, Leonard J.

CORPORATE SOURCE: Division of Hematology-Oncology, Department of Medicine, University of Pittsburgh, Pittsburgh, PA, United States.

AUTHOR: Appleman, L. J., Dr. (correspondence)

CORPORATE SOURCE: Division of Hematology-Oncology, Department of Medicine, University of Pittsburgh, Pittsburgh, PA, United States.

SOURCE: Oncology, (December 2010) Vol. 24, No. 14.
Refs: 14
ISSN: 0890-9091; E-ISSN: 0890-9091 CODEN: OCLGE9
PUBLISHER: UBM Medica Healthcare Publications, PO Box 390427, Minneapolis, MN 55439, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 009 Surgery
016 Cancer
026 Immunology, Serology and Transplantation
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 23 Feb 2011
Last Updated on Embase: 28 Feb 2011

L15 ANSWER 80 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2011079774 EMBASE
TITLE: A renaissance in the medical treatment of advanced **prostate cancer.**
AUTHOR: Rove, Kyle O.
CORPORATE SOURCE: Division of Urology, University of Colorado Denver School of Medicine, Aurora, CO, United States.
AUTHOR: Flaig, Thomas W.
CORPORATE SOURCE: Division of Medical Oncology, University of Colorado Denver School of Medicine, Aurora, CO, United States.
AUTHOR: Rove, K. O., Dr. (correspondence)
CORPORATE SOURCE: Division of Urology, University of Colorado Denver School of Medicine, Aurora, CO, United States.
SOURCE: Oncology, (December 2010) Vol. 24, No. 14.
Refs: 45
ISSN: 0890-9091; E-ISSN: 0890-9091 CODEN: OCLGE9
PUBLISHER: UBM Medica Healthcare Publications, PO Box 390427, Minneapolis, MN 55439, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 23 Feb 2011
Last Updated on Embase: 28 Feb 2011
AB **Prostate cancer** will be diagnosed in one of six men during their lifetimes, and a small portion of these will progress after primary and salvage therapies. For many years, there were few treatment options for these patients after routine hormonal maneuvers, and standard of care since the early 2000s has consisted primarily of docetaxel, which improved survival over the previous first-line therapy mitoxantrone. In recent years, however, new therapies have begun to emerge to treat this devastating form of **prostate cancer.** This review examines the mechanisms behind these therapeutics and the key trials seeking to validate their clinical use.

L15 ANSWER 81 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2010014964 EMBASE
TITLE: Cytotoxic compounds in the treatment of castration-resistant **prostate cancer.**

AUTHOR: Lee, Patrick; Aragon-Ching, Jeanny B.
CORPORATE SOURCE: Division of Hematology and Oncology, George Washington University Medical Center, 2150 Pennsylvania Avenue, NW, Washington, DC 20037, United States. jaragonching@mfa.gwu.edu
AUTHOR: Aragon-Ching, J. B. (correspondence)
CORPORATE SOURCE: Division of Hematology and Oncology, George Washington University Medical Center, 2150 Pennsylvania Avenue, NW, Washington, DC 20037, United States. jaragonching@mfa.gwu.edu
SOURCE: Anti-Cancer Agents in Medicinal Chemistry, (2009) Vol. 9, No. 10, pp. 1040-1045.
Refs: 79
ISSN: 1871-5206
PUBLISHER: Bentham Science Publishers B.V., P.O. Box 294, Bussum, 1400 AG, Netherlands.
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
022 Human Genetics
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 1 Mar 2010
Last Updated on Embase: 1 Mar 2010

AB **Prostate cancer** is the most common non-cutaneous **cancer** among men in the United States. Most will be diagnosed at an early stage, but a significant number will still develop metastatic castration resistant disease. Docetaxel has demonstrated improved quality of life and overall survival in metastatic castration-resistant **prostate cancer** but virtually all patients will ultimately become refractory to taxane therapy. Second-line options are limited and new effective chemotherapeutic agents or combinations are needed in this setting. This review will focus on cytotoxic compounds in clinical investigation either in combination with taxanes in the first or second-line setting and other novel compounds, such as platinums and microtubule-targeting agents that are in active clinical investigation. © 2009 Bentham Science Publishers Ltd.

L15 ANSWER 82 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 18
ACCESSION NUMBER: 2009:59403 HCAPLUS
DOCUMENT NUMBER: 151:115508
TITLE: Phase I and Pharmacokinetic Study of XRP6258 (RPR 116258A), a Novel Taxane, Administered as a 1-Hour Infusion Every 3 Weeks in Patients with Advanced Solid **Tumors**
AUTHOR(S): Mita, Alain C.; Denis, Louis J.; Rowinsky, Eric K.; DeBono, Johann S.; Goetz, Andrew D.; Ochoa, Leonel; Forouzesh, Bahram; Beeram, Muralidhar; Patnaik, Amita; Molpus, Kathleen; Semiond, Dorothee; Besenval, Michele; Tolcher, Anthony W.
CORPORATE SOURCE: Institute for Drug Development, Cancer Therapy and Research Center and University of Texas Health Science Center at San Antonio, San Antonio, TX, USA
SOURCE: Clinical Cancer Research (**2009**), 15(2), 723-730
CODEN: CCREF4; ISSN: 1078-0432
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English

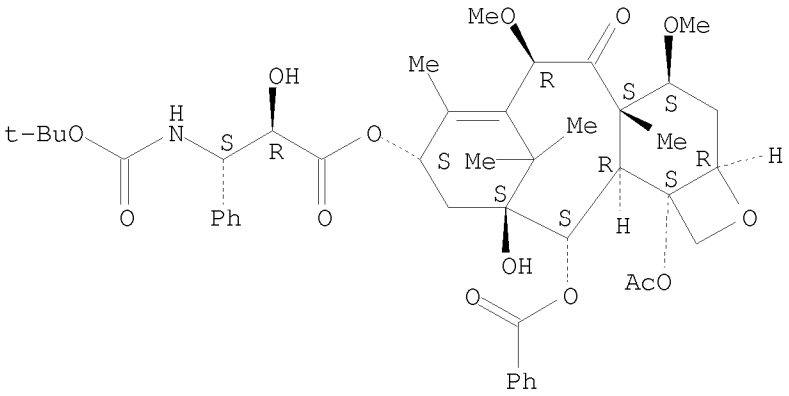
AB Purpose: To assess the feasibility of administering XRP6258, a new taxane with a low affinity for the multidrug resistance 1 protein, as a 1-h i.v. infusion every 3 wk. The study also sought to determine the maximum tolerated dose and the recommended dose, to describe the pharmacokinetic (PK) behavior of the compound, and to seek preliminary evidence of **anticancer** activity. Exptl. Design: Twenty-five patients with advanced solid malignancies were treated with 102 courses of XRP6258 at four dose levels ranging from 10 to 25 mg/m2. Dose escalation was based on the occurrence of dose-limiting toxicity (DLT) at each dose level, provided that PK variables were favorable. The maximum tolerated dose was defined as the dose at which at least two patients developed a DLT at the first course. Results: Neutropenia was the principal DLT, with one patient experiencing febrile neutropenia and two others showing prolonged grade 4 neutropenia at the 25 mg/m2 dose level. Nonhematol. toxicities, including nausea, vomiting, diarrhea, neurotoxicity, and fatigue, were generally mild to moderate in severity. XRP6258 exhibited dose-proportional PK, a triphasic elimination profile, a long terminal half-life (77.3 h), a high clearance (mean CL, 53.5 L/h), and a large volume of distribution (mean Vss, 2,034 L/m2). Objective **antitumor** activity included partial responses in two patients with metastatic prostate **carcinoma**, one unconfirmed partial response, and two minor responses. Conclusion: The recommended phase II dose of XRP6258 on this schedule is 20 mg/m2. The general tolerability and encouraging **antitumor** activity in taxane-refractory patients warrant further evaluations of XRP6258.

IT **183133-96-2, XRP 6258**
RL: ADV (Adverse effect, including toxicity); **PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**pharmacokinetics** of XRP6258 in patients with advanced solid **tumors**)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 77 THERE ARE 77 CAPLUS RECORDS THAT CITE THIS RECORD (79 CITINGS)

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 83 OF 101 EMBASE

reserved on STN
ACCESSION NUMBER: 2009457731 EMBASE
TITLE: Recent Progress and Pitfalls in Testing Novel Agents in Castration-Resistant **Prostate Cancer**.
AUTHOR: Bellmunt, Joaquim (correspondence)
CORPORATE SOURCE: University Hospital del Mar, IMIM, U. Pompeu Fabra, Barcelona, Spain. jbellmunt@imas.imim.es
AUTHOR: Bellmunt, Joaquim (correspondence); Rosenberg, Jonathan E.; Choueiri, Toni K.
CORPORATE SOURCE: Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA, United States. jbellmunt@imas.imim.es
SOURCE: European Urology, (October 2009) Vol. 56, No. 4, pp. 606-608.
Refs: 15
ISSN: 0302-2838 CODEN: EUURAV
PUBLISHER: Elsevier, P.O. Box 211, Amsterdam, 1000 AE, Netherlands.
PUBLISHER IDENT.: S 0302-2838(09)00728-3
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 21 Sep 2009
Last Updated on Embase: 21 Sep 2009

L15 ANSWER 84 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2009203301 EMBASE
TITLE: Tubulin-interactive natural products as **anticancer** agents.
AUTHOR: Kingston, David G. I.
CORPORATE SOURCE: Department of Chemistry, Virginia Polytechnic Institute, State University, Blacksburg, VA 24061-0212. dkingston@vt.edu
AUTHOR: Kingston, D. G. I. (correspondence)
CORPORATE SOURCE: Department of Chemistry, Virginia Polytechnic Institute, State University, Blacksburg, VA 24061-0212. dkingston@vt.edu
SOURCE: Journal of Natural Products, (27 Mar 2009) Vol. 72, No. 3, pp. 507-515.
Refs: 163
ISSN: 0163-3864 CODEN: JNPRDF
PUBLISHER: American Chemical Society, 2540 Olentangy River Road, P.O. Box 3337, Columbus, OH 43210-3337, United States.
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 006 Internal Medicine
016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 8 May 2009
Last Updated on Embase: 8 May 2009
AB This review provides an overview of the discovery, structures, and biological activities of **anticancer** natural products that act by inhibiting or promoting the assembly of tubulin to microtubules. The emphasis is on providing recent information on those compounds in clinical use or in advanced clinical trials. The vinca alkaloids, the combretastatins, NPI-2358, the halichondrin B analogue eribulin,

dolastatin 10, noscapine, hemiasterlin, and rhizoxin are discussed as tubulin polymerization inhibitors, while the taxanes and the epothilones are the major classes of tubulin polymerization promoters presented, with brief treatments of discodermolide, eleutherobin, and laulimalide. The challenges and future directions of tubulin-interactive natural products-based drug discovery programs are also discussed briefly.

L15 ANSWER 85 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2009493213 EMBASE
TITLE: New agents in metastatic **prostate cancer**.
AUTHOR: Fizazi, Karim (correspondence); Massard, Christophe
CORPORATE SOURCE: Department of Medicine, University of Paris XI, Villejuif, France.
SOURCE: European Journal of Cancer, (September 2009) Vol. 45, No. SUPPL. 1, pp. 379-380.
Refs: 15
ISSN: 0959-8049 CODEN: EJCAEL
PUBLISHER: Elsevier Ltd, Langford Lane, Kidlington, Oxford, OX5 1GB, United Kingdom.
PUBLISHER IDENT.: S 0959-8049(09)70056-5
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 006 Internal Medicine
016 Cancer
028 Urology and Nephrology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 5 Nov 2009
Last Updated on Embase: 5 Nov 2009

L15 ANSWER 86 OF 101 BIOSIS COPYRIGHT (c) 2013 The Thomson Corporation on STN

ACCESSION NUMBER: 2010:357817 BIOSIS
DOCUMENT NUMBER: PREV201000357817
TITLE: A dose escalating study of **cabazitaxel** (XRP6258) in combination with capecitabine, in patients (pts) with metastatic breast **cancer** (MBC) progressing after anthracycline and taxane therapy.
AUTHOR(S): Villanueva, C. [Reprint Author]; Awada, A.; Campone, M.; Machiels, J. P.; Besse, T.; Magherini, E.; Dubin, F.; Semiond, D.; Pivot, X.
CORPORATE SOURCE: Hop Jean Minjoz, F-25030 Besancon, France
SOURCE: EJC Supplements, (**SEP 2009**) Vol. 7, No. 2, pp. 268.
Meeting Info.: 15th Congress of the European-Cancer-Organization/34th Multidisciplinary Congress of the European-Society-for-Medical-Oncology. Berlin, GERMANY. September 20 -24, 2009. European Canc Org; European Soc Med Oncol.
ISSN: 1359-6349.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jun 2010
Last Updated on STN: 23 Jun 2010

L15 ANSWER 87 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2009585532 EMBASE

TITLE: Editorial: New therapeutic agents for castration-refractory **prostate cancer.**
AUTHOR: Patten, David (correspondence); Sartor, Oliver
CORPORATE SOURCE: Department of Medicine, Tulane Medical School, New Orleans, LA, United States.
AUTHOR: Sartor, Oliver
CORPORATE SOURCE: Division of Hematology/Oncology, Tulane Medical School, New Orleans, LA, United States.
AUTHOR: Sartor, Oliver
CORPORATE SOURCE: Department of Urology, Tulane Medical School, New Orleans, LA, United States.
SOURCE: Clinical Genitourinary Cancer, (2009) Vol. 7, No. 2, pp. E4-E6.
Refs: 15
ISSN: 1558-7673
PUBLISHER: Cancer Information Group, LP, 3500 Maple Avenue, Suite 750, Dallas, TX 75219, United States.
PUBLISHER IDENT.: T6323758T2710605
COUNTRY: United States
DOCUMENT TYPE: Journal; Editorial
FILE SEGMENT: 006 Internal Medicine
016 Cancer
028 Urology and Nephrology
037 Drug Literature Index
LANGUAGE: English
ENTRY DATE: Entered Embase: 14 Dec 2009
Last Updated on Embase: 14 Dec 2009

L15 ANSWER 88 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2008294234 EMBASE
TITLE: New tubulin targeting agents currently in clinical development.
AUTHOR: Carlson, Robert O., Dr. (correspondence)
CORPORATE SOURCE: Discovery Biology, Myriad Pharmaceuticals Inc., 320 Wakara Way, Salt Lake City, UT 84103, United States. rcarlson@myriad.com
SOURCE: Expert Opinion on Investigational Drugs, (May 2008) Vol. 17, No. 5, pp. 707-722.
Refs: 117
ISSN: 1354-3784 CODEN: EOIDER
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 27 Jun 2008
Last Updated on Embase: 27 Jun 2008
AB Background: The first tubulin targeting agents were approved as **cancer** chemotherapeutics over 40 years ago and tubulin as an **antitumor** target continues to attract significant drug discovery and development attention. Mechanism of action as defined by tubulin binding sites and effect on microtubules distinguishes these agents, but the end result is equivalent in that microtubule disruption leads to cell cycle arrest at G2/M phase of the cell cycle and subsequent apoptotic cell death. Objectives: The goal of this review is to describe the state of clinical development of tubulin targeting agents as of early 2008, with descriptions of clinical experience slanted toward the most advanced trials for each agent. Method: Objective information in this review was obtained exclusively from

public sources that included journals, scientific meeting abstracts, posters and oral presentations, websites and public presentations from companies. Opinions expressed in this review are exclusively from the author. Results/conclusions: A large number of tubulin targeting agents are currently in clinical development, including microtubule stabilizing and destabilizing compounds acting through all three of the characterized tubulin binding sites. With the approval of ixabepilone for refractory breast **cancer**, the epothilones appear best positioned to make an impact among the new microtubule stabilizing compounds. There are 17 microtubule destabilizing agents under clinical assessment, with many only in Phase I and results to date include at best modest efficacy signals with no obvious indication trend. © 2008 Informa UK Ltd.

L15 ANSWER 89 OF 101 MEDLINE ® on STN DUPLICATE 19
ACCESSION NUMBER: 2008503223 MEDLINE
DOCUMENT NUMBER: PubMed ID: 18685415
TITLE: Systemic therapy after first-line docetaxel in metastatic castration-resistant prostate **cancer**.
AUTHOR: Beardsley Emma K; Chi Kim N
CORPORATE SOURCE: BC Cancer Agency, Vancouver, Canada.
SOURCE: Current opinion in supportive and palliative care, **(2008 Sep)** Vol. 2, No. 3, pp. 161-6. Ref: 31
Journal code: 101297402. E-ISSN: 1751-4266. L-ISSN: 1751-4258.
DIGITAL OBJECT IDENTIFIER: 10.1097/SPC.0b013e32830c48a3
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: MEDLINE; Priority Journals
ENTRY MONTH: 200810
ENTRY DATE: Entered STN: 8 Aug 2008
Last Updated on STN: 21 Oct 2008
Entered Medline: 20 Oct 2008
OS.CITING REF COUNT: 3 There are 3 MEDLINE records that cite this record
REFERENCE COUNT: 31 There are 31 cited references for this document.
AB PURPOSE OF REVIEW: There is an urgent need for systemic treatment options for patients with castration-resistant prostate **cancer** who have progressed after receiving first-line docetaxel chemotherapy. The purpose of this article is to review recent developments in this area.

RECENT FINDINGS: Retreatment with docetaxel has been employed with evidence of activity in selected populations. Mitoxantrone, the previous first-line standard based on its palliative effect, has also been used with clinical responses observed; however, the symptom benefit in this setting has not been established. Several classes of cytotoxic agents have been tested including platinum agents (satraplatin), epothilones (ixabepilone and patupilone) and taxanes (**XRP-6258**). A number of targeted therapies have also been clinically evaluated including inhibitors of cytoprotective chaperones (OGX-011) and the vascular endothelial growth factor receptor (sorafenib, sunitinib, and cediranib). An area generating great interest has been the development of agents that target the androgen receptor axis more effectively (MDV3100 and abiraterone) with encouraging early phase trial results.

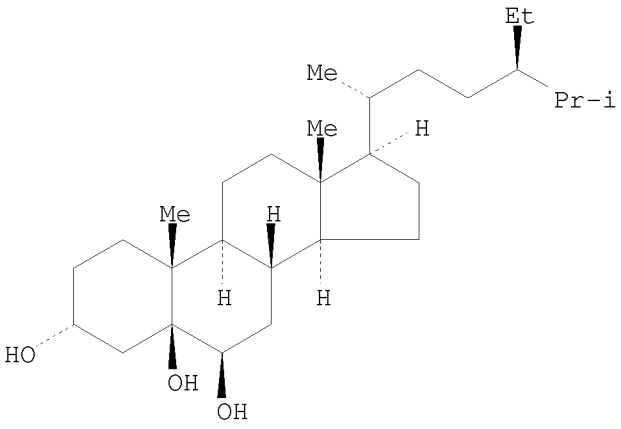
SUMMARY: There is no accepted standard systemic treatment for patients with castration resistant prostate **cancer** and progressive disease after docetaxel. Novel agents are in phase II and III clinical testing in this setting.

L15 ANSWER 90 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2007:619578 HCAPLUS

DOCUMENT NUMBER: 147:46112
TITLE: Treatment of **cancer** and other diseases
INVENTOR(S): Habib, Nabil
PATENT ASSIGNEE(S): Nabil Habib Lab, Lebanon; Vianova Labs, Inc.
SOURCE: PCT Int. Appl., 86pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007064691	A1	20070607	WO 2006-US45665	20061130 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW				
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
CA 2632903	A1	20070607	CA 2006-2632903	20061130 <--
EP 1968607	A1	20080917	EP 2006-844623	20061130 <--
R: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LI, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR				
US 20090226431	A1	20090910	US 2009-85892	20090306 <--
US 8293726	B2	20121023		
PRIORITY APPLN. INFO.:			US 2005-60741725 P	20051202 <--
			WO 2006-US45665 W	20061130 <--

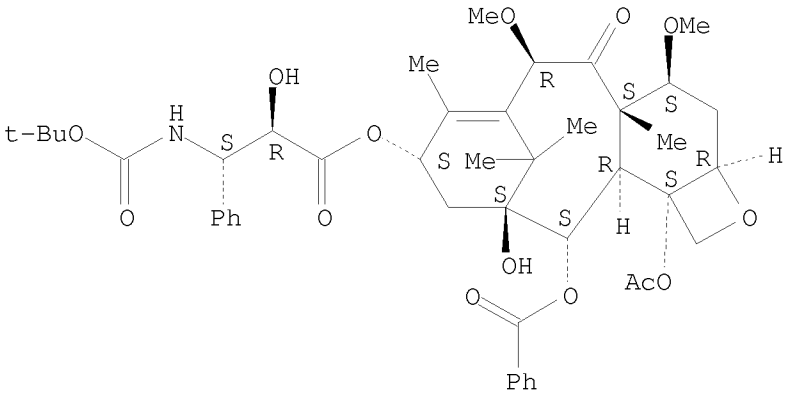
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 147:46112
GI



AB The present invention relates to a novel compound (e.g., 24-ethyl-cholestane-3β,5α,6α-triol), its production, its use, and to methods of treating **neoplasms** and other **tumors** as well as other diseases including hypercholesterolemia, autoimmune diseases, viral diseases (e.g., hepatitis B, hepatitis C, or HIV), and diabetes.

IT **183133-96-2, TXD 258**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(**treatment** of **cancer** and other diseases using ethylcholestane triol and combination with other agents)
RN 183133-96-2 HCAPLUS
CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 91 OF 101 EMBASE
ACCESSION NUMBER: 2007375639 EMBASE
TITLE: Medical management of advanced **prostate cancer**: A multidisciplinary team approach.
AUTHOR: Molins, Joaquim Bellmunt, Dr. (correspondence)
CORPORATE SOURCE: Hospital Del Mar, Solid Tumor Oncology (GU and GI) Medical Oncology Service, Paseo Maritimo 25-29, Barcelona 08003, Spain. jbellmunt@imas.imim.es
AUTHOR: Gelaberti i Mas, Antoni
CORPORATE SOURCE: Hospital Del Mar, Urology Department, Paseo Maritimo 25-29, Barcelona 08003, Spain. agelabert@imas.imim.es
SOURCE: Expert Review of Anticancer Therapy, (Jul 2007) Vol. 7, No. 7, pp. 977-979.
ISSN: 1473-7140; E-ISSN: 1744-8328 CODEN: ERATBJ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 016 Cancer
017 Public Health, Social Medicine and Epidemiology
028 Urology and Nephrology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered Embase: 1 Oct 2007
Last Updated on Embase: 1 Oct 2007

L15 ANSWER 92 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2008001236 EMBASE
TITLE: How do microtubule-targeted drugs work? An overview.
AUTHOR: Jordan, Mary Ann (correspondence); Kamath, Kathy
CORPORATE SOURCE: Department of Molecular, Cellular, and Developmental Biology, Neuroscience Research Institute, University of California Santa Barbara, Santa Barbara, CA 93106, United States. jordan@lifesci.ucsb.edu
SOURCE: Current Cancer Drug Targets, (Dec 2007) Vol. 7, No. 8, pp. 730-742.
Refs: 168
ISSN: 1568-0096 CODEN: CCDTB9
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 015 Chest Diseases, Thoracic Surgery and Tuberculosis
016 Cancer
025 Hematology
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 4 Feb 2008
Last Updated on Embase: 4 Feb 2008

AB The importance of microtubules in mitosis makes them a superb target for a group of highly successful, chemically diverse **anticancer** drugs. Knowledge of the mechanistic differences among the many drugs of this class is vital to understanding their tissue and cell specificity, the development of resistance, the design of novel improved drugs, optimal scheduling of treatment, and potential synergistic combinations. This overview covers microtubule assembly dynamics, the exquisite regulation of microtubule dynamics in cells by endogenous regulators, the important role of microtubule dynamics in mitosis, the diversity and number of microtubule-targeted drugs undergoing clinical development, the antimitotic mechanisms of microtubule-targeted drugs with emphasis on suppression of microtubule dynamics by vinblastine and taxol, the role of drug uptake and retention in the efficacy of microtubule-targeted drugs, and the anti-angiogenic and vascular-disrupting mechanisms of microtubule targeted drugs. In view of the success of this class of drugs, it has been argued that microtubules represent the single best **cancer** target identified to date, and it seems likely that drugs in this class will continue to remain an important chemotherapeutic class of drugs even as more selective chemotherapeutic approaches are developed. © 2007 Bentham Science Publishers Ltd.

L15 ANSWER 93 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN

ACCESSION NUMBER: 2006:578383 HCAPLUS
DOCUMENT NUMBER: 145:60921
TITLE: Genetic markers, methods, and kits for predicting/monitoring **cancer** patients' response to taxoids
INVENTOR(S): Grueneberg, Dorre; Huang, Xi; Natesan, Sridaran; August, Paul
PATENT ASSIGNEE(S): Aventis Pharmaceuticals Inc., USA
SOURCE: PCT Int. Appl., 77 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 11
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

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CN 2005-80041847	A3	20051201 <--
EP 2005-852717	A3	20051201 <--
IL 2005-183718	A3	20051201 <--
JP 2007-545523	A3	20051201 <--
KR 2007-7013060	A3	20051201 <--
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WO 2005-US43578	W	20051201 <--
IN 2007-CN2444	A3	20070607 <--
AU 2008-323673	A3	20081110 <--
IN 2011-CN2444	A3	20110412

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

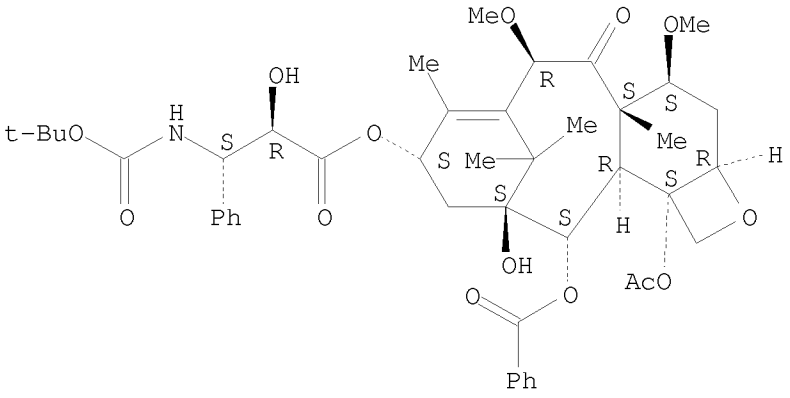
AB The present invention relates to novel and useful methods that predict or monitor a patient's response to a mol. of the taxoid family by measuring the increase or decrease of specific genetic markers as compared to controls. The present invention also provides kits that predict or monitor a patient's response to a mol. of the taxoid family by measuring nucleic acid or protein levels of particular genetic markers and comparing their levels to controls or reference markers. Thus, siRNA's were used to identify genes in human colon **cancer** cell line HCT116 which rendered these cells more resistant or more sensitive to docetaxel treatment. Twenty-four genetic markers were identified in this way.

IT **183133-96-2, XRP 6258**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(genetic markers, methods, and kits for predicting/monitoring **cancer** patients' response to taxoids)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 94 OF 101 EMBASE COPYRIGHT (c) 2013 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2006519050 EMBASE

TITLE: Development of new **cancer** therapeutic agents targeting mitosis.

AUTHOR: Miglarese, Mark R., Dr. (correspondence)
CORPORATE SOURCE: Array BioPharma, Inc., 3200 Walnut Street, Boulder, CO 80301, United States. mark.miglarese@arraybiopharma.com
AUTHOR: Carlson, Robert O.
CORPORATE SOURCE: Myriad Pharmaceuticals, 320 Wakara Way, Salt Lake City, UT 84103, United States. rcarlson@myriad.com
SOURCE: Expert Opinion on Investigational Drugs, (Nov 2006) Vol. 15, No. 11, pp. 1411-1425.
Refs: 125
ISSN: 1354-3784 CODEN: EOIDER
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
039 Pharmacy
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered Embase: 16 Nov 2006
Last Updated on Embase: 16 Nov 2006

AB Targeting cellular proliferation persists as a mainstay of **cancer** therapeutic strategy. Although microtubule-targeting drugs (such as taxanes and vinca alkaloids) have been used successfully in the clinic to treat a variety of **cancers**, they carry substantial liabilities that have spurred drug companies to aggressively pursue new tubulin-targeting drug candidates with improved efficacy and toxicity profiles. The recent discoveries of new mitotic targets for **cancer** therapy (such as kinesin spindle protein, Aurora kinases and Polo-like kinase-1) have also stimulated intense work focused on identifying novel antimitotic drugs directed at these new targets. A number of novel antimitotic drugs have demonstrated encouraging activity in preclinical models and have progressed into clinical development. This review focuses on selected new antimitotic drugs under evaluation in clinical trials. © 2006 Informa UK Ltd.

L15 ANSWER 95 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2005:1290072 HCAPLUS
DOCUMENT NUMBER: 144:46998
TITLE: The x-ray crystal structure of BRCA1 tandem BRCT repeat and BACH1 phosphopeptide complex and methods and compositions for **antitumor** drug design
INVENTOR(S): Yaffe, Michael B.; Clapperton, Julie A.; Manke, Isaac A.; Lowery, Drew M.; Ho, Timmy; Haire, Lesley F.; Smerdon, Stephen J.
PATENT ASSIGNEE(S): Massachusetts Institute of Technology, USA
SOURCE: PCT Int. Appl., 360 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005115454	A2	20051208	WO 2005-US15981	20050509 <--
WO 2005115454	A3	20071115		

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NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
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			US 2005-126022	A3	20050509 <--
			WO 2005-US15981	W	20050509 <--
			US 2008-229740	A1	20080826 <--

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

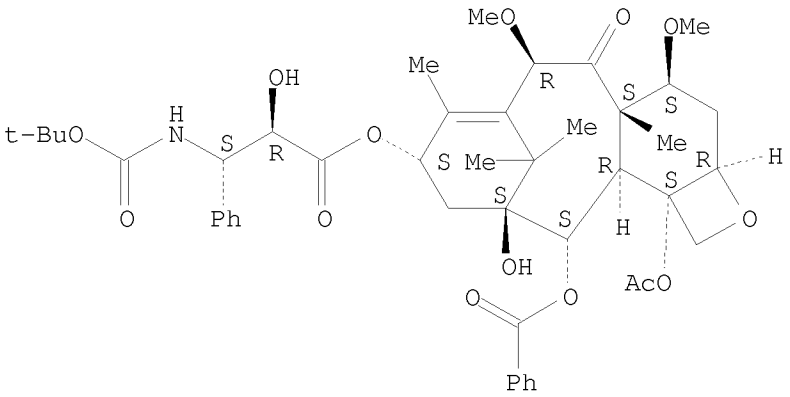
AB The present invention relates to compds. (e.g., peptidomimetics and non-peptides) that treat, prevent or stabilize cellular proliferative disorders and methods of treating, preventing, or stabilizing such disorders. The invention also provides three-dimensional structures of a BRCT domain-BACH1 phosphopeptide complex.

IT **183133-96-2, TXD 258**
RL: BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(x-ray crystal structure of BRCA1 tandem BRCT repeat and BACH1 phosphopeptide complex and methods and compns. for **antitumor drug** design)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



L15 ANSWER 96 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2005:409543 HCAPLUS

DOCUMENT NUMBER: 142:457053
TITLE: Human protein IAP (inhibitor of apoptosis protein) nucleobase oligomers, including dsRNA, shRNA, and siRNA, and their use for enhancing apoptosis in **cancer** therapy
INVENTOR(S): Lacasse, Eric; McManus, Daniel
PATENT ASSIGNEE(S): Aegera Therapeutics, Inc., Can.
SOURCE: PCT Int. Appl., 112 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005042558	A1	20050512	WO 2004-CA1902	20041029 <--
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RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
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EP 1682565	A1	20060726	EP 2004-789809	20041029 <--
R: DE, FR, GB				
JP 2007510408	T	20070426	JP 2006-537024	20041029 <--

PRIORITY APPLN. INFO.: US 2003-60516192 P 20031030 <--
WO 2004-CA1902 W 20041029 <--

AB The invention provides nucleobase oligomers and oligonucleotide duplexes that inhibit expression of an IAP (inhibitor of apoptosis protein), and methods for using them to induce apoptosis in a cell. Specifically, the invention provides nucleic acid sequences for siRNAs and shRNAs that target human XIAP, HIAP-1 or HIAP-2 genes. The nucleobase oligomers and oligomer complexes of the present invention may also be used to form pharmaceutical compns. The invention also features methods for enhancing apoptosis in a cell by administering a nucleobase oligomer or oligomer complex of the invention in combination with a chemotherapeutic or chemosensitizing agent. RNAi sequences and vectors producing shRNA (short hairpin RNA) were transfected into HeLa cells and evaluated for their effect on XIAP, cIAP-1, or cIAP-2 protein levels. XIAP protein could also be reduced by RNAi clones in transfected breast **cancer** cell line MDA-MB-231. In addition, cell survival was reduced in XIAP RNAi transfected breast **cancer** cell line after the transfected cells were treated with TRAIL (**tumor** necrosis factor-related apoptosis inducing ligand).

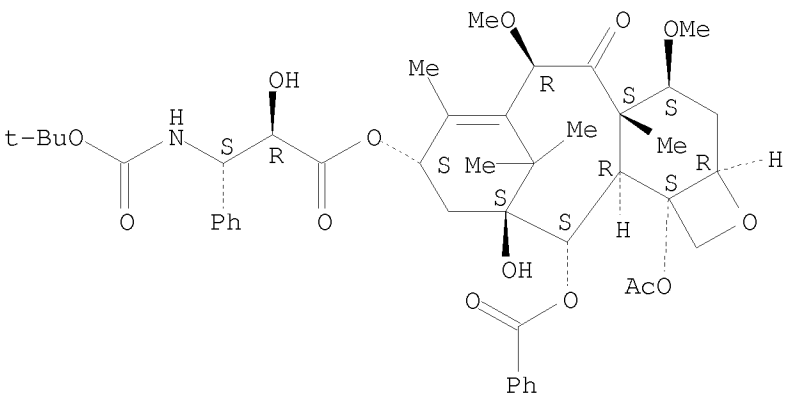
IT **183133-96-2, TXD 258**
RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(human protein IAP (inhibitor of apoptosis protein) nucleobase oligomers, including dsRNA, shRNA, and siRNA, and their use for enhancing apoptosis in **cancer therapy**)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX

NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 1 THERE ARE 1 CAPLUS RECORDS THAT CITE THIS RECORD (1 CITINGS)
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 97 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2005:409357 HCAPLUS
DOCUMENT NUMBER: 142:457052
TITLE: Sequences of antisense IAP (inhibitor of apoptosis protein) oligomers and their use for treatment of proliferative diseases with a chemotherapeutic agent
INVENTOR(S): Lacasse, Eric; McManus, Daniel; Durkin, Jon P.
PATENT ASSIGNEE(S): Aegera Therapeutics, Inc., Can.
SOURCE: PCT Int. Appl., 285 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005042030	A1	20050512	WO 2004-CA1900	20041029 <--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 20050119217	A1	20050602	US 2004-975790	20041028 <--
US 8012944	B2	20110906		
AU 2004284855	A1	20050512	AU 2004-284855	20041029 <--
CA 2542884	A1	20050512	CA 2004-2542884	20041029 <--
EP 1691842	A1	20060823	EP 2004-789807	20041029 <--
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BR 2004015779	A	20061226	BR 2004-15779	20041029 <--

CN 1901939	A	20070124	CN 2004-80039601		20041029 <--
JP 2007509861	T	20070419	JP 2006-537023		20041029 <--
ZA 2006003399	A	20070926	ZA 2006-3399		20041029 <--
NZ 547191	A	20090828	NZ 2004-547191		20041029 <--
RU 2376018	C2	20091220	RU 2006-117024		20041029 <--
SG 157422	A1	20091229	SG 2009-7918		20041029 <--
CN 101862459	A	20101020	CN 2010-10218334		20041029 <--
TW 332841	B	20101111	TW 2004-132984		20041029 <--
IL 175242	A	20130530	IL 2004-175242		20041029 <--
MX 2006004920	A	20070216	MX 2006-4920		20060502 <--
IN 2006MN00614	A	20070420	IN 2006-MN614		20060526 <--
NO 2006002420	A	20060731	NO 2006-2420		20060529 <--
KR 2006127393	A	20061212	KR 2006-7010619		20060530 <--
PRIORITY APPLN. INFO.:			US 2003-60516263	P	20031030 <--
			CN 2004-80039601	A3	20041029 <--
			WO 2004-CA1900	W	20041029 <--

ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT

AB The invention claims the use of an antisense oligomer to human XIAP, IAP-1 or IAP-2 genes and a chemotherapeutic agent, and compns. and kits thereof, for the treatment of proliferative diseases. The invention further claims sequences for nucleobase oligomers that are antisense IAP (inhibitor of apoptosis protein) oligomers. The antisense IAP nucleobase oligomers specifically hybridize with polynucleotides encoding an IAP and reduce the amount of an IAP protein produced in a cell. Thus by reducing the IAP protein, the invention provides methods for inducing **cancer** cells to undergo apoptosis and for overriding anti-apoptotic signals in **cancer** cells. As an example of the invention, mice with s.c. H460 human lung **carcinoma** xenografts were injected **intratumorally** with XIAP antisense mixed-base 2'-O-Me RNA oligonucleotides (C5 and/or G4) and the drug vinorelbine. At the end of the 24 d treatment period, the mean relative **tumor** growth was reduced .apprx.70% in treated mice. The inhibition of **tumor** growth was correlated with down-regulation of human XIAP protein expression and an increased number of dead cells. The mice did not show any signs of cytotoxicity such as body weight loss.

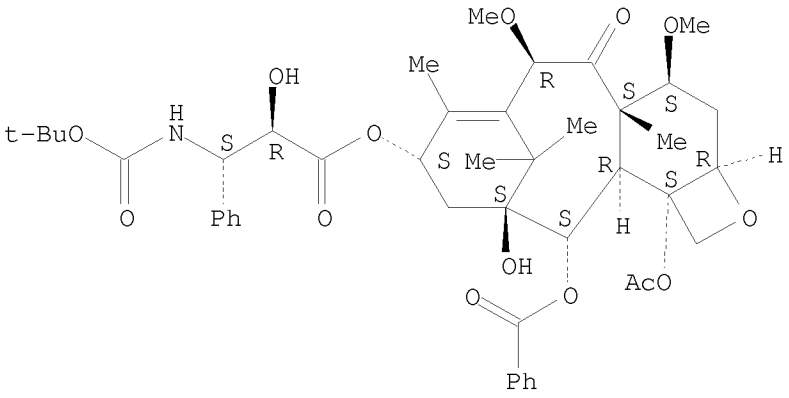
IT **183133-96-2, TXD 258**

RL: **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses) (sequences of antisense IAP (inhibitor of apoptosis protein) oligomers and their use for **treatment** of proliferative diseases with **chemotherapeutic** agent)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (7 CITINGS)
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 98 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2005:283298 HCAPLUS
DOCUMENT NUMBER: 142:349042
TITLE: Combinations of chlorpromazine compounds and antiproliferative drugs for the treatment of **neoplasms**
INVENTOR(S): Lee, Margaret S.; Nichols, James M.; Zhang, Yanzhen; Keith, Curtis
PATENT ASSIGNEE(S): Combinatorx, Incorporated, USA
SOURCE: PCT Int. Appl., 65 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 7
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005027842	A2	20050331	WO 2004-US30368	20040916 <--
WO 2005027842	A3	20051222		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2004273910	A1	20050331	AU 2004-273910	20040916 <--
CA 2538570	A1	20050331	CA 2004-2538570	20040916 <--
EP 1670477	A2	20060621	EP 2004-788798	20040916 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, PL, SK, HR				
BR 2004014568	A	20061107	BR 2004-14568	20040916 <--
CN 1878556	A	20061213	CN 2004-80033294	20040916 <--
JP 2007505914	T	20070315	JP 2006-527024	20040916 <--
MX 2006003066	A	20060620	MX 2006-3066	20060317 <--
NO 2006001325	A	20060606	NO 2006-1325	20060323 <--

KR 2007012618 A 20070126 KR 2006-7007244 20060414 <--
PRIORITY APPLN. INFO.: US 2003-60504310 P 20030918 <--
WO 2004-US30368 W 20040916 <--

OTHER SOURCE(S): MARPAT 142:349042

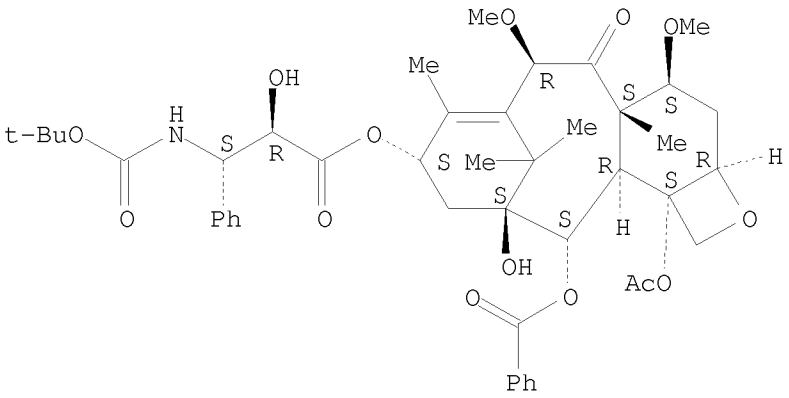
AB The invention discloses a method for treating a patient having a **cancer** or other **neoplasm** by administering chlorpromazine or a chlorpromazine analog and an antiproliferative agent simultaneously or within 14 days of each other in amts. sufficient to treat the patient.

IT **183133-96-2, TXD 258**
RL: **PAC (Pharmacological activity); THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(chlorpromazine compound-antiproliferative **drug antitumor** combination)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

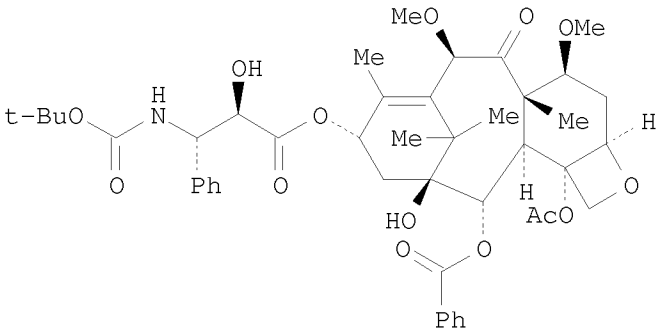
Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 4 THERE ARE 4 CAPLUS RECORDS THAT CITE THIS RECORD (6 CITINGS)

L15 ANSWER 99 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN DUPLICATE 20

ACCESSION NUMBER: 2003:355197 HCAPLUS
DOCUMENT NUMBER: 139:190599
TITLE: Nonlinear accumulation in the brain of the new taxoid TXD258 following saturation of P-glycoprotein at the blood-brain barrier in mice and rats
AUTHOR(S): Cisternino, Salvatore; Bourasset, Fanchon; Archimbaud, Yves; Semiond, Dorothee; Sanderink, Gerard; Scherrmann, Jean-Michel
CORPORATE SOURCE: INSERM , Hopital Fernand Widal, Paris, 75475/10, Fr.
SOURCE: British Journal of Pharmacology (**2003**), 138(7), 1367-1375
CODEN: BJPCBM; ISSN: 0007-1188
PUBLISHER: Nature Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

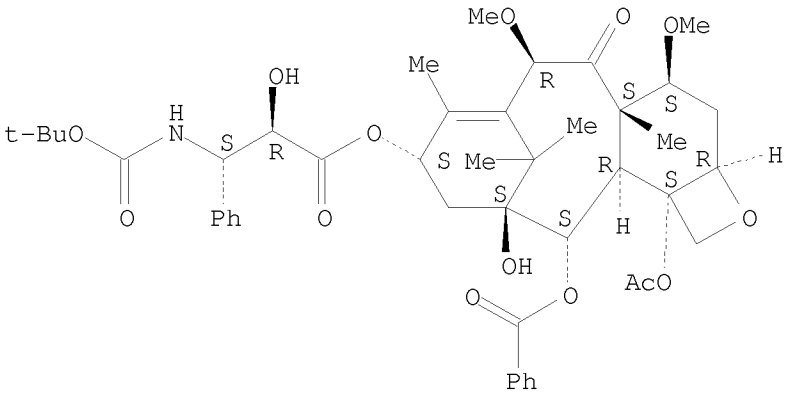
AB TXD258 (I), a new taxoid **antitumor** agent, is a poor substrate for the P-glycoprotein (P-gp) in Caco-2 cells. In this study, we investigated the amount of drug accumulating in the brains of rats and mice under a variety of conditions (dose and infusion time, species and plasma concentration) using conventional in vivo pharmacokinetic techniques and in situ brain perfusion. Mice were infused with radiolabeled TXD258 at 15, 30, 45 and 90 mg m-2 for 45 s or 1 h and rats were infused with 15 and 60 mg m-2 over 2.3 min. The radioactivity in the plasma and brains was measured. The brain concns. of TXD258 in mice and rats were maximal from 2 min to 1 h postinfusion and radioactivity was still detectable at 168 h. While the plasma concentration of TXD258 increased linearly in mice with the infused dose, the brain content increased more than proportionally with the dose between 15 and 90 mg m-2. This nonlinear uptake of TXD258 also occurred in the plasma and brain of the rat. These findings suggest that the protein-mediated efflux across the blood-brain barrier (BBB) becomes saturated In situ brain perfusion studies confirmed that TXD258 is a P-gp substrate at the BBB of mice and rats. The P-gp of both species was saturated at the half-inhibitory concentration (.apprx. 13 μM) produced by i.v. infusion. Thus, the observed nonlinear accumulation of TXD258 in the brain seems to occur by saturation of the P-gp at the rodent BBB. This saturation could have several advantages, such as overcoming a P-gp-mediated efflux, but the nonlinear pharmacokinetics could increase the risk of toxicity.

IT **183133-96-2, TXD 258**
RL: **PKT (Pharmacokinetics)**; BIOL (Biological study)
(nonlinear accumulation in brain of taxoid TXD258 following saturation of P-glycoprotein at the blood-brain barrier in mice and rats)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 49 THERE ARE 49 CAPLUS RECORDS THAT CITE THIS RECORD (49 CITINGS)
REFERENCE COUNT: 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 100 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN
ACCESSION NUMBER: 2000:133500 HCAPLUS
DOCUMENT NUMBER: 132:175817
TITLE: New use of taxoid derivatives for treating abnormal cell proliferation in the brain
INVENTOR(S): Bissery, Marie-Christine; Vrignaud, Patricia; Roberts, Simon; Brealey, Clive
PATENT ASSIGNEE(S): Rhone-Poulenc Rorer S.A., Fr.
SOURCE: PCT Int. Appl., 35 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000009120	A1	20000224	WO 1999-EP6291	19990813 <--
W: AE, AL, AU, BA, BB, BG, BR, CA, CN, CR, CU, CZ, DM, EE, GD, GE, HR, HU, ID, IL, IN, IS, JP, KP, KR, LC, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, RU, SG, SI, SK, SL, TR, TT, UA, US, UZ, VN, YU, ZA, AM, AZ, BY, KG, KZ, MD, TJ, TM				
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EP 982027	A1	20000301	EP 1998-115401	19980817 <--
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EP 982028	A1	20000301	EP 1998-115650	19980820 <--
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US 6346543	B1	20020212	US 1999-371838	19990811 <--
CA 2340921	A1	20000224	CA 1999-2340921	19990813 <--
CA 2340921	C	20090106		
AU 9957419	A	20000306	AU 1999-57419	19990813 <--
AU 766168	B2	20031009		
BR 9913025	A	20010508	BR 1999-13025	19990813 <--
EP 1109547	A1	20010627	EP 1999-944531	19990813 <--
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     TR 2001000557        T2   20010723    TR 2001-557             19990813 <--
     HU 2001003748        A2   20020429    HU 2001-3748            19990813 <--
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     EE 4903              B1   20071015
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             CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
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     BR 9913071           A    20010515    BR 1999-13071           19990818 <--
     EP 1105119           A2   20010613    EP 1999-944532          19990818 <--
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     MX 2001001721        A    20050603    MX 2001-1721            20010215 <--
     BG 105264            A    20011130    BG 2001-105264          20010216 <--
     BG 65365             B1   20080430
     ZA 2001001414        A    20010821    ZA 2001-1414            20010220 <--
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PRIORITY APPLN. INFO.:                      EP 1998-115401       A  19980817 <--
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                                            US 1999-60123843     P  19990311 <--
                                            EP 1998-115650       A  19980820 <--
                                            WO 1999-EP6291       W  19990813 <--
                                            WO 1999-EP6292       W  19990818 <--
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S):         MARPAT 132:175817
GI
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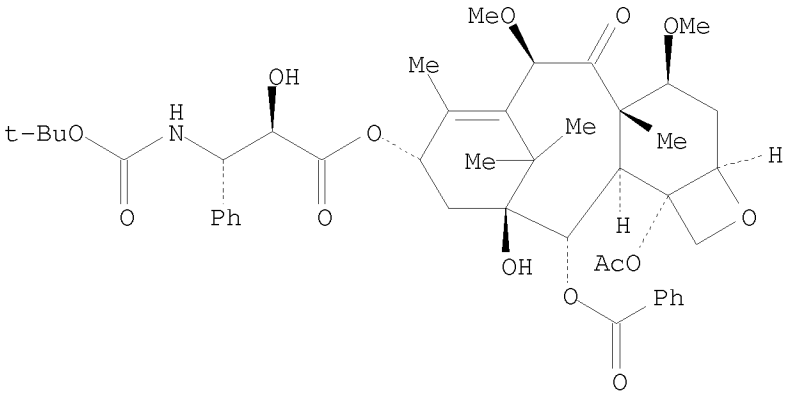


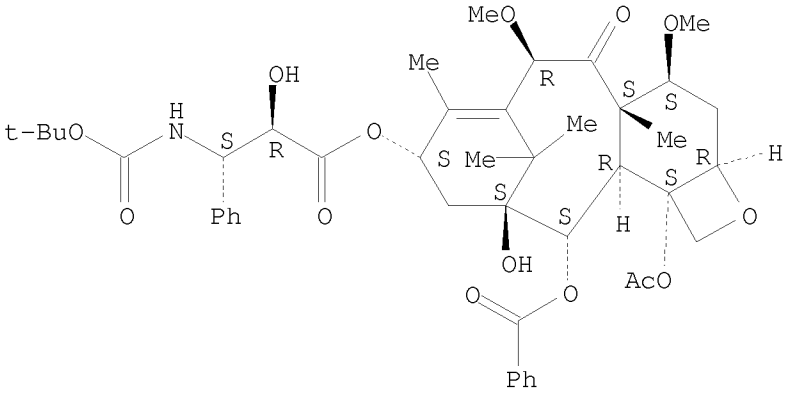
AB The invention relates to a new use of taxoid derivs. It relates more precisely to a method for treating abnormal cell proliferation in the brain of mammals by administering a taxoid derivative

IT **183133-96-2**
RL: **BAC (Biological activity or effector, except adverse)**; BSU (Biological study, unclassified); **THU (Therapeutic use)**; BIOL (Biological study); USES (Uses)
(taxoid derivs. for **treating** abnormal cell proliferation in brain)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



OS.CITING REF COUNT: 5 THERE ARE 5 CAPLUS RECORDS THAT CITE THIS RECORD (5 CITINGS)

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L15 ANSWER 101 OF 101 HCAPLUS COPYRIGHT 2013 ACS on STN

ACCESSION NUMBER: 1996:687356 HCAPLUS

DOCUMENT NUMBER: 125:329087

ORIGINAL REFERENCE NO.: 125:61651a,61654a

TITLE: Novel taxoids as **antitumor** agents
INVENTOR(S): Bouchard, Herve; Bourzat, Jean-Dominique; Commercon, Alain
PATENT ASSIGNEE(S): Rhone-Poulenc Rorer S.A., Fr.
SOURCE: PCT Int. Appl., 61 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: French
FAMILY ACC. NUM. COUNT: 10
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9630355	A1	19961003	WO 1996-FR440	19960325 <--
W: AL, AU, BB, BG, BR, CA, CN, CZ, EE, FI, GE, HU, IS, JP, KP, KR, LK, LR, LT, LV, MG, MK, MN, MX, NO, NZ, PL, RO, SG, SI, SK, TR, TT, UA, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
FR 2732340	A1	19961004	FR 1995-3545	19950327 <--
FR 2732340	B1	19970430		
FR 2742754	A1	19970627	FR 1995-15381	19951222 <--
FR 2742754	B1	19980116		
IL 117636	A	20040831	IL 1996-117636	19960324 <--
IL 159378	A	20050517	IL 1996-159378	19960324 <--
CA 2214319	A1	19961003	CA 1996-2214319	19960325 <--
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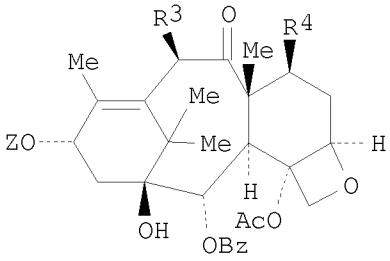
ASSIGNMENT HISTORY FOR US PATENT AVAILABLE IN LSUS DISPLAY FORMAT
OTHER SOURCE(S): MARPAT 125:329087
GI



I

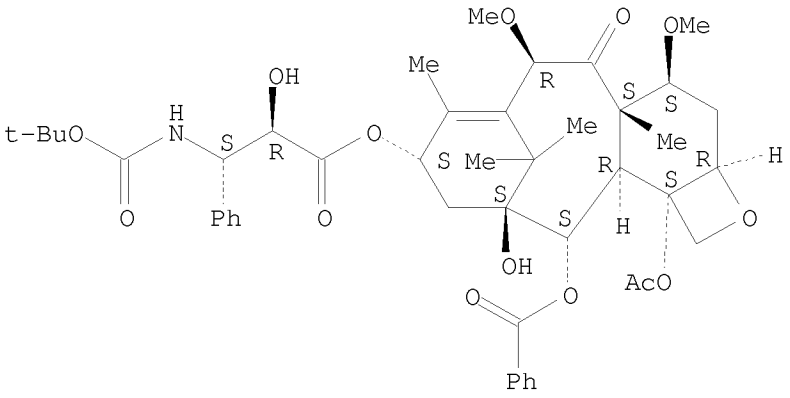
AB Novel taxoids I [Z = H, (2R,3S)-R1NHCHR2CH(OH)CO; R1 = acyl, esterified carboxyl; R2 = (un)substituted alkyl, alkenyl, alkynyl, cycloalkyl, Ph, naphthyl; R3, R4 = (un)substituted alkoxy, alkenyloxy, alkynyloxy, cycloalkyloxy, cycloalkenyloxy] were prepared for use as **antitumor** and antileukemic agents (no data). Thus, I [R1 = CO2CMe3, R2 = Ph, R3, R4 = OMe] was prepared from 10-deacetylbaccatin III and 3-tert-butoxycarbonyl-2-(4-methoxyphenyl)-4-phenyl-1,3-oxazolidine-5-carbolic acid in 6 steps.

IT **183133-96-2P**
RL: SPN (Synthetic preparation); **THU (Therapeutic use)**; BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of new taxoids as **antitumor** agents)

RN 183133-96-2 HCAPLUS

CN Benzenepropanoic acid, β-[[(1,1-dimethylethoxy)carbonyl]amino]-α-hydroxy-, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-12b-(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-11-hydroxy-4,6-dimethoxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester, (αR,βS)- (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).



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REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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(FILE 'HOME' ENTERED AT 10:55:45 ON 10 SEP 2013)

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L4 QUE E1-E20

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STN INTERNATIONAL LOGOFF AT 11:01:31 ON 10 SEP 2013

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of: **Gupta et al.**

Examiner: **James ANDERSON**

Art Unit: **1629**

Application No.: **13/456,720**

Conf No. **1083**

Filed: **April 26, 2012**

Title: **NOVEL ANTITUMORAL USE OF CABAZITAXEL**

REPLY TO OFFICE ACTION PURSUANT TO 37 C.F.R. § 1.111

This paper is in response to the Office Action dated January 16, 2013, having a response due by April 16, 2013. The period for response is extended three months to expire July 16, 2013, pursuant to the Petition for Extension of Time under 37 C.F.R. 1.136(a) submitted herewith. This response is timely filed.

Entry of the following amendments and consideration of the following remarks are respectfully requested.

Amendments to the specification start on page 2.

Amendments to the claims start on page 3.

Remarks to amendments and the outstanding office action begin on page 6.

Amendment Pursuant to 37 C.F.R. § 1.121

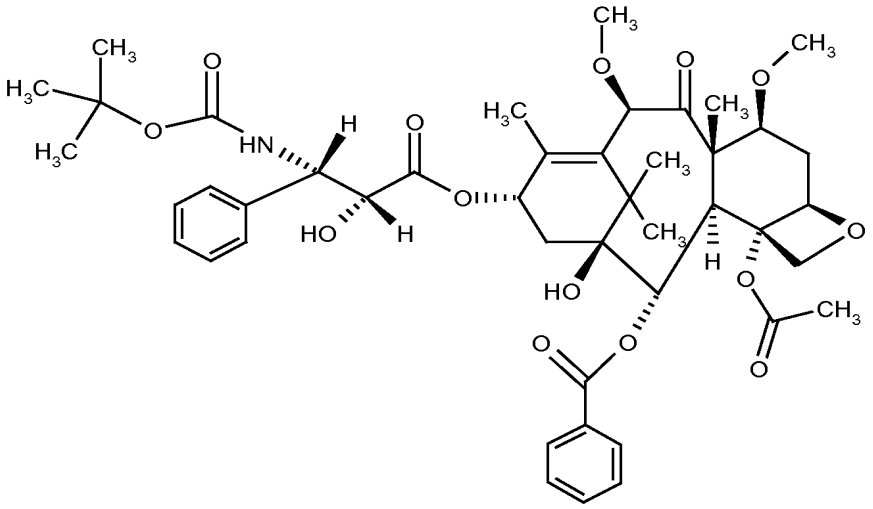
In the Specification:

Please replace the paragraph beginning at page 7, line 1 of the specification with the following rewritten paragraph:

Cabazitaxel may be administered in base form (cf. above formula), or in the form of a hydrate. It may also be a solvate, i.e. a molecular complex characterized by the incorporation of the crystallization solvent into the crystal of the molecule of the active principle (see in this respect page 1276 of *J. Pharm. Sci.* 1975, *64*(8), 1269-1288). In particular, it may be an acetone solvate, and, more particularly, may be the solvate described in WO **2005/028462** ~~**2005/02846**~~. It may be an acetone solvate of cabazitaxel containing between 5% and 8% and preferably between 5% and 7% by weight of acetone (% means content of acetone/content of acetone+cabazitaxel × 100). An average value of the acetone content is 7%, which approximately represents the acetone stoichiometry, which is 6.5% for a solvate containing one molecule of acetone. The procedure described below allows the preparation of an acetone solvate of cabazitaxel:

In the Claims:

1. (Currently amended) A method for treating prostate cancer in a patient in need thereof comprising administering to said patient a compound of formula



which may be in base form or in the form of a hydrate or a solvate,

in combination with prednisone or prednisolone, wherein said patient has hormone refractory metastatic prostate cancer and wherein said patient has been previously treated with a docetaxel containing regimen.

2. (Original) The method according to claim 1, where the treated patients are not catered for by a taxane-based treatment.

3. (Cancelled)

4. (Original) The method according to claim 1, where the prostate cancer is an advanced metastatic disease.

5. (Cancelled)

6. (Original) The method according to claim 1, where the compound is in the form of an acetone solvate.

7. (Original) The method according to claim 6, in which the acetone solvate contains between 5% and 8% by weight of acetone.

8. (Original) The method according to claim 1, where the compound is administered at a dose of between 15 and 25 mg/m^2, the prednisone or prednisolone being administered at a dose of 10 mg/day.

9. (Original) The method according to claim 8, where the compound is administered at a dose of 25 mg/m^2.

10. (Original) The method according to claim 1, comprising repeating the administration of such compound as a new cycle every 3 weeks.

11. (Original) The method according to claim 10, wherein the median number of cycles is 6.

12. (Cancelled)

13. (Currently amended) The method according to claim 1~~claim 12~~, where the compound is cabazitaxel.

14. (Original) The method according to claim 1, wherein said compound is administered in an amount to provide an AUC of about 991 ng•h/mL (CV 34%).

15. (Original) The method according to claim 1, wherein said compound is administered in an amount to provide an C_{max} of about 226 ng•h/mL (CV 107%).

16. (Original) The method according to claim 1 wherein said compound is administered in an amount to provide a plasma clearance of 48.5 L/h (CV 39%).

17. (Original) The method according to claim 1, further comprising monitoring blood counts and measuring neutrophil levels in the patient.

18. (Original) The method according to Claim 17, wherein said monitoring comprises taking a blood sample from the patient.

19. (Original) The method according to Claim 18, further comprising discontinuing cabazitaxel treatment in a patient with a neutrophil count of ≤1,500 cells/mm^3.

20. - 23. (Cancelled)

24. (Original) A method of increasing the survival of a patient with hormone refractory metastatic prostate cancer, comprising administering a clinically proven effective amount of a compound as defined in claim 1 to the patient in combination with prednisone or prednisolone.

25. – 33. (Cancelled)

Remarks

In the Office Action, the Examiner noted that claims 1 to 33 are pending in the application, that claims 20 to 23 and 25 to 33 are withdrawn from consideration, and that claims 1 to 19 and 24 are rejected.

The specification is amended to replace the reference "WO2005/02846" with "WO2005/028462" to correct an inadvertent typographical error. A copy of the WO2005/028462 publication is provided herewith with the attached IDS, for the convenience of the Examiner.

Claim 1 is amended to more specifically define the patient. Support for this amendment can be found throughout the specification and in the original claims, for example in original claims 3, 5 and 12.

Claims 3, 5 and 12 are cancelled without prejudice.

Claim 13 is amended to change its dependency from herein cancelled claim 12 to claim 1, from which claim 12 originally depended.

Claims 20 to 23 and 25 to 33, which are directed to non-elected subject matter, are cancelled without prejudice.

No new matter is added by these amendments.

Applicant reserves the right to file one or more continuation, continuation-in-part, or divisional applications on the deleted subject matter.

As presently amended, claims 1, 2, 4, 6 to 11, 13 to 19 and 24 are pending in this application.

Discussion of Objection to the Specification

The specification is objected to for the given reason that "[a]t page 7, first paragraph, Applicant makes reference to 'WO 2005/02846' as describing acetone solvates of cabazitaxel. It is unclear what WIPO document this is referring to as the number is incomplete." (Office Action, page 3).

The objection to the specification is believed overcome in view of the above-described amendment wherein the WIPO reference number has been corrected. Reconsideration and withdrawal of this objection are therefore respectfully requested.

Discussion of Rejection under 35 U.S.C. § 103(a)

Claims 1 to 5, 8 to 19 and 24 are rejected under 35 U.S.C. § 103(a) as being allegedly unpatentable over Mita et al. (Clin Cancer Res, 2009, 15(2) pp. 723-730, hereinafter "Mita") in view of Tannock et al. (N Eng J Med, 2004, 351, pp. 1502-1512, hereinafter "Tannock"). This rejection is traversed.

It is the Examiner's position that one would have been motivated to "combine the teachings of the references so as to administer cabazitaxel in combination with prednisone" because "Mita et al. teach that cabazitaxel is effective in treating prostate cancer metastatic to liver and bones whose disease has progressed through surgical castration, bicalutamide, diethery stilbestrol, and mitoxantrone and predisone and hormone- and docetaxel-refractory prostate caner metastatic to bone and iliac lymph nodes when administered as a single agent." (Office Action, page 8). Further, it is the Examiner position that the "motivation to add prednisone to such treatment is clearly seen in Tannock et al., who teach that administration of the taxane, docetaxel, in combination with prednisone is effective in treating hormone-refractory prostate cancer." (Id.).

To render a claimed invention obvious under 35 U.S.C. § 103, the cited reference themselves, coupled with the knowledge generally available in the art at the time of the invention, must contain some suggestion or incentive that would have motivated the skilled artisan to combine or modify them in the manner necessary to arrive at the claimed invention (*See,* MPEP § 2143.01). In addition, the proposed combination or modification must have had a reasonable expectation of success, determined from the vantage point of the skilled artisan at the time the invention was made. (*See,* MPEP § 2143.02). Finally, the prior art references must teach or suggest all limitations of the claims; i.e., each of the limitations must "be found in the prior art, and not be based on applicant's disclosure." (MPEP § 2143).

Applicant submits that the claimed elements of the present invention were not known in the prior art and the combination of Mita and Tannock would not have provided a reasonable expectation of predictable results. Accordingly, Applicant respectfully submits that any presumption of obviousness based on the combination of these references is not warranted.

The present application, which describes the results from a Phase III clinical trial, demonstrates that administration of cabazitaxel in combination with prednisone to patients with hormone refractory metastatic prostate cancer, who were previously treated with a docetaxel-containing regimen resulted in a statistically significant longer overall survival compared to patients receiving a mitoxantrone plus prednisone. (*See*, Specification, p. 18).

The primary Mita reference describes Phase I and pharmacokinetic studies of cabazitaxel in a limited number of patients with a variety of solid tumors. The studies were designed to evaluate the safety and dosage of cabazitaxel, but "preliminary evidence of antitumor activity" was to be documented. (Mita at 724, left column). While eight of the twenty-five patients has prostate tumors (Id. at 725, Table 1), Mita indicated that evidence of anticancer activity was noted in two patients, including one patient with "hormone- and docetaxel-refractory prostate cancer metastatic to bone and iliac lymph nodes." (Id. at 727). However, it is important to note that Mita nowhere suggests that one skilled in the art should use cabazitaxel for the treatment of prostate cancer based on these results, as the efficacy data provided is only "preliminary" evidence. Accordingly, given the extremely limited nature of the patients described in Mita and the complexity of treatment of cancer, one skilled in the art would not have the requisite reasonable expectation that the results of this phase 1 trial would translate to patients with hormone refractory metastatic prostate cancer, who were previously treated with a docetaxel-containing regimen when evaluated in a statistically relevant setting (such as a Phase III trial).

The secondary reference Tannock describes positive results of a comparison study of docetaxel or mitoxantrone plus prednisone in patients with advanced prostate cancer. Purportedly in view of this reference, the Examiner argues that "the skilled artisan would predict that addition of prednisone to the treatment regimen of Mita et al. would also be effective in treating hormone-refractory prostate cancer, including prostate cancers refractory to docetaxel therapy" (Office Action, page 8, emphasis added).

Applicants again note that Mita simply provides insufficient evidence to show that cabazitaxel is effective for treating hormone-refractory prostate cancer.

Moreover, Mita clearly indicates that "routine use of corticosteroids...were not permitted." (Mita at p. 724, right column). Furthermore, there is nothing in Tannock which would provide one skilled in the art with the reasonable expectation (or even prediction as asserted in the Office Action), that a combination comprising docetaxel would have any similar effectiveness when used in combination with cabazitaxel. The Office Action provides no evidence or even arguments explaining why one skilled in the art would reasonably have such an expectation, especially in patients with docetaxel-resistant prostate cancer.

Therefore, Applicant respectfully submits that, based on the preliminary and limited nature of description of effectiveness with respect to cabazitaxel in patients and the lack of evidence of any correlation between docetaxel- and cabazitaxel-based prednisone combinations, the present claims would be non-obvious to one skilled in art over the combination of Mita and Tannock. Accordingly, reconsideration and withdrawal of this obviousness-based rejection are respectfully requested.

Claims 6 and 7 are rejected under 35 U.S.C. § 103(a) as being allegedly unpatentable over Mita, in view of Tannock as applied to claims 1 to 5, 8 to 19 and 24 and further in view of Didier et al. (US2005/0065138).

Didier et al. which is cited for allegedly teaching "acetone solvates of cabazitaxel" and "acetone solvates containing between 5% and 8% if acetone" (Office Action, page 9), dose not remedy the deficiencies of Mita and Tannock, as described above. Accordingly, Didier et al., in combination with Mita and Tannock does not render claims 6 and 7 obvious. Reconsideration and withdrawal of this rejection of claims 6 and 7 are therefore respectfully requested.

Conclusion

There being no remaining issues, this application is believed in condition for favorable reconsideration and early allowance, and such actions are earnestly solicited.

In the event the Examiner wishes to contact the undersigned regarding any matter, please call (collect if necessary) the telephone number listed below.

The Director is hereby authorized to charge any additional fees which may be required by this paper, or credit any overpayment to Deposit Account No. **18-1982**.

Respectfully submitted,

/Kelly L. Bender/
Kelly Bender, Reg. No. 52,610
Attorney for Applicant

Sanofi US
U.S. Patent Operations
55 Corporate Drive
Mail Code: 55A-505A
Bridgewater, New Jersey 08807
email: **uspatent.e-filing@sanofi.com**
Telephone: (908) 981-**6782**
Telefax: (908) 981-7832
Sanofi US Ref. FR2009/121 US CNT

Date: July 16, 2013

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Application of:
GUPTA, et al.

Examiner:
James D. Anderson

Application No.:
13/456,720

Art Unit:
1629

Filed:
April 26, 2012

Title: **NOVEL ANTITUMORAL USE OF CABAZITAXEL**

CERTIFICATE OF EFS-WEB TRANSMISSION

I hereby certify that the correspondence below is being transmitted via the USPTO's electronic filing system in accordance with 1.6(a)(4), on

July 16, 2013
Date of Deposit

/Brian Pritchett/
Signature

TO: Commissioner for Patents
P. O. Box 1450
Alexandria, VA 22313-1450

Attached are the following documents:

			Number of Pages
☐	Application Data Sheet		
☐	Declaration		
☐	Drawings		
☒	Extension of Time		2
☒	Supplemental Information Disclosure Statement and Form 1449		5
☒	Response to Non-Final Office Action		10
☐	Specification, Claims and Abstract	Specification	
		Claims	
		Abstract	
☐	Transmittal Letter:		
☒	Other *(specify):* **REFERENCES**		19
☐	Other *(specify):*		
☐	Other *(specify):*		

PTO/AIA/22 (10-12)
Approved for use through 1/31/2013. OMB 0651-0031
U.S. Patent and Trademark Office; U.S. DEPARTMENT OF COMMERCE

PETITION FOR EXTENSION OF TIME UNDER 37 CFR 1.136(a)

Docket Number (Optional): FR2009/121 US CNT

Application Number	Filed
13/456,720	2012-04-26

For: NOVEL ANTITUMORAL USE OF CABAZITAXEL

Art Unit	Examiner
1629	James D. Anderson

This is a request under the provisions of 37 CFR 1.136(a) to extend the period for filing a reply in the above-identified application.

The requested extension and fee are as follows (check time period desired and enter the appropriate fee below):

	Fee	Small Entity Fee	
☐ One month (37 CFR 1.17(a)(1))	$150	$75	$
☐ Two months (37 CFR 1.17(a)(2))	$570	$285	$
☒ Three months (37 CFR 1.17(a)(3))	$1,290	$645	$ 1,290.00
☐ Four months (37 CFR 1.17(a)(4))	$2,010	$1,005	$
☐ Five months (37 CFR 1.17(a)(5))	$2,730	$1,365	$

☐ Applicant claims small entity status. See 37 CFR 1.27.

☐ A check in the amount of the fee is enclosed.

☐ Payment by credit card. Form PTO-2038 is attached.

☐ The Director has already been authorized to charge fees in this application to a Deposit Account.

☒ The Director is hereby authorized to charge any fees which may be required, or credit any overpayment, to Deposit Account Number 18-1982.

☐ Payment made via EFS-Web.

WARNING: Information on this form may become public. Credit card information should not be included on this form. Provide credit card information and authorization on PTO-2038.

I am the

☐ applicant.

☐ attorney or agent of record. Registration number _____.

☒ attorney or agent acting under 37 CFR 1.34. Registration number 52,610.

/Kelly L. Bender/ — Signature

07-16-2013 — Date

Kelly L. Bender — Typed or printed name

908-981-6782 — Telephone Number

NOTE: This form must be signed in accordance with 37 CFR 1.33. See 37 CFR 1.4 for signature requirements and certifications. Submit multiple forms if more than one signature is required, see below*.

☐ * Total of _____ forms are submitted.

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The **Privacy Act of 1974 (P.L. 93-579)** requires that you be given certain information in connection with your submission of the attached form related to a patent application or patent. Accordingly, pursuant to the requirements of the Act, please be advised that: (1) the general authority for the collection of this information is 35 U.S.C. 2(b)(2); (2) furnishing of the information solicited is voluntary; and (3) the principal purpose for which the information is used by the U.S. Patent and Trademark Office is to process and/or examine your submission related to a patent application or patent. If you do not furnish the requested information, the U.S. Patent and Trademark Office may not be able to process and/or examine your submission, which may result in termination of proceedings or abandonment of the application or expiration of the patent.

The information provided by you in this form will be subject to the following routine uses:

1. The information on this form will be treated confidentially to the extent allowed under the Freedom of Information Act (5 U.S.C. 552) and the Privacy Act (5 U.S.C 552a). Records from this system of records may be disclosed to the Department of Justice to determine whether disclosure of these records is required by the Freedom of Information Act.
2. A record from this system of records may be disclosed, as a routine use, in the course of presenting evidence to a court, magistrate, or administrative tribunal, including disclosures to opposing counsel in the course of settlement negotiations.
3. A record in this system of records may be disclosed, as a routine use, to a Member of Congress submitting a request involving an individual, to whom the record pertains, when the individual has requested assistance from the Member with respect to the subject matter of the record.
4. A record in this system of records may be disclosed, as a routine use, to a contractor of the Agency having need for the information in order to perform a contract. Recipients of information shall be required to comply with the requirements of the Privacy Act of 1974, as amended, pursuant to 5 U.S.C. 552a(m).
5. A record related to an International Application filed under the Patent Cooperation Treaty in this system of records may be disclosed, as a routine use, to the International Bureau of the World Intellectual Property Organization, pursuant to the Patent Cooperation Treaty.
6. A record in this system of records may be disclosed, as a routine use, to another federal agency for purposes of National Security review (35 U.S.C. 181) and for review pursuant to the Atomic Energy Act (42 U.S.C. 218(c)).
7. A record from this system of records may be disclosed, as a routine use, to the Administrator, General Services, or his/her designee, during an inspection of records conducted by GSA as part of that agency's responsibility to recommend improvements in records management practices and programs, under authority of 44 U.S.C. 2904 and 2906. Such disclosure shall be made in accordance with the GSA regulations governing inspection of records for this purpose, and any other relevant (*i.e.*, GSA or Commerce) directive. Such disclosure shall not be used to make determinations about individuals.
8. A record from this system of records may be disclosed, as a routine use, to the public after either publication of the application pursuant to 35 U.S.C. 122(b) or issuance of a patent pursuant to 35 U.S.C. 151. Further, a record may be disclosed, subject to the limitations of 37 CFR 1.14, as a routine use, to the public if the record was filed in an application which became abandoned or in which the proceedings were terminated and which application is referenced by either a published application, an application open to public inspection or an issued patent.
9. A record from this system of records may be disclosed, as a routine use, to a Federal, State, or local law enforcement agency, if the USPTO becomes aware of a violation or potential violation of law or regulation.

(19) Europäisches Patentamt
European Patent Office
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(11) EP 2 177 630 A1

(12) EUROPEAN PATENT APPLICATION

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C12Q 1/68 *(2006.01)*

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(71) Applicant: **Institut Gustave Roussy**
94800 Villejuif (FR)

(72) Inventors:
- **Chauchereau, Anne**
  92260 Fontenay-aux-Roses (FR)
- **Al Nakouzi, Nader**
  92763 Antony (FR)

(74) Representative: **Gallois, Valérie et al**
Cabinet BECKER & ASSOCIES
25, rue Louis Le Grand
75002 Paris (FR)

(54) Methods for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family

(57) The present invention concerns *in vitro* methods for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family based on a resistance expression signature, kits for performing the methods, and methods for screening or identifying a compound suitable for improving the treatment of a cancer with a molecule of the taxoid family or for reducing the resistance development during the treatment of a cancer with the molecule of the taxoid family

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Description

FIELD OF THE INVENTION

[0001] The present invention relates to method for predicting the response to a treatment with a molecule of the taxoid family, kits and method for screening compounds useful for improve the treatment with the molecule.

BACKGROUND OF THE INVENTION

[0002] Prostate cancer became, based on frequency and in Western countries, the first cancer in men, behind the lung cancer. This disease is the second cause of cancer death in men. Since 2005, more than 60,000 men are touched by prostate cancer (PCa) each year and 10,000 men died of this disease. The efficiency of docetaxel chemotherapy (Taxotere®) in prostate cancer (CaP) has been demonstrated for the first time in 2004 in two clinical trials, i.e. TAX 327 and SWOG 99-16, with an increase in survival. Accordingly, docetaxel became today a treatment of choice of metastatic hormone-refractory prostate cancers and phase III clinical trials are ongoing to assess its efficacy for the treatment of high-risk localized prostate cancer. Taxotere® is currently approved in 5 different cancer types in Europe and the US: Prostate cancer, breast cancer, lung cancer, gastric cancer and head and neck cancer. However, in spite of the survival benefit provided by this molecule, docetaxel has a great toxicity and almost half of the patients treated with docetaxel develop a resistance to the chemotherapy either from the beginning, or in a secondary way. Moreover, docetaxel is not effective on all the types of cancer. For instance, in case of breast cancer, only 30 to 50% of the metastatic tumours respond to docetaxel. Resistance to taxanes is common and there is an increasing need to try and identify those patients who will respond to treatment.

[0003] A genomic analysis was performed with two cell lines (PC3 and DU145) resistant to a docetaxel dose of 11 nM (Patterson et al, Oncogene, 2006, 25: 6113-6122). The article discloses an expression signature of 30 genes. The authors also demonstrated the effect of STAT1 and Clusterin in an *in vitro* model for the docetaxel resistance. However, the validation of the expression of these two genes in the docetaxel-resistance has not been performed on tumours. The authors further demonstrated that resveratrol leads to a decreased expression of clusterin in docetaxel resistant cells and, then to an increase of apoptosis (Sallman et al, Mol. Can. Ther., 2007, 6 : 2938-2947). Other groups used docetaxel resistance cell lines (PC3-R) in their research (Lo Nigro et al, BJU Int., 2008, 102 : 622-7). Some other groups used prostate cancer cell lines treated during a short period (24-72 h) with docetaxel for studying the role of genes in the docetaxel response.

[0004] In addition, a patent application WO 2006/062811 concerns a method for measuring resistance or sensitivity to docetaxel.

[0005] Therefore, there is still a strong need of a diagnostic method for predicting responsiveness to docetaxel and avoiding useless treatments. Indeed, before the initiation of the treatment, it is currently impossible to identify the patients who will respond to or who will have a resistance to docetaxel.

SUMMARY OF THE INVENTION

[0006] The present invention provides an expression signature specific of the docetaxel resistance in human prostate cancer. Based on this signature, the present invention provides a method for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family.

[0007] Accordingly, the present invention concerns an *in vitro* method for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family, wherein the method comprises: 1) providing a biological sample from said subject; 2) determining in the biological sample the expression level of at least 5 genes selected from the group consisting of the genes listed in Tables 1 and 2, thereby predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family. Optionally, the method comprises determining the expression level of at least 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes from those listed in Tables 1 and 2. Preferably, the cancer is selected from the group consisting of the breast cancer, the lung cancer, the prostate cancer, the gastric cancer and the head and neck cancer. More preferably the cancer is the prostate cancer. Preferably, the expression level is compared to a reference expression level, for instance the expression level of the genes in cell-lines or patients sensitive to the treatment by the molecule of the taxoid family. In particular, the over-expression of genes from Table 1 and/or the under-expression of genes from Table 2 are indicative of a resistance to the treatment by the molecule of the taxoid family. The expression level of genes can be determined by the quantity of protein or mRNA encoded by said genes. Preferably, the biological sample is a cancer sample.

[0008] Preferably, the at least 5 genes are selected from one of the following groups or a combination thereof:

a) RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3,

MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, WNT2B, GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and LOC152573, preferably RPIB9, CXCL2, TFPI2, TNF, ABCB1, ADAMTS5, PURG, OAS3, GAS1, BIRC3, MAL, GALNT14, TM4SF1, RXFP1, ATP8A1, SOX9, SLC39A8, EDG7, ITGA2, SLC1A3, CALCRL and LOC152573;

b) RPIB9, TFPI2, ABCB1, BIRC3, WNT2B, SFRP1, FSTL1, AHR, CDKN1C, ABCB2, CYR61, WNT5A, ABCC3, JAG1, STAT1, WNT7B, CASP8, LZTS1, FZD8, GALNT14, RXFP1 and LOC152573;

c) ABCC3, CD55, COL16A1, DHRS3, FSTL1, GLS, HDL, HIVE1, LAMA2, LAMB3, LIPG, LITAF, MAL, MFHAS1, NFKBIZ, NRP1, NRP2, OAS3, OLR1, PSCDBP, RFTN1, SCARAB1, SEMA3B, SEMA3C, SFRP11, SLC1A3, ST6GAL1, TLR3, TM4SF1 and TNF;

d) ADAMTS1, ADRA2C, AKAP12, CDKN1C, CYR61, FBN1, GAS1, GPC3, IGF2, IGFBP3, JAG1, MGST1, NTN4, PDE1A, PDE4B, PDE4D, PDE4DIP, PDGFB, PHLDA1, PIM1, PPP2R2C, RGS16, SCD, SLC1A1, SMPDL3A, TFPI2 and VCAN;

e) ABCB1, AHR, AHRR, AMPH, BIRC3, CXCL2, CYP1A1, IL1R1, NQO1, PLAT, PLXNA2, SLC16A10, SLC3A1, SLC7A8, SLPI, TAP1, UGT8, UGT2B4, UGT2B7, UGT2B10, UGT2B11 and UGT2B28;

f) AQP1, ARHGDIB, BAMBI, CREB5, CXCR4, EPAS1, FGF2, FGFBP1, GRB10, IL15, MT2A, NUPR1, PDK1, PROS1, PTPN3, RPS6KA2, TFDP2, WNT2B, WNT5A and WNT7B;

g) AGT, ATP8A2, BDNF, EDG6, GAL, GATA2, ITGA2, LRP11, LZTS1, MYB, NCALD, PNOC, PTGES, SRGAP3, TAC3 and TTN;

h) AFF1, ASGR1, BLVRA, CASP8, CD40, KCNH2, NRG1, NRL, PHEX, PLAC8, SMAD7, SMAD9, SOX9, SPG20 and STAT1;

i) TNF, ABCB11, CYP1A1, AHRR, AHR, PP2R2C, ABCC3, NQO1, PIK3C3, UGT2B7, UGT2B11, UGT2B28, UGT2B4, UGT2B10, CHST7, MGST11 and UGT8; and,

j) Wnt2B, Wnt5A, Wnt7B, SFRP1, FSTL1, Jag1, Cyr61, LOC152573, FZD8 and FOXL2.

[0009] In a preferred embodiment, the molecule of the taxoid family is selected from the group consisting of docetaxel, larotaxel, XRP6258, BMS-184476, BMS-188797, BMS-275183, ortataxel, RPR 109881A, RPR 116258, NBT-287, PG-paclitaxel, ABRAXANE®, Tesetaxel, IDN 5390, Taxoprexin, DHA-paclitaxel, and MAC-321. More preferably, the molecule of the taxoid family is docetaxel.

[0010] The present invention also concerns kits and DNA chips suitable for this method. Accordingly, the present invention concerns a kit for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family, wherein the kit comprises detection means selected from the group consisting of a pair of primers, a probe and an antibody specific to at least 5 genes selected from the group consisting of the genes listed in Tables 1 and 2 or a DNA chip comprising a solid support which carries nucleic acids that are specific to at least 5 genes selected from the group consisting of the genes listed in Tables 1 and 2. Preferably, the at least 5 genes of the kit or DNA chip according to the present invention are selected from one of the following groups or a combination thereof:

a) RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, WNT2B, GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and LOC152573, preferably RPIB9, CXCL2, TFPI2, TNF, ABCB1, ADAMTS5, PURG, OAS3, GAS1, BIRC3, MAL, GALNT14, TM4SF1, RXFP1, ATP8A1, SOX9, SLC39A8, EDG7, ITGA2, SLC1A3, CALCRL and LOC152573;

b) RPIB9, TFPI2, ABCB11, BIRC3, WNT2B, SFRP11, FSTL1, AHR, CDKN1C, ABCB2, CYR61, WNT5A, ABCC3, JAG1, STAT1, WNT7B, CASP8, LZTS1, FZD8, GALNT14, RXFP1 and LOC152573;

c) ABCC3, CD55, COL16A1, DHRS3, FSTL1, GLS, HDL, HIVE1, LAMA2, LAMB3, LIPG, LITAF, MAL, MFHAS1, NFKBIZ, NRP1, NRP2, OAS3, OLR1, PSCDBP, RFTN1, SCARAB1, SEMA3B, SEMA3C, SFRP11, SLC1A3, ST6GAL1, TLR3, TM4SF1 and TNF;

d) ADAMTS1, ADRA2C, AKAP12, CDKN1C, CYR61, FBN1, GAS1, GPC3, IGF2, IGFBP3, JAG1, MGST1, NTN4, PDE1A, PDE4B, PDE4D, PDE4DIP, PDGFB, PHLDA1, PIM1, PPP2R2C, RGS16, SCD, SLC1A1, SMPDL3A, TFPI2 and VCAN;

e) ABCB1, AHR, AHRR, AMPH, BIRC3, CXCL2, CYP1A1, IL1R1, NQO1, PLAT, PLXNA2, SLC16A10, SLC3A1, SLC7A8, SLPI, TAP1, UGT8, UGT2B4, UGT2B7, UGT2B10, UGT2B11 and UGT2B28;

f) AQP1, ARHGDIB, BAMBI, CREB5, CXCR4, EPAS1, FGF2, FGFBP1, GRB10, IL15, MT2A, NUPR1, PDK1, PROS1, PTPN3, RPS6KA2, TFDP2, WNT2B, WNT5A and WNT7B;

g) AGT, ATP8A2, BDNF, EDG6, GAL, GATA2, ITGA2, LRP11, LZTS1, MYB, NCALD, PNOC, PTGES, SRGAP3, TAC3 and TTN;

h) AFF1, ASGR1, BLVRA, CASP8, CD40, KCNH2, NRG1, NRL, PHEX, PLAC8, SMAD7, SMAD9, SOX9, SPG20

and STAT1;
i) TNF, ABCB11, CYP1A1, AHRR, AHR, PP2R2C, ABCC3, NQO1, PIK3C3, UGT2B7, UGT2B11, UGT2B28, UGT2B4, UGT2B10, CHST7, MGST11 and UGT8; and,
j) Wnt2B, Wnt5A, Wnt7B, SFRP1, FSTL1, Jag1, Cyr61, LOC152573, FZD8 and FOXL2.

[0011] The present invention further concerns methods for screening or identifying a compound suitable for improving the treatment of a cancer with a molecule of the taxoid family or for reducing the resistance development during the treatment of a cancer with a molecule of the taxoid family. In a first embodiment, the method comprises: 1) providing a cell-line with at least 5 genes over-expressed and/or under-expressed respectively selected from the group of over-expressed genes of Table 1 and under-expressed genes of Table 2; 2) contacting said cell-line with a test compound; 3) determining the expression level of said at least 5 genes; and, 4) selecting the compound which decreases the expression level of over-expressed genes and increases the expression level of under-expressed genes. In a second embodiment, the method comprises: 1) providing a cell-line sensitive to the molecule of the taxoid family; 2) contacting said cell-line with a test compound and the molecule of the taxoid family; 3) determining the expression level of said at least 5 genes selected from the genes listed in Tables 1 and 2; and, 4) selecting the compound which inhibits the appearance of an over-expression and/or an under-expression of at least 5 genes respectively selected from the group of genes of Table 1 and genes of Table 2. In a third embodiment, the method comprises: 1) providing a cell-line with at least on gene over-expressed and/or under-expressed respectively selected from the group consisting of RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB11, PURG, ADAMTS5, MCTP11, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, and WNT2B for the over-expressed genes, and GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and LOC152573 for the under-expressed genes; 2) contacting said cell-line with a test compound; 3) determining the expression level of said at least one gene; and, 4) selecting the compound which decreases the expression level of over-expressed genes and increases the expression level of under-expressed genes. Preferably, the molecule of the taxoid family is selected from the group consisting of docetaxel, larotaxel, XRP6258, BMS-184476, BMS-188797, BMS-275183, ortataxel, RPR 109881A, RPR 116258, NBT-287, PG-paclitaxel, ABRAXANE®, Tesetaxel, IDN 5390, Taxoprexin, DHA-paclitaxel, and MAC-321. More preferably, the molecule of the taxoid family is docetaxel.

BRIEF DESCRIPTION OF THE DRAWINGS

[0012]

FIGURE 1 : RT PCR validation of the RPIP9 transcript. RPIP9 is the most over-expressed gene of the resistant phenotype. This gene shows a 34 fold change on the micro-array. In quantitative RT PCR, the expression of this gene showed a 450 fold ratio with the probe 1 (Taqman Applied Hs00379227_ml) and more than 1000 fold ratio with the probe 2 (Taqman Applied Hs00289927_ml). NT : without docetaxel treatment.
FIGURE 2 : RT PCR validation of the ABC proteins expression. ABCB1 transcript (Mdr-1) codes for P-gp1 protein which is an ATP-dependent membrane transporter responsible of cellular efflux of substances, in particular anti-tumoral drugs. This gene is frequently over-expressed in resistant phenotype. This gene belongs to the 10 most over-expressed genes in the present signature. At the highest docetaxel concentration, this gene showed a 16.22 fold change on the microarray. FIG 2A : In quantitative RT-PCR (QRT-PCR), the expression of this gene shows a ratio up to 2000 x (Taqman Applied HS00184491_ml). FIG 2B : The P-gp1 protein is also found to be over-expressed in a dose-dependent manner in resistant IGR-CaP1 cells at various doses of docetaxel. FIG 2C : The genes coding for two other proteins of the same family, i.e., ABCB2 and ABCC3, are also over-expressed, with lower fold changes. ABCB2 has a mean fold change of 3.6 on the microarray and a ratio up to 8.3 in QRT-PCR (Taqman Applied Hs00388682_ml). ABCC3 has a mean fold change of 3.1 on the microarray and a ratio up to 14.3 in QRT-PCR (Taqman Applied Hs00358656_ml).
FIGURE 3 : RT PCR validation of BIRC3 and TFPI2 gene expression. These two genes belong to the 15 most over-expressed genes in the present signature with a fold-change of 10.4 and 21.8, respectively. By QRT-PCR, the expression of these genes shows a ratio of up to 36 x (Taqman Applied Hs00154109_ml) and 64 x (Taqman Applied Hs00197918_ml), respectively.
FIGURE 4 : RT PCR validation of the expression of STAT1, Clusterin, AHR and CDKN1C genes. FIG 4A : The gene encoding STAT1 shows a fold-change of 2.47 on the microarray and a fold change up to 5 by RT PCR (Taqman Applied Hs00234829_ml). The clusterin gene is slightly over-expressed in the resistant cells (Taqman Applied Hs00156548_ml). FIG 4B : Over-expression of nuclear proteins AHR (Taqman Applied Hs00907314_ml) and CDKN1C (Taqman Applied Hs00175938_ml) with a fold change on microarray of 5.4 and 5.38 on the microarray, respectively.

FIGURE 5 : RT PCR validation of under-expressed genes in the signature. GALNT14 gene (Taqman Applied Hs00226180_ml) belongs to the most under-expressed genes in the present signature with a fold-change - 10.26 on the microarray. The gene encoding Caspase 8 is also found to be under-expressed by QRT-PCR (Taqman Applied Hs01018151_ml) (Mean Fold change of - 4.51 on the microarray).

FIGURE 6 : Validation of under-expressed LZST1 gene. FIG 6A : LZST1 gene has been found to be under-expressed by QRT-PCR (Taqman Applied Hs00232762_ml) (Mean Fold change of - 4.53 on the microarray). FIG 6B : Whereas LZST1 protein is present in sensitive cells, it is absent in resistant cells at high docetaxel concentrations.

FIGURE 7 : RT PCR validation of under-expressed LOC152573 gene. FIG 7A : the LOC152573 gene encoding the human homolog of SHISA3 is the most under-expressed gene of the present signature (Mean Fold change of - 159.4 on the microarray). QRT-PCR analysis confirms this result in docetaxel resistant IGR-CaP1 cells (Taqman Applied Hs01380806_ml). FIG 7B : A strong decrease of the hSHISA gene expression has also been observed in LNCaP cells resistant to 2.5 nM of docetaxel and PC3 cells resistant to 0.5 nM of docetaxel.

FIGURE 8 : RT PCR validation of the expression of Wnt pathway genes belonging to the present signature. FIG 8A and FIG 8B : two Taqman primers were used for determining the amount of the two forms of Wnt2B (S1 : Taqman Applied Hs00244632_ml ; S2 : Taqman Applied Hs00257131_ml). FIG 8C : Genes encoding the other members of the Wnt family. Wnt5a and Wnt7b are over-expressed in a less extent (Taqman Applied Wnt5a : Hs00998537_ml ; Wnt7b : Hs00536497_ml). FIG 8D : Genes encoding other members of the Wnt pathway (Taqman Applied SFRP1 : Hs00610060_ml; FSTL1 : Hs00200053_ml; Jag1 : Hs01070032_ml). FIG 8E : The gene encoding the Frizzled 8 receptor (FDZ8) is under-expressed in docetaxel resistant cells (Taqman Applied : Hs00259040_s1).

DETAILED DESCRIPTION OF THE INVENTION

[0013] The present invention provides the identification of protein coding genes involved in the mechanism of docetaxel resistance in prostate cancer treatment. The inventors prepared *in vitro* cellular models of docetaxel resistant prostate cancer by selecting cell clones by pharmaceutical pressure from several cellular model of prostate cancer (i.e., LNCap, PC3 and IGR-CaP1 cell lines). IGR-CaP1 cell line became resistant to increasing doses of docetaxel (0.5nM ; 5nM ; 12nM ; 25nM, 50nM ; 100nM ; 200nM). LNCaP and PC3 cell lines became resistant to docetaxel concentrations of 0.5 nM and 2.5 nM. A micro-array genomic analysis was performed by comparing sensitive and resistant IGR-CaP1 cell lines at four docetaxel concentrations (5; 12; 25 and 50 nM). This analysis led to the identification of 378 genes associated with the resistant phenotype for all the docetaxel concentrations (by 2D clusterization with a P value $<10^{-10}$, genes with fold change > 2). In this signature, 191 genes were over-expressed and 187 genes were under-expressed. The over-expression of some signature genes was confirmed by quantitative RT-PCR (e.g., genes RPIP9; ABCB1; ABCB2; ABCC3; BIRC3; TFPI2; AHR; STAT1 ; CDKN1 ; WNT2B; WNT5A; WNT7B; SFRP1; FSTL1; Jag1) and/or by Western blot (ABCB1 protein expression). The over-expression of some genes of this signature has also been observed over-expressed by RT PCR in LNCaP cell line resistant to docetaxel concentrations of 0.5 nM and 2.5nM (e.g., ABCB1 and WNT2B genes). The under-expression of some signature genes was also confirmed by quantitative RT-PCR (e.g., genes GALNT14; LZTS1; LOC152573; FZD8) and/or by Western blot (LZTS1 protein expression). The under-expression of some genes of this signature has also been observed underexpressed by RT PCR in LNCaP cell line resistant to docetaxel concentrations of 0.5 nM and 2.5nM and PC3 cell line resistant to a docetaxel concentration of 0.5 nM (e.g., LOC152573/hSHISA3 gene). On this basis, the inventors identified a set of genes whose combined expression profiles allow to distinguish patients between responder and non-responder to a treatment with a molecule of the taxoid family. A "responder" or "responsive" patient refers to a patient who shows or will show a clinically significant recovery when treated in the cancer when treated with a molecule of the taxoid family. In particular, the size of the tumor will no more increase, decrease or the tumor will disappear.

[0014] Therefore, the present invention discloses an expression signature useful for *in vitro* method for predicting whether a patient suffering of a cancer would be responsive to a treatment with a molecule of the taxoid family. The method comprises determining the expression level of genes from the present expression signature (see Tables 1 and 2) in a biological sample of said patient. In particular, the method comprises determining the expression level of at least 5 genes of Tables 1 and 2 in a biological sample of said patient. Preferably, the method comprises determining the expression level of at least 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes of Tables I and 2. Alternatively, the method comprises determining the expression level of 5 to 378 genes of Tables 1 and 2, optionally of 7 to 370, 8 to 360, 9 to 350, 10 to 325, 15 to 300, 20 to 250, 30 to 200, 40 to 150, 50 to 100, 60 to 90 or 70 to 80.

[0015] By "predicting" or "prediction" is intended herein the likelihood that a patient will respond or not to a molecule of the taxoid family and also the extent of the response. Predictive methods of the invention can be used clinically to make treatment decisions by choosing the most appropriate treatment modalities for any particular patient. Therefore, the present invention also concerns a method for selecting a patient suffering of a cancer for a treatment with a molecule of the taxoid family, comprising determining the expression level of at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes of Tables I and 2 in a biological sample of said patient and selecting the patient

predicted to be responsive to a treatment with a molecule of the taxoid family.

[0016] In a first embodiment, the genes are selected from Tables 1 and 2 on the criteria of "fold change". Accordingly, the genes with the greatest fold change (in absolute value) are chosen. For instance, the genes associated with a fold change greater (in absolute value) than 2, preferably than 3, 4, 5, 6, 7, 8, 9 or 10, are selected. In a particular embodiment, the genes are selected from the group consisting of RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, WNT2B, GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and LOC152573, preferably from the group consisting of RPIB9, CXCL2, TFPI2, TNF, ABCB1, ADAMTS5, PURG, OAS3, GAS1, BIRC3, MAL, GALNT14, TM4SF1, RXFP1, ATP8A1, SOX9, SLC39A8, EDG7, ITGA2, SLC1A3, CALCRL and LOC152573. Alternatively, the genes can be selected among the genes validated by RT-PCR, in particular in the group consisting of RPIB9, TFPI2, ABCB1, BIRC3, WNT2B, SFRP1, FSTL1, AHR, CDKN1C, ABCB2, CYR61, WNTSA, ABCC3, JAG1, STAT1, WNT7B, CASP8, LZTS1, FZD8, GALNT14, RXFP1 and LOC152573.

[0017] In a second embodiment, the genes are selected from Tables 1 and 2 on the criteria of a network, that is to say that the genes are selected in one particular network. Accordingly, the genes can be selected in the group consisting of one of the following networks or a combination thereof comprising:

1) ABCC3, CD55, COL16A1, DHRS3, FSTL1, GLS, HDL, HIVE1, LAMA2, LAMB3, LIPG, LITAF, MAL, MFHAS1, NFKBIZ, NRP1, NRP2, OAS3, OLR1, PSCDBP, RFTN1, SCARAB1, SEMA3B, SEMA3C, SFRP1, SLC1A3, ST6GAL1, TLR3, TM4SF1 and TNF;
2) ADAMTS1, ADRA2C, AKAP12, CDKN1C, CYR61, FBN1, GAS1, GPC3, IGF2, IGFBP3, JAG1, MGST1, NTN4, PDE1A, PDE4B, PDE4D, PDE4DIP, PDGFB, PHLDA1, PIM1, PPP2R2C, RGS16, SCD, SLC1A1, SMPDL3A, TFPI2 and VCAN;
3) ABCB1, AHR, AHRR, AMPH, BIRC3, CXCL2, CYP1A1, IL1R1, NQO1, PLAT, PLXNA2, SLC16A10, SLC3A1, SLC7A8, SLPI, TAP1, UGT8, UGT2B4, UGT2B7, UGT2B10, UGT2B11 and UGT2B28;
4) AQP1, ARHGDIB, BAMBI, CREB5, CXCR4, EPAS1, FGF2, FGFBP1, GRB10, IL15, MT2A, NUPR1, PDK1, PROS1, PTPN3, RPS6KA2, TFDP2, WNT2B, WNT5A and WNT7B;
5) AGT, ATP8A2, BDNF, EDG6, GAL, GATA2, ITGA2, LRP11, LZTS1, MYB, NCALD, PNOC, PTGES, SRGAP3, TAC3 and TTN; and
6) AFF1, ASGR1, BLVRA, CASP8, CD40, KCNH2, NRG1, NRL, PHEX, PLAC8, SMAD7, SMAD9, SOX9, SPG20 and STAT1.

[0018] In a third embodiment, the genes are selected from Tables 1 and 2 on the criteria of their belonging to the signaling pathway of xenobiotic metabolism. Accordingly, the genes can be for instance selected from the group consisting of TNF, ABCB1, CYP1A1, AHRR, AHR, PP2R2C, ABCC3, NQO1, PIK3C3, UGT2B7, UGT2B11, UGT2B28, UGT2B4, UGT2B10, CHST7, MGST1 and UGT8. In another embodiment, the genes are selected from Tables 1 and 2 because of their membership to the Wnt pathway. Accordingly, the genes can be for instance selected from the group consisting of Wnt2B, Wnt5A, Wnt7B, SFRP1, FSTL1, Jag1, Cyr61, LOC152573, FZD8 and FOXL2.

[0019] Of course, the genes can also be selected from a combination of these particular groups.

[0020] The method can comprise the step of comparing the expression levels of the genes determined in the sample to reference or control expression levels. The reference or control expression levels are determined with a sample of cells, preferably cancer cells, which are sensitive to the molecule of the taxoid family. Alternatively, reference or control expression levels are determined with a sample of patients or subjects sensitive to the treatment with the molecule of the taxoid family. Hence, an over-expressed gene herein refers to a gene having an increased expression in comparison to the expression level of this gene in a sensitive cell, and an under-expressed gene herein refers to a gene having a decreased expression in comparison to the expression level of this gene in a sensitive cell. However, the man skilled in art understands that other references can be used. For instance, the invention also contemplates a reference level corresponding to the expression level in a cell resistant to the molecule of the taxoid family.

[0021] In particular, when the genes selected from the Table 1 are over-expressed, one can predict that the patient would be resistant to a treatment with a molecule of the taxoid family. On the contrary, when the genes selected from the Table 1 are not over-expressed, one can predict that the patient would be responsive to a treatment with a molecule of the taxoid family. At the opposite, when the genes selected from the Table 2 are under-expressed, one can predict that the patient would be resistant to a treatment with a molecule of the taxoid family. On the contrary, when the genes selected from the Table 2 are not under-expressed, one can predict that the patient would be responsive to a treatment with a molecule of the taxoid family.

[0022] In addition, the genes can be selected in such a way that they comprise some over-expressed genes and some under-expressed ones. In this embodiment, the selected genes can comprise at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, or 150 genes of Table 1 and at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90,

100, or 150 genes of Table 2. Alternatively, they can be selected in such a way that they comprise only over-expressed or under-expressed genes. In a preferred embodiment, the genes are selected among the genes having the greatest fold change.

[0023] In addition to the genes selected from Tables 1 and 2, the method can also comprise the determination of the expression level for control genes. The control genes are chosen among the genes known to have a constant expression level, in particular between sensitive and resistant cells to a molecule of the taxoid family. In addition, the expression level of at least one control gene is determined in order to normalize the result. For instance, the control gene can be GAPDH, 18S RNA, beta-actine or lamin.

[0024] The molecule of the taxoid family refers to a class of anti-tumoral drugs belonging to the taxane family. It can be selected from the group consisting of paclitaxel, docetaxel and analogs, prodrugs or formulations thereof. In particular, analogs, prodrugs or formulations thereof can be for instance selected in the group consisting of larotaxel (also called XRP9881; Sanofi-Aventis), XRP6258 (Sanofi-Aventis), BMS-184476 (Bristol-Meyer-Squibb), BMS-188797 (Bristol-Meyer-Squibb), BMS-275183 (Bristol-Meyer-Squibb), ortataxel (also called IDN 5109, BAY 59-8862 or SB-T-101131 ; Bristol-Meyer-Squibb), RPR 109881A (Bristol-Meyer-Squibb), RPR 116258 (Bristol-Meyer-Squibb), NBT-287 (TAPESTRY), PG-paclitaxel (also called CT-2103, PPX, paclitaxel poliglumex, paclitaxel polyglutamate or Xyotax™), ABRAXANE® (also called Nab-Paclitaxel ; ABRAXIS BIOSCIENCE), Tesetaxel (also called DJ-927), IDN 5390 (INDENA), Taxoprexin (also called docosahexanoic acid-paclitaxel ; PROTARGA), DHA-paclitaxel (also called Taxoprexin®), and MAC-321 (WYETH). Also see the review of Hennenfent & Govindan (2006, Annals of Oncology, 17, 735-749). In a preferred embodiment of the present invention, the molecule of the taxoid family is the docetaxel.

[0025] The expression level of the selected genes can be determined by measuring the amounts of RNA, in particular mRNA, DNA, in particular cDNA, or protein using a variety of techniques well-known by the man skilled in art.

[0026] The cancer can be selected from the group consisting of the breast cancer, the lung cancer, the prostate cancer, the gastric cancer and the head and neck cancer. In a preferred embodiment, the cancer is the prostate cancer.

[0027] The term "biological sample" means any biological sample derived from a patient, preferably a sample which contains nucleic acids or proteins. Examples of such samples include fluids, tissues, cell samples, organs, biopsies, etc. Most preferred samples are cancer tissue samples, in particular breast, lung, prostate, stomach, ovary or head and neck tumor samples. Blood, plasma, saliva, urine, seminal fluid, etc, may also be used. Cancer cells obtain form blood as circulating tumor cells may also be used. The biological sample may be treated prior to its use, e.g. in order to render nucleic acids or proteins available. Techniques of cell lysis, concentration or dilution of nucleic acids or proteins, are known by the skilled person.

[0028] Generally, the expression level as determined is a relative expression level (mRNA or protein).

[0029] More preferably, the determination comprises contacting the sample with selective reagents such as probes, primers or ligands, and thereby detecting the presence, or measuring the amount, of proteins or nucleic acids of interest originally in the sample. Contacting may be performed in any suitable device, such as a plate, microtiter dish, test tube, well, glass, column, and so forth. In specific embodiments, the contacting is performed on a substrate coated with the reagent, such as a nucleic acid array or chip or a specific ligand array. The substrate may be a solid or semi-solid substrate such as any suitable support comprising glass, plastic, nylon, paper, metal, polymers and the like. The substrate may be of various forms and sizes, such as a slide, a membrane, a bead, a column, a gel, etc. The contacting may be made under any condition suitable for a detectable complex, such as a nucleic acid hybrid or an antibody-antigen complex, to be formed between the reagent and the nucleic acids or proteins of the sample.

[0030] In a preferred embodiment, the expression level may be determined by determining the quantity of mRNA.

[0031] Methods for determining the quantity of mRNA are well known in the art. For example the nucleic acid contained in the samples (e.g., cell or tissue prepared from the patient) is first extracted according to standard methods, for example using lytic enzymes or chemical solutions or extracted by nucleic-acid-binding resins following the manufacturer's instructions. The extracted mRNA is then detected by hybridization (e. g., Northern blot analysis) and/or amplification (e.g., RT-PCR). Preferably quantitative or semi-quantitative RT-PCR is preferred. Real-time quantitative or semi-quantitative RT-PCR is particularly advantageous.

[0032] Other methods of Amplification include ligase chain reaction (LCR), transcription-mediated amplification (TMA), strand displacement amplification (SDA) and nucleic acid sequence based amplification (NASBA).

[0033] Nucleic acids having at least 10 nucleotides and exhibiting sequence complementarity or homology to the mRNA of interest herein find utility as hybridization probes or amplification primers. It is understood that such nucleic acids need not be identical, but are typically at least about 80% identical to the homologous region of comparable size, more preferably 85% identical and even more preferably 90-95% identical. In certain embodiments, it will be advantageous to use nucleic acids in combination with appropriate means, such as a detectable label, for detecting hybridization. A wide variety of appropriate indicators are known in the art including, fluorescent, radioactive, enzymatic or other ligands (e. g. avidin/biotin).

[0034] Probes typically comprise single-stranded nucleic acids of between 10 to 1000 nucleotides in length, for instance of between 10 and 800, more preferably of between 15 and 700, typically of between 20 and 500. Primers typically are

shorter single-stranded nucleic acids, of between 10 to 25 nucleotides in length, designed to perfectly or almost perfectly match a nucleic acid of interest, to be amplified. The probes and primers are "specific" to the nucleic acids they hybridize to, i.e. they preferably hybridize under high stringency hybridization conditions (corresponding to the highest melting temperature Tm, e.g., 50 % formamide, 5x or 6x SCC. SCC is a 0.15 M NaCl, 0.015 M Na-citrate). For instance, the probes and primers can be selected from the Taqman Applied ones cited in the present application.

[0035] The nucleic acid primers or probes used herein may be assembled as a kit. Such a kit includes consensus primers and molecular probes. A preferred kit also includes the components necessary to determine if amplification has occurred. The kit may also include, for example, PCR buffers and enzymes; positive control sequences, reaction control primers; and instructions for amplifying and detecting the specific sequences.

[0036] In another preferred embodiment, the expression level is determined by DNA chip analysis. Such DNA chip or nucleic acid microarray consists of different nucleic acid probes that are chemically attached to a substrate, which can be a microchip, a glass slide or a microsphere-sized bead. A microchip may be constituted of polymers, plastics, resins, polysaccharides, silica or silica-based materials, carbon, metals, inorganic glasses, or nitrocellulose. Probes comprise nucleic acids such as cDNAs or oligonucleotides that may be about 10 to about 60 base pairs. To determine the expression level, a sample from a test subject, optionally first subjected to a reverse transcription, is labelled and contacted with the microarray in hybridization conditions, leading to the formation of complexes between target nucleic acids that are complementary to probe sequences attached to the microarray surface. The labelled hybridized complexes are then detected and can be quantified or semi-quantified. Labelling may be achieved by various methods, *e.g.* by using radioactive or fluorescent labelling. Many variants of the microarray hybridization technology are available to the man skilled in the art (see e.g. the review by Hoheisel, et 2006)

[0037] Other methods for determining the expression level of said genes include the determination of the quantity of proteins encoded by said genes.

[0038] Such methods comprise contacting a biological sample with a binding partner capable of selectively interacting with a marker protein present in the sample. The binding partner is generally an antibody, that may be polyclonal or monoclonal, preferably monoclonal.

[0039] The presence of the protein can be detected using standard electrophoretic and immunodiagnostic techniques, including immunoassays such as competition, direct reaction, or sandwich type assays. Such assays include, but are not limited to, Western blots; agglutination tests; enzyme-labeled and mediated immunoassays, such as ELISAs; biotin/avidin type assays; radioimmunoassays; immunoelectrophoresis; immunoprecipitation, etc. The reactions generally include revealing labels such as fluorescent, chemiluminescent, radioactive, enzymatic labels or dye molecules, or other methods for detecting the formation of a complex between the antigen and the antibody or antibodies reacted therewith.

[0040] The aforementioned assays generally involve separation of unbound protein in a liquid phase from a solid phase support to which antigen-antibody complexes are bound. Solid supports which can be used in the practice of the invention include substrates such as nitrocellulose (*e. g.*, in membrane or microtiter well form); polyvinylchloride (*e. g.*, sheets or microtiter wells); polystyrene latex (*e.g.*, beads or microtiter plates); polyvinylidine fluoride; diazotized paper; nylon membranes; activated beads, magnetically responsive beads, and the like.

[0041] More particularly, an ELISA method can be used, wherein the wells of a microtiter plate are coated with an antibody against the protein to be tested. A biological sample containing or suspected of containing the marker protein is then added to the coated wells. After a period of incubation sufficient to allow the formation of antibody-antigen complexes, the plate(s) can be washed to remove unbound moieties and a detectably labeled secondary binding molecule added. The secondary binding molecule is allowed to react with any captured sample marker protein, the plate washed and the presence of the secondary binding molecule detected using methods well known in the art.

[0042] The invention further provides a tool for implementing said methods, e.g. a DNA chip comprising a solid support which carries nucleic acids that are specific to at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes selected from the group consisting of the genes listed in Tables 1 and 2. The DNA chip can further comprise nucleic acids for control gene, for instance a positive and negative control or a nucleic acid for an ubiquitous gene in order to normalize the results. In addition, the present invention also provides a kit for implementing said methods comprising detection means that are specific to at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes selected from the group consisting of the genes listed in Tables 1 and 2. In particular, the detection means can be a pair of primers, a probe or an antibody. The kit can further comprise control reagents and other necessary reagents.

[0043] In a particular embodiment, the genes are selected for the tool or kit as above detailed for the methods of the invention. Preferably, the at least 5 genes are selected from one of the following groups or a combination thereof:

a) RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, WNT2B, GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and

LOC152573, preferably RPIB9, CXCL2, TFPI2, TNF, ABCB1, ADAMTS5, PURG, OAS3, GAS1, BIRC3, MAL, GALNT14, TM4SF1, RXFP1, ATP8A1, SOX9, SLC39A8, EDG7, ITGA2, SLC1A3, CALCRL and LOC152573;

b) RPIB9, TFPI2, ABCB1, BIRC3, WNT2B, SFRP1, FSTL1, AHR, CDKN1C, ABCB2, CYR61, WNT5A, ABCC3, JAG1, STAT1, WNT7B, CASP8, LZTS1, FZD8, GALNT14, RXFP1 and LOC152573;

c) ABCC3, CD55, COL16A1, DHRS3, FSTL1, GLS, HDL, HIVE1, LAMA2, LAMB3, LIPG, LITAF, MAL, MFHAS1, NFKBIZ, NRP1, NRP2, OAS3, OLR1, PSCDBP, RFTN1, SCARAB1, SEMA3B, SEMA3C, SFRP1, SLC1A3, ST6GAL1, TLR3, TM4SF1 and TNF;

d) ADAMTS1, ADRA2C, AKAP12, CDKN1C, CYR61, FBN1, GAS1, GPC3, IGF2, IGFBP3, JAG1, MGST1, NTN4, PDE1A, PDE4B, PDE4D, PDE4DIP, PDGFB, PHLDA1, PIM1, PPP2R2C, RGS16, SCD, SLC1A1, SMPDL3A, TFPI2 and VCAN;

e) ABCB1, AHR, AHRR, AMPH, BIRC3, CXCL2, CYP1A1, IL1R1, NQO1, PLAT, PLXNA2, SLC16A10, SLC3A1, SLC7A8, SLPI, TAP1, UGT8, UGT2B4, UGT2B7, UGT2B10, UGT2B11 and UGT2B28;

f) AQP1, ARHGDIB, BAMBI, CREB5, CXCR4, EPAS1, FGF2, FGFBP1, GRB10, IL15, MT2A, NUPR1, PDK1, PROS1, PTPN3, RPS6KA2, TFDP2, WNT2B, WNT5A and WNT7B;

g) AGT, ATP8A2, BDNF, EDG6, GAL, GATA2, ITGA2, LRP11, LZTS1, MYB, NCALD, PNOC, PTGES, SRGAP3, TAC3 and TTN;

h) AFF1, ASGR1, BLVRA, CASP8, CD40, KCNH2, NRG1, NRL, PHEX, PLAC8, SMAD7, SMAD9, SOX9, SPG20 and STAT1;

i) TNF, ABCB1, CYP1A1, AHRR, AHR, PP2R2C, ABCC3, NQO1, PIK3C3, UGT2B7, UGT2B11, UGT2B28, UGT2B4, UGT2B10, CHST7, MGST1 and UGT8; and,

j) Wnt2B, Wnt5A, Wnt7B, SFRP1, FSTL1, Jag1, Cyr61, LOC152573, FZD8 and FOXL2.

[0044] The present invention also relates to the use of a DNA chip or a kit of the invention for preparing a kit for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family. Preferably, the cancer is selected from the group consisting of the breast cancer, the lung cancer, the prostate cancer, the gastric cancer and the head and neck cancer. More preferably the cancer is the prostate cancer. In a preferred embodiment, the molecule of the taxoid family is selected from the group consisting of docetaxel, larotaxel, XRP6258, BMS-184476, BMS-188797, BMS-275183, ortataxel, RPR 109881A, RPR 116258, NBT-287, PG-paclitaxel, ABRAXANE®, Tesetaxel, IDN 5390, Taxoprexin, DHA-paclitaxel, and MAC-321. More preferably, the molecule of the taxoid family is docetaxel.

[0045] The present invention further concerns methods for screening or identifying a compound suitable for improving the treatment of a cancer with a molecule of the taxoid family or for reducing the resistance development during the treatment of a cancer with a molecule of the taxoid family. In a first embodiment, the method comprises: 1) providing a cell-line with at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes over-expressed and/or under-expressed respectively selected from the group of over-expressed genes of Table 1 and under-expressed genes of Table 2; 2) contacting said cell-line with a test compound; 3) determining the expression level of said at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes; and, 4) selecting the compound which decreases the expression level of over-expressed genes and increases the expression level of under-expressed genes. In a second embodiment, the method comprises: 1) providing a cell-line sensitive to the molecule of the taxoid family; 2) contacting said cell-line with a test compound and the molecule of the taxoid family; 3) determining the expression level of said at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes selected from the genes listed in Tables 1 and 2; and, 4) selecting the compound which inhibits the appearance of an over-expression and/or an under-expression of at least 5, 6, 7, 8, 9, 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes respectively selected from the group of genes of Table 1 and genes of Table 2. In a third embodiment, the method comprises: 1) providing a cell-line with at least on gene over-expressed and/or under-expressed respectively selected from the group consisting of RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, and WNT2B for the over-expressed genes, and GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and LOC152573 for the under-expressed genes; 2) contacting said cell-line with a test compound; 3) determining the expression level of said at least one gene; and, 4) selecting the compound which decreases the expression level of over-expressed genes and increases the expression level of under-expressed genes.

[0046] Preferably, the cell-line is a cancer cell-line. In particular, the cancer cell-line is specific of the targeted cancer. For instance, if the prostate cancer is to be treated, then the cell-line is a prostate cancer cell-line.

[0047] In a preferred embodiment, the molecule of the taxoid family is selected from the group consisting of docetaxel, larotaxel, XRP6258, BMS-184476, BMS-188797, BMS-275183, ortataxel, RPR 109881A, RPR 116258, NBT-287, PG-paclitaxel, ABRAXANE®, Tesetaxel, IDN 5390, Taxoprexin, DHA-paclitaxel, and MAC-321. More preferably, the molecule of the taxoid family is docetaxel. Preferably, the cancer is selected from the group consisting of the breast cancer,

the lung cancer, the prostate cancer, the gastric cancer and the head and neck cancer. More preferably the cancer is the prostate cancer.

[0048] The example illustrates the invention without limiting its scope.

EXAMPLES

Methods

Cell culture and selection of docetaxel-resistant clones

[0049] The human androgen-independent prostate carcinoma cell line PC3 was obtained from American Type Culture Collection (Rockville, MD, USA) and was maintained in DMEM medium containing 10% heat-inactivated fetal bovine serum (FBS) with 100U/ml penicillin, 100 mg/ml streptomycin. The human androgeno-dependent prostate carcinoma cell line LNCaP was maintained in RPMI medium complemented with 10% FBS and antibiotics. The human androgen-independent IGR-CaP1 cell line recently obtained for a localized prostate cancer was maintained in RPMI medium complemented with 10% FBS and antibiotics. Docetaxel-resistant clones were selected by culturing the cells in docetaxel in a dose-escalation manner. Initial culture was done in 0.5nM docetaxel. Cellular clones surviving in the presence of 0.5nM docetaxel were maintained in culture during four passages, and then the concentration of docetaxel in the medium was increased to 2.5nM and subsequently to 12nM, 25nM, 50nM, 100nM and 200nM. The same selection methodology was followed with each increase in docetaxel concentration. Once cells were freely dividing in each dose of docetaxel mediums, they were considered as resistant and labelled IGR-CaP1-R, LNCaP-R and PC3-R. IGR-CaP1-R clones were obtained surviving in medium containing respectively 2.5nM, 5nM, 12nM, 25nM, 50nM, 100nM and 200nM docetaxel. LNCaP-R clones survived in medium containing 0.5nM, 2.5nM and 5nM docetaxel. PC3-R clones survived in medium containing 0.5nM and 2.5nM docetaxel. All the cell cultures were maintained at 70% confluency and medium was changed every 48 h.

Cell Cycle analysis

[0050] Effects of treatments on the stages of the cell cycle were determined using the PI staining technique. Briefly, parental and docetaxel-resistant IGR-CaP1 cells were grown in flasks at a density of $4x10^6$ cells. After allowing for overnight attachment the cells were treated or not with 12nM docetaxel. Cells were incubated for 48 hr then collected by trypsinization, making sure to include the floating cells. After washing in PBS the cells were fixed and permeabilized using Fix&Perm kit (InVitrogen) according to the manufacturer protocol. Cells were treated with 20μg DNAse-free RNAse for 30 min and stained with 100μg propidium iodide (PI) for 30 min. Then percentage of cells in G1, S, G2, M, and subG1 phases were analyzed with FACS Calibur cytometer (Becton Dickinson).

Total RNA Preparation and Reverse Transcription

[0051] Total RNA from parental and docetaxel-resistant IGR-CaP1 cells was isolated using TriReagent (Sigma-Aldrich) and purified with RNeasy Micro Kit (Qiagen) according to manufacturer's protocols. Quality of RNA preparation, based on the RNA Integrity Number (RIN), was assessed using the Agilent RNA 6000 Nano Kit as developed on the Agilent 2100 Bioanalyzer device (Agilent Technologies, Palo Alto, CA). All specimens included in this study displayed a RIN of 10. RNA samples were frozen in nuclease-free water (Qiagen).

Oligo Microarray Technology

[0052] Parental and resistant-cell line total RNAs were directly compared by using Agilent oligonucleotide dual-color technology, running dye-swap and duplicate experiments. Total RNA from the parental IGR-CaP1 cell line without treatment was used as the RNA reference. Total RNA from IGR-CaP1 cells resistant to treatment with 5nM, 12nM, 25nM and 50nM of docetaxel respectively, were used as samples. Probe synthesis and labeling were performed by Agilent's Low Fluorescent Low input Linear Amplification Kit. Hybridization was performed on the Agilent 4x44K Human 1A (G4112F) long (60-bp) oligonucleotide microarrays (Agilent Technologies) by using reagents and protocols provided by the manufacturer. Feature extraction software provided by Agilent (Version A.9.5.3.1) was used to quantify the intensity of fluorescent images and to normalize results using the linear and lowess subtraction method. Primary analysis was performed by using Resolver software (version 7.1) (Rosetta Laboratories, Milan) to identify genes differentially expressed between parental and resistant cell lines with a fold change > 2 and P value < 10^{-10}. Using this procedure for each of the 4 combined experiments, a list of 378 genes was extracted and was considered as a signature of gene potentially implicated in resistance to docetaxel. These genes were sorted out by the mean of the fold change observed respectively

for the 4 doses of resistance towards docetaxel.

TaqMan Real-Time Quantitative Reverse Transcription-PCR Analysis.

[0053] Real-time quantitative RT-PCR was performed using the ABI Prism 7900 Sequence Detection System (Perkin-Elmer Applied Biosystems). The same procedure was applied from the total RNA used in the microarray analysis and for independent RNA samples. One μg of total RNA was reversed transcribed using the GeneAmp RNA PCR Kit according to the manufacturer's recommendations (Applied Biosystems).

[0054] Quantitative real-time PCR was performed in a final volume of 25 μl according to the manufacturer's recommendations (Applied Biosystems). PCR primers and probe for the selected target genes were designed by Applied Biosystems and used according to the manufacturer's recommendations. The amount of sample RNA was normalized by the amplification of an endogenous control (18S). The relative quantification of the transcripts was derived by using the standard curve method (Applied Biosystems User Bulletin 2, ABI PRISM 7700 Sequence Detection System). The following Taqman probes were used : RPIB9 Hs00379227_ml and Hs00289927_m1; ABCB1 Hs00184491_m1; ABCB2 Hs00388682_ml; ABCC3 Hs00358656_ml: BIRC3 Hs00154109_ml; TFPI2 Hs00197918_ml; STAT1 Hs00234829_ml; CLU Hs00156548_ml, AHR Hs00907314_ml; CDKN1C Hs00175938_m1; GALNT14 Hs00226180_m1; CASP8 Hs01018151_ml; LZTS1 Hs00232762_ml; LOC152573 Hs01380806_ml; WN2B Hs00244632_ml and Hs00257131_ml; WNT5A Hs00998537_ml; WNT7B Hs00536497_ml; SFRP1 Hs00610060_ml; FSTL1 Hs00200053_ml; JAG1 Hs01070032_ml; FDZ8 Hs00259040_s1.

Western blot analysis

[0055] Parental and resistant cellular clones were cultured in 175cm2 flask in the presence of the appropriate concentration of docetaxel. Cells were lysed in RIPA buffer to prepare whole cell extracts and denatured in NuPage LDS sample buffer (Invitrogen). Protein concentration of the soluble extracts was determined by using the MicroBCA protein assay (Pierce).

[0056] Proteins from 50μg of whole cell extracts were resolved by electrophoresis on NuPage 4-12% Bis-Tris gels (Invitrogen) and immunoblots were developed using the enhanced chemoluminescence-based detection kit (Pierce). The following antibodies were used: anti-ABCB1 (Mdr-1 D-11) and anti-LZTS1 (FEZ1 C-20) from Santa-Cruz Biotechnology Inc. The equal loading of protein sample was verified with a β-actin-specific antibody (Sigma).

RESULTS

[0057] **Generation of acquired resistance to Docetaxel *in vitro*.** Prostate cancer IGR-CaP1 cells were used to generate successive docetaxel-resistant cell lines. The addition of docetaxel induced a selection process, whereby a large majority of cells initially underwent cell death until the ability to proliferate was regained. The inventors obtained IGR-CaP1 resistant (IGR-CaP1-R) clones which survived in medium containing respectively 5nM, 12nM, 25nM, 50nM of docetaxel. Cell cycle analysis was done to show acquired resistance to drug. The resistant cell lines showed cell cycle similar to the parental IGR-CaP1 cells, suggesting that acquired resistance had been gained (not shown).

[0058] **Genome-wide analysis of docetaxel-resistant lines using microarray**. Human genome-wide analysis of gene expression changes was realized in order to stringently identify human genes that might represent the molecular signature of resistance or sensitivity to docetaxel in prostate cancer. Untreated IGR-CaP1 parental cell lines were used as baseline. Hierarchical clustering of combined experiments using a 2-fold change criteria and a *P* value of $<10^{-10}$ revealed a total of 378 genes that were up or down-regulated by >2-fold in each of the resistant cell lines. 191 genes were over-expressed (Table 1) and 187 were down-regulated (Table 2) in docetaxel-resistant cells. These genes were sorted out by the mean of the fold change observed respectively for the 4 doses of resistance towards docetaxel (Table 3 and Table 4).. Functional analysis of the resistant cell lines was performed using Ingenuity® Pathways Analysis (IPA). Highly significant functions and canonical pathways were found for resistant cell lines as organ development, cancer, cellular growth and proliferation, cellular movement, cell-to-cell signalling and interaction, or cell death.

Target verification by real-time RT-PCR and western blot

[0059] To verify the alterations of gene expression at the mRNA level, which appeared on the microarray, the inventors chose representative genes with varying expression profiles for real-time Taqman RT-PCR and Western Blot analysis. The inventors measured gene expression levels in a panel of 22 genes.

[0060] The inventors first measured the expression of the Top gene of the signature, RPIP9/RPIB9/RUNDC3B, encoding Rap2-binding protein 9. Two sets of probes were chosen to measure gene expression of RPIB9 as multiple splice variants were transcribed (Fig 1A). The two probes showed a high level in gene expression in docetaxel-resistant cells

in a dose dependent manner (Fig1B). Over expression was more pronounced (more than 1000 fold) with the 3' probe set, suggesting that long variants containing RUN domain were more expressed. The same results were obtained on an independent set of total RNAs (not shown). The function of RPIP9 protein is not known but RPIP9 gene was shown to be overexpressed in breast carcinoma and correlated with a poor prognosis.

[0061] A key mechanism underlying multidrug resistance relates to the overexpression of the ATP-dependent transporter family known as the ATP-binding cassette (ABC) family. One of the most described members of these drug efflux pumps was the P-glycoprotein (P-gp) encoded by the MDR-1 gene. This gene had been frequently found overexpressed in drug-resistant phenotype. The gene ABCB1/MDR1 is one of the most over-expressed genes of the signature. The same alterations of gene expression were observed by real-time RT-PCR analysis, although the fold change in the expression level was much higher (Fig 2A). The same results were obtained on an independent set of total RNAs (not shown). Western blot analysis showed that expression of the MDR1 gene product was increased in a dose-dependent manner in docetaxel-resistant cells (Fig 2B). Two other genes encoding members of the ATP-binding cassette family, ABCB2 and ABCC3 were confirmed to be overexpressed in resistant cells, although to a lesser level compared to the ABC gene (Fig 2C). Interestingly, ABCC3 was recently identified as a mediator of taxane resistance in HER2-amplified breast cancer.

[0062] The genes BIRC3 and TFPI2 were found in the Top 15 of over-expressed genes of the signature with a fold-change expression of 10.4 and 21.8 respectively in the resistant cells. BIRC3 encoding baculoviral IAP repeat-containing 3 belongs to a family of proteins that inhibits apoptosis (IAP family). Interestingly, it had been suggested that IAP proteins may have an important contribution to the resistance to the apoptotic effect of cisplatin in prostate cancer. The TFPI2 gene, encoding tissue factor pathway inhibitor 2, is a potent inhibitor of matrix-metalloproteinase. This protein was shown to be most prominently up-regulated in MYCN-amplified neuroblastomas. RT-PCR analysis confirmed that BIRC3 and TFPI2 genes were overexpressed in taxane-resistant cells up to 36 fold and 64 fold respectively (Fig 3).

[0063] As the genes STATE1 and clusterin were showed overexpressed in DU145-DR and PC3-DR docetaxel-resistant cells in the study of Patterson et al., (2006), the inventors verified the expression level of these two genes by RT-PCR in the IGR-CaP1-R model. STAT1 was also found overexpressed in the present signature with a fold change of 2.47 but Clusterin was not retained in the present microarray analysis. As shown in Fig 4A, the genes STAT1 and Clusterin were up-regulated in IGR-CaP1-R resistant cells, although to a modest extent. CDKN1C was another gene that had been shown overexpressed in DU145-DR and PC3-DR docetaxel-resistant cells. In the present microarray analysis, the inventors found that CDKN1C gene was also overexpressed with a 5.38 fold change in resistant cells. This result was confirmed by the RT-PCR experiment (Fig 4B). AHR is a ligand-activated transcription factor that mediates a pleiotropic response to environmental contaminants and that has recently been shown to be implicated in the development of cancers from different anatomical origins. Moreover, AHR had been identified as a putative Wnt/β-Catenin pathway target gene in prostate cancer cells. The AHR gene showed a 5.40 fold overexpression in resistant cells in the present microarray analysis. The inventors confirmed this result and showed a high dose-dependent overexpression in taxane-resistant cells by the RT-PCR approach (Fig 4B). The high increase in AHR gene expression was confirmed on an independent set of total RNA (not shown).

[0064] GALNT14 belongs to a large subfamily of glycosyltransferases residing in the Golgi apparatus. GALNT enzymes catalyze the first step in the O-glycosylation of mammalian proteins by transferring N-acetyl-D-galactosamine (GalNAc) to peptide substrates. The GALNT14 gene was one of the top down-regulated genes in the present signature with a fold-change expression of -10.26 in the resistant cells. The dose-dependent down-regulation of the expression of GALNT14 in resistant cells was confirmed by the RT-PCR analysis (Fig 5). The high decrease in GALNT14 gene expression was confirmed on an independent set of total RNA (not shown). The caspase 8 gene encodes a member of the cysteine-aspartic acid protease (caspase) family. Sequential activation of caspases plays a central role in the execution-phase of cell apoptosis. This gene was founded moderately under-expressed in the RT-PCR analysis (Fig 5).

[0065] LZTS1 encoding leucine zipper, putative tumor suppressor 1 was shown under-expressed in the present signature. The under-expression of this gene was confirmed by RT-PCR analysis (Fig 6A) and western blot analysis (Fig 6B) in docetaxel-resistant cells. The same results were obtained on an independent set of total RNAs (not shown). LZTS1 was of particular interest since it has been described as a tumor suppressor gene. The FEZ1/LZTS1 (LZTS1) protein was shown to be frequently downregulated in esophageal, breast, and prostate cancers. LZTS1 is expressed in normal tissues, and its introduction in cancer cells inhibits cell growth and suppresses tumorigenicity. Absence or low expression of LZTS 1 was correlated to high tumor grading on lung tumors suggesting that it may serve as a novel prognostic indicator.

[0066] The gene LOC152573 encodes the hypothetical protein BC012029 also named hSHISA3. The function of hSHISA3 is not known but by analogy with the mouse homologs, it is supposed to play an essential role in the maturation of presomitic mesoderm cells by individual attenuation of both FGF and WNT signalling. LOC152573/hSHISA3 corresponded to the most under-regulated gene in resistant cells with a fold change of -159.40 in the microarray analysis. The inventors confirmed the high decrease of its expression on independent set of total RNAs of resistant IGR-CaP1-R cells (Fig7A) as well as in extracts obtained from LNCaP-R and PC3-R docetaxel-resistant cells (Fig 7B).

[0067] Finally, the inventors checked for the confirmation of the genes implicated in the WNT pathway that were recovered in the microarray analysis. WNT family members function in a variety of developmental processes including regulation of cell growth and differentiation and are characterized by a WNT-core domain. Additionally, WNT signaling has emerged as an important pathway that underlies the initial notion of prostate cancer. Both human cancers and mouse models have confirmed that mutations or altered expression of components of this pathway are associated with prostate tumors. The WNT2B encoding a member of the WNT family of highly conserved, secreted signalling factors, was shown as one of the most over-expressed gene in the signature with a fold change of 9.42 in resistant cells. Two sets of probes were chosen to measure gene expression of WNT2B as this gene produces two alternative transcript variants (Fig 8A). The two probes showed a high level in gene expression in docetaxel-resistant cells in a dose dependent manner (Fig 8B). Others members of the WNT gene family, WNT5A and WNT7B genes, were also showed to be overexpressed in drug-resistant cells, although to a lesser extent (Fig 8C). The gene SFRP1 (Secreted frizzled-related protein 1) acting as soluble modulator of WNT signalling, FSTL1 encoding a protein with similarity to follistatin, and JAG1 encoding the ligand for the receptor Notch 1 were also shown to be up-regulated in the drug-resistant cells, although to different extent (Fig 8D). On the contrary, the gene FZD8, encoding a member of the frizzled gene family, showed a high decrease of its expression in drug-resistant cells (Fig 8E).

[0068] Overall, the results of real-time RT-PCR for these selected genes were in direct agreement with the microarray data. The same alternations of gene expression were observed by real-time RT-PCR analysis, although the fold change in the expression level was not exactly same between these two different analytical methods. Western Blot analyses were also in direct agreement with the microarray data. These results support the findings obtained from the present microarray analysis.

Table 1 : List of the over-expressed genes (at least two-fold) in the docetaxel resistant cell-lines.

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
RPIB9	RPIB9,RPIP9,FLJ30671,MG C26655	Rap2-binding protein 9	NM_138290
CXCL2	CXCL2,GRO2,GROb,MIP2, MIP2A, SCYB2,MGSA-b,MIP-2a,CINC-2a,MGSA beta	GR02 oncogene	NM_002089
AL137761	AL137761	Homo sapiens mRNA; cDNA DKFZp586L2424 (from clone DKFZp586L2424), [AL137761]	AL137761
TFP12	TFP12,PP5,TFP1-2,FLJ21164	tissue factor pathway inhibitor 2	NM_006528
THC2051204	THC2051204	Q300_MOUSE (Q02722) Protein Q300, partial (17%) [THC2051204]	
TNF	TNF,DIF,TNFA,TNFSF2,TNF -alpha	tumor necrosis factor (TNF superfamily, member 2)	NM_000594
ABCB1	ABCB1,CLCS,MDR1,P-gp, PGY1,ABC20,CD243,GP 170	ATP-binding cassette, sub-family B (MDR/TAP), member 1	NM_000927
PURG	PURG,PURG-A,PURG-B,MGC119274	purine-rich element binding protein G	M_013357
ADAMTS5	ADAMTS5,ADMP-2,ADAMTS11,FLJ36738	a disintegrin-like and metalloprotease (reprolysin type) with thrombospondin type 1 motif, 5 (aggrecanase-2)	NM_007038
MCTP1	MCTP1,FLJ22344	Homo sapiens cDNA FLJ34011 fis, clone FCBBF2001868, weakly similar to RABPHILIN-3A, [AK091330]	AK091330

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
SPTLC2L		Homo sapiens cDNA FLJ90790 fis, clone THYR01001529, weakly similar to SERINE PALMITOYLTRANSFERASE 2 (EC 2,3,1,50), [AK075271]	AK075271
OAS3	OAS3,p100,MGC133260	2'-5'-oligoadenylate synthetase 3 (100 kD)	NM_006187
MCTP1	MCTP1,FLJ22344	hypothetical protein FLJ22344	NM_024717
GAS1	GAS1	growth arrest-specific 1	NM_002048
BIRC3	BIRC3,AIP1,AP12,MIHC,CIA P2,HAIP1,HIAP1,MALT2,RN F49	baculoviral IAP repeat-containing 3	M_001165
BQ186674	BQ186674	UI-E-EJ1-ajr-f-10-0-UI,rl UI-E-EJ1 Homo sapiens cDNA clone UI-E-EJ1-ajr-f-10-0-UI 5', mRNA sequence [BQ186674]	BQ186674
MAL	MAL	mal, T-cell differentiation protein	NM_002371
UBXD3	UBXD3,FLJ25429	Homo sapiens UBX domain containing 3 (UBXD3), mRNA [NM_152376]	NM_152376
WNT2B	WNT2B,WNT13,XWNT2	wingless-type MMTV integration site family, member 2B	NM_024494
BM716045	BM716045	UI-E-EJO-aht-1-14-0-UI,r1 UL-E-EJO Homo sapiens cDNA clone UI-E-EJO-aht-1-14-0-UI 5', mRNA sequence [BM716045]	BM716045
SFRP1	SFRP1,FRP,FRP1,FrzA,FRP -1,SARP2	secreted frizzled-related protein 1	NM_003012
PLEKHH2	PLEKHH2,KIAA2028,PLEKH H1L	Homo sapiens pleckstrin homology domain containing, family H (with MyTH4 domain) member 2 (PLEKHH2), mRNA [NM_172069]	NM_172069
GNG11	GNG11,GNGT11	guanine nucleotide binding protein 11	NM_004126
CDH16	CDH16	cadherin 16, KSP-cadherin	NM_004062
AKR1C1	AKR1C1,C9,DD1,DDH,DDH 1,H-37,MBAB, HAKRC,MGC8954 ,2-ALPHA-HSD,20-ALPHA-HSD	aldo-keto reductase family 1, member C1 (dihydrodiol dehydrogenase 1; 20-alpha (3-alpha)-hydroxysteroid dehydrogenase)	NM_001353
MGC42367	MGC42367	similar to 2010300C02Rik protein	NM_207362

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
SFRP1	SFRP1,FRP,FRP1,FrzA,FRP -1,SARP2	secreted frizzled-related protein 1	NM_003012
AQP1	AQP1,CO,CHIP28,AQP-CHIP,MGC26324	Homo sapiens aquaporin 1 (channel-forming integral protein, 28kDa) (AQP1), transcript variant 1, mRNA [NM_198098]	NM_198098
RAFTLIN	RAFTLIN,MIG2,PIG9,PIB10, KIAA0084,MGC141678	raft-linking protein	NM_015150
FAM111A	FAM111A,FLJ22794,KIAA1895,DKFZp686A06175	hypothetical protein FLJ22794	NM_022074
FAM111A	FAM111A,FLJ22794,KIAA1895,DKFZp686A06175	hypothetical protein FLJ22794	NM_022074
ADAMTS1	ADAMTS1,C3-C5,METH1,KIAA1346	a disintegrin-like and metalloprotease (reprolysin type) with thrombospondin type 1 motif, 1	NM_006988
FHOD3	FHOD3,FHOS2,Formactin2	hypothetical protein FLJ22297	NM_025135
DUSP23	DUSP23,VHZ,LDP-3,DUSP25,FLJ20442,RP11-190A12,1	hypothetical protein FLJ20442	NM_017823
ITGB8	ITGB8	Homo sapiens, clone IMAGE: 4794726, mRNA, [BC042028]	BC042028
ADAMTS1	ADAMTS1,C3-CS,METH1,KIAA1346	a disintegrin-like and metalloprotease (reprolysin type) with thrombospondin type 1 motif, 1	NM_006988
THC2134488	THC2134488	Unknown	
IL15	IL15,IL-15,MGC9721	Homo sapiens interleukin 15 (IL15), transcript variant 1, mRNA [NM_172174]	NM_172174
PLEKHH2	PLEKHH2,KIAA2028,PLEKH H1L	Homo sapiens pleckstrin homology domain containing, family H (with MyTH4 domain) member 2 (PLEKHH2), mRNA [NM_172069]	NM_172069
AKR1C1	AKR1C1,C9,DD1,DDH,DDH 1,H-37,MBAB, HAKRC,MGC8954 ,2-ALPHA-HSD,20-ALPHA-HSD	aldo-keto reductase family 1, member C1 (dihydrodiol dehydrogenase 1; 20-alpha (3-alpha)-hydroxysteroid dehydrogenase)	NM_001353
IGFBP3	IGFBP3,IBP3,BP-53	insulin-like growth factor binding protein 3	NM_000598
CYP1A1	CYP1A1,AHH,AHRR,CP11, CYP1,P1-450,P450-C, P450DX	cytochrome P450, subfamily I (aromatic compound-inducible), polypeptide 1	NM_000499

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
PHLDA1	PHLDA1,PHRIP,TDAG51,DT 1P1B11,MGC131738	pleckstrin homology-like domain, family A, member 1	AF220656
TLR3	TLR3,CD283	toll-like receptor 3	NM_003265
GPC3	GPC3,SGB,DGSX,SDYS,SG BS,SGBS1	glypican 3	NM_004484
AHRR	AHRR,AHH,AHHR,KIAA123 4	Homo sapiens aryl-hydrocarbon receptor repressor (AHRR), mRNA [NM_020731]	NM_020731
CACNG6	CACNG6	Homo sapiens calcium channel, voltage-dependent, gamma subunit 6 (CACNG6), transcript variant 1, mRNA [NM_145814]	NM_145814
AQP1	AQP1,CO,CHIP28,AQP-CHIP,MGC26324	aquaporin 1 (channel-forming integral protein, 28kD)	NM_000385
AKR1C3	AKR1C3,DD3,HAKRB,HAKR e, HA1753,HSD17B5,hIuPGF S,KIAA0119	aldo-keto reductase family 1, member C3 (3-alpha hydroxysteroid dehydrogenase, type II)	NM_003739
FSTL1	FSTL1,FRP,FSL1, Follistatin-like	follistatin-like 1	NM_007085
AHR	AHR	aryl hydrocarbon receptor	NM_001621
C1orf88	C1orf88,FLJ23853,MGC126 550,RP5-1125M8,4	Homo sapiens hypothetical protein LOC128344 (LOC128344), mRNA [NM_ 181643]	NM_181643
CDKN1C	CDKN1C,BWS,WBS,p57,BW CR,KIP2	cyclin-dependent kinase inhibitor 1C (p57, Kip2)	NM_000076
A_32_P32463	A_32_P32463	Unknown	
PCDH9	PCDH9	protocadherin 9	NM_020403
NTN4	NTN4,PR03091,FLJ23180	netrin 4	M_021229
MID1	MID1,OS,FXY,OSX,OGS1,X PRF, BBBG1,GBBB1,RNF59, ZNFXY,TRIM18	midline 1 (Opitz/BBB syndrome)	NM_033290
CSPG2	CSPG2,VERSICAN,DKFZp6 86K06110	chondroitin sulfate proteoglycan 2 (versican)	NM_004385
AL133090	AL133090	Homo sapiens mRNA; cDNA DKFZp434E0528 (from clone DKFZp434E0528), [AL133090]	AL133090
DDC	DDC,AADC	dopa decarboxylase (aromatic L-amino acid decarboxylase)	NM_000790
AKR1C1	AKR1C1,C9,DD1,DDH,DDH 1,H-37,MBAB, HAKRC,MGC8954 ,2-ALPHA-HSD,20-ALPHA-HSD	aldo-keto reductase family 1, member C1 (dihydrodiol dehydrogenase 1; 20-alpha (3-alpha)-hydroxysteroid dehydrogenase)	NM_001353
PHLDA1	PHLDA1,PHRIP,TDAG51,DT 1P1B11,MGC131738	pleckstrin homology-like domain, family A, member 1	NM_007350

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
KCNH2	KCNH2,ERG1,HERG,LQT2, SQT1,HERG1,Kv11,1	potassium voltage-gated channel, subfamily H (eag-related), member2	NM_000238
ITGB8	ITGB8	integrin, beta 8	NM_002214
ST6GAL1	ST6GAL1,CD75,SIAT1,ST6 Gall, MGC48859,ST6Gal I	Homo sapiens sialyltransferase 1 (beta-galactoside alpha-2,6-sialyltransferase) (SIAT1), transcript variant 1, mRNA [NM_173216]	NM_173216
TMEFF1	TMEFF1,H7365,C9orf2	transmembrane protein with EGF-like and two follistatin-like domains 1	NM_003692
FBXL16	FBXL16,Fbl16,C16orf22,FLJ 33735,MGC33974,c380A1,1	Homo sapiens F-box and leucine-rich repeat protein 16 (FBXL16), mRNA [NM_153350]	NM_153350
ATP8A2	ATP8A2,IB,ATP,ML-1,ATPIB, DKFZP434B1913	ATPase, aminophospholipid transporter-like, Class I, type 8A, member 2	AL390129
CART1	CART1	cartilage paired-class homeoprotein 1	NM_006982
C9orf150	C9orf150,bA3L8,2,FLJ38505 , FLJ90271,HYST0841,MGC4 6502	Homo sapiens chromosome 9 open reading frame 150 (C9orf150), mRNA [M_203403]	NM_203403
PHLDA1	PHLDA1,PHRIP,TDAG51,DT 1P1B11,MGC131738	Homo sapiens cDNA clone IMAGE:5531727, partial cds, [BC037430]	BC037430
HDAC9	HDAC9,HD7,HDAC,HDRP,M ITR, HDAC7,HDAC7B,HDAC 9B,HDAC9FL, KIAA0744,DK FZp779K1053	histone deacetylase 7B	NM_014707
GLS	GLS,GLS1,FLJ10358,KIAA0 838,DKFZp686O15119	glutaminase	NM_014905
GATS	GATS,DKFZp686B07267	opposite strand transcription unit to STAG3	BC065200
CNTNAP3	CNTNAP3,CASPR3,CNTNA P3A, RP11-290L7,1,RP11-138L21,1	cell recognition molecule CASPR3	NM_033655
PDE4B	PDE4B,DPDE4,PDEIVB,MG C126529,DKFZp686F2182	phosphodiesterase 4B, cAMP-specific (dunce (Drosophila)-homolog phosphodiesterase E4)	L12686
DKFZp586I1420	DKFZp586I1420	Homo sapiens hypothetical protein DKFZp586I1420 (DKFZp586I1420) on chromosome 7 [NRL_002186]	NRL_002186
ZNRF2	ZNRF2,RNF202	Homo sapiens zinc and ring finger 2 (ZNRF2), mRNA [NM_147128]	NM_147128

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
FGF2	FGF2,BFGF,FGFB,HBGH-2	fibroblast growth factor 2 (basic)	NM_002006
SP5	SP5	Homo sapiens Sp5 transcription factor (SP5), mRNA [M_001003845]	NM_001003845
LAMA2	LAMA2,LAMM	laminin, alpha 2 (merosin, congenital muscular dystrophy)	NM_000426
THC2056328	THC2056328	Unknown	
KIAA1666	KIAA1666,DKFZp434H0735	KIAA1666 protein	AL117509
A_23_P103951	A_23_P103951	Unknown	
IL1R1	IL1R1,P80,IL1R,IL1RA,CD12 1A, D2S1473,IL-1R-alpha	interleukin 1 receptor, type I	NM_000877
CD55	CD55,CR,TC,DAF	decay accelerating factor for complement (CD55, Cromer blood group system)	NM_000574
MANEAL	MANEAL,FLJ31434,MGC78681,RP11-109P14,3	Homo sapiens hypothetical protein FLJ31434 (FLJ31434), mRNA [NM_152496]	NM_152496
AK026140	AK026140	Homo sapiens cDNA: FLJ22487 fis, clone HRC10931, [AK026140]	AK026140
FXYD2	FXYD2,HOMG2,ATP1G1,M GC12372	FXYD domain-containing ion transport regulator 2	NM_021603
CD40	CD40,p50,Bp50,CDW40,MG C9013,TNFRSF5	tumor necrosis factor receptor superfamily, member 5	NM_001250
KIAA1505	KIAA1505	KIAA1505 protein	NM_020879
DEPDC6	DEPDC6,DEP,6,FLJ12428,F LJ13854,DKFZp564B1778	hypothetical protein FLJ12428	NM_022783
GLS	GLS,GLS1,FLJ10358,KIAA0838,DKFZp686O15119	glutaminase	NM_014905
PPP2R2C	PPP2R2C,PR52,IMYPNO,IMYPNO1,MGC33570	Homo sapiens protein phosphatase 2 (formerly 2A), regulatory subunit B (PR 52), gamma isoform (PPP2R2C), transcript variant 1, mRNA [M_020416]	NM_020416
NRP1	NRP1,NRP,CD304,VEGF16 5R, DKFZp781F1414,DKFZp 686A03134	neuropilin 1	NM_003873
SP5	SP5	Homo sapiens Sp5 transcription factor (SP5), mRNA [M_001003845]	NM_001003845
ARHGDIB	ARHGDIB,D4,GDIA2,GDID4, LYGDI,Ly-GDI,RAP1GN1	Rho GDP dissociation inhibitor (GDI) beta	NM_001175
RAI2	RAI2	retinoic acid induced 2	NM_021785

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Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
LOC284262	LOC284262	hypothetical protein LOC284262	AL832945
TXNRD3	TXNRD3,TGR,TR2,TRXR3	thioredoxin reductase 2	AF171055
HIVEP1	HIVEP1,CIRIP,MBP-1,ZNF40,CRYBP1,PRDII-BF1	human immunodeficiency virus type I enhancer-binding protein 1	NM_002114
BC042017	BC042017	Homo sapiens, clone IMAGE: 5311842, mRNA, [BC042017]	BC042017
ABCB2	TAP1,APT1,PSF1,ABC17,A BCB2,RING4,TAP1N,D6S11 4E, FLJ26666,TAP1*0102N	transporter 1, ATP-binding cassette, sub-family B (MDR/TAP)	NM_000593
GLS	GLS,GLS1,FLJ10358,KIAA0 838,DKFZp686O15119	Homo sapiens mRNA; cDNA DKFZp686O15119 (from clone DKFZp686O15119), [CR749593]	CR749593
RASSF8	RASSF8,HoJ-1,C12orf2	Homo sapiens C120RF2 mRNA for C12ORF2, complete cds, [AB093206]	AB093206
CR622072	CR622072	full-length cDNA clone CSODF032YA11 of Fetal brain of Homo sapiens (human), [CR622072]	CR622072
LITAF	LITAF,PIG7,CMT1C,SIMPLE , TP53I7,FLJ38636,MGC1166 98,MGC116700,MGC116701 , MGC125274,MGC125275,M GC125276	LPS-induced TNF-alpha factor	NM_004862
IGF2	IGF2,INSIGF,pp9974,C11orf 43,FLJ22066,FLJ44734	Homo sapiens putative insulin-like growth factor II associated protein (LOC492304), mRNA [NM_001007139]	NM_001007 139
CYR61	CYR61,CCN1,GIG1,IGFBP1 0	cysteine-rich, angiogenic inducer, 61	NM_001554
PHF15	PHF15,JADE2,KIAA0239	KIAA0239 protein	M_015288
ProSAPiP1	ProSAPiP1,KIAA0552	ProSAPiP1 protein	NM_014731
THC2227602	THC2227602	002979 (002979) ORF2280 gene homolog (Fragment), partial (18%) [THC2227602]	
LOC389722	LOC389722,RP11-290L7,1,RP11-138L21,1	similar to cell recognition molecule CASPR3	AK054645
ANKRD18A	ANKRD18A,KIAA2015	Homo sapiens mRNA for KIAA2015 protein, [AB095935]	AB095935
FBN1	FBN1,FBN,SGS,WMS,MASS ,MFS1,OCTD	fibrillin 1 (Marfan syndrome)	NM_000138
RPS6KA2	RPS6KA2,RSK,HU-2,RSK3,p90-RSK3,pp90RSK3,MAPKAPK 1C,S6K-alpha, S6K-alpha2	ribosomal protein S6 kinase, 90kD, polypeptide 2	NM_021135

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
GRB10	GRB10,RSS,IRBP,MEG1,G RB-IR, KIAA0207	Homo sapiens growth factor receptor-bound protein 10 (GRB10), transcript variant 4, mRNA [NM_001001555]	NM_001001 555
AY336981	AY336981	Homo sapiens transcription factor GTF21RD2 isoform 3 (GTF2IRD2) mRNA, complete cds, alternatively spliced, [AY336981]	AY336981
RASSF8	RASSF8,HoJ-1,C12orf2	Homo sapiens C12ORF2 mRNA for C12ORF2, complete cds, [AB093206]	AB093206
SLPI	SLPI,ALP,MPI,ALK1,BLPI,H USI, WAP4,WFDC4,HUSI-I	secretory leukocyte protease inhibitor (antileukoproteinase)	NM_003064
SLPI	SLPI,ALP,MPI,ALK1,BLPI,H USI, WAP4,WFDC4,HUSI-I	secretory leukocyte protease inhibitor (antileukoproteinase)	NM_003064
THC2095463	THC2095463	Q8LQA6 (Q8LQA6) OJ1125_C04,6 protein, partial (21 %) [THC2095463]	
COL16A1	COL16A1,447AA,FP1572	collagen, type XVI, alpha 1	NM_001856
GRAMD3	GRAMD3,NS3TP2,FLJ21313	hypothetical protein FLJ21313	NM_023927
FXYD2	FXYD2,HOMG2,ATP1G1,M GC12372	FXYD domain-containing ion transport regulator 2	NM_021603
PDGFB	PDGFB,SIS,SSV,PDGF2,c-sis,FLJ12858	platelet-derived growth factor beta polypeptide (simian sarcoma viral (v-sis) oncogene homolog)	NM_002608
FAM107B	FAM107B,C10orf45,FLJ4550 5,MGC11034,MGC90261	hypothetical protein MGC11034	NM_031453
LOC440934	LOC440934	hypothetical gene supported by BC008048	BC008048
VCX	VCX,VCX1,VCXB1,VCX-B1,VCX10R,VCX-10r,MGC118975	variable charge, X chromosome	NM_013452
LAMB3	LAMB3,LAMNB1	laminin, beta 3 (nicein (125kD), kalinin (140kD), BM600 (125kD))	NM_000228
CYR61	CYR61,CCN1,GIG1,IGFBP1 0	cysteine-rich, angiogenic inducer, 61	NM_001554
NCALD	NCALD,MGC33870,MGC748 58	neurocalcin delta	NM_032041
WNT5A	WNT5A,hWNT5A	wingless-type MMTV integration site family, member 5A	NM_003392
ABCC3	ABCC3,MLP2,MRP3,ABC31, MOAT-D, cMOAT2,EST90757	ATP-binding cassette, sub-family C (CFTR/MRP), member 3	NM_003786

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
GLIS1	GLIS1,FLJ36155	Homo sapiens GLIS family zinc finger 1 (GLIS1), mRNA [NM_147193]	NM_147193
JAG1	JAG1,AGS,AHD,AWS,HJ1,CD339,JAGL1,MGC104644	jagged 1 (Alagille syndrome)	NM_000214
NRL	NRL,RP27,D14S46E	neural retina leucine zipper	NM_006177
AGT	AGT,ANHU,SERPINA8	angiotensinogen (serine (or cysteine) proteinase inhibitor, clade A (alpha-1 antiproteinase, antitrypsin), member 8)	NM_000029
TMSB4X	TMSB4X,FX,TB4X,PTMB4,T MSB4	Homo sapiens cDNA FLJ31414 fis, clone NT2NE2000260, weakly similar to THYMOSIN BETA-4,	AK055976
CCPG1	CCPG1,CPR8,KIAA1254	Homo sapiens cell cycle progression 1 (CCPG1), mRNA [NM_020739]	NM_020739
ADRA2C	ADRA2C,ADRA2L2,ADRARL2,ADRA2RL2,ALPHA2CAR	adrenergic, alpha-2C-, receptor	NM_000683
BM665539	BM665539	UI-E-CL1-afb-b-14-0-UI,s1 UI-E-CL1 Homo sapiens cDNA clone UI-E-CL1-afb-b-14-0-UI 3', mRNA sequence [BM665539]	BM665539
TEX15	TEX15,DKFZP434M2415	testis expressed sequence 15	NM_031271
SEMA3B	SEMA3B,SemA,SEMA5,SE MAA,semaV,LUCA-1,FLJ34863	sema domain, immunoglobulin domain (Ig), short basic domain, secreted, (semaphorin) 3B	NM_004636
NYD-SP18	NYD-SP18	testes development-related NYD-SP18	NM_032599
ASNS	ASNS,TS11	asparagine synthetase	NM_001673
NFKBIZ	NFKBIZ,IKBZ,INAP,MAIL,FLJ30225,FLJ34463	molecule possessing ankyrin repeats induced by lipopolysaccharide (MAIL), homolog of mouse	NM_031419
AK096677	AK096677	Homo sapiens cDNA FLJ39358 fis, clone PEBLM2004015, [AK096677]	AK096677
CA313037	CA313037	CA313037 UI-CF-FNO-aex-g-14-0-UI,s1 UI-CF-FNO Homo sapiens cDNA clone UI-CF-FNO-aex-g-14-0-UI 3', mRNA sequence [CA313037]	CA313037
PTPRM	PTPRM,RPTPM,RPTPU,PT PRL1,hR-PTPu,R-PTP-MU	protein tyrosine phosphatase, receptor type, M	NM_002845

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
SLC1A1	SLC1A1,EAAC1,EAAT3	solute carrier family 1 (neuronal/ epithelial high affinity glutamate transporter, system Xag), member 1	NM_004170
GRB10 0	GRB10,RSS,IRBP,MEG1,G RB-IR, KIAA0207	Homo sapiens growth factor receptor-bound protein 10 (GRB10), transcript variant 4, mRNA [NM_001001555]	NM_001001 555
NQO1	NQO1,DTD,QR1,DHQU,DIA 4,NMOR1,NMOR1	diaphorase (NADH/NADPH) (cytochrome b-5 reductase)	NM_000903
A_24_P40105 1	A_24_P401051	Unknown	
GPR161	GPR161,RE2,FLJ33952	Homo sapiens cDNA FLJ33952 fis, clone CTONG2018614, [AK091271]	AK091271
A_32_P32905	A_32_P32905	Unknown	
LOC389652	LOC389652	similar to asparagine synthetase; glutamine-dependent asparagine synthetase; TS11 cell cycle control protein	XM_372040
SRGAP3	SRGAP3,WRP,MEGAP,SRG AP2,ARHGAP14,KIAA0411	Homo sapiens SLIT-ROBO Rho GTPase activating protein 3 (SRGAP3), mRNA [NM_ 014850]	NM_014850
PDE4D	PDE4D,DPDE3,STRK1,HSP DE4D, PDE4DN2	phosphodiesterase 4D, cAMP-specific (dunce (Drosophila)-homolog phosphodiesterase E3)	NM_006203
THC2055165	THC2055165	Unknown	
LOC63920	LOC63920	transposon-derived Buster3 transposase-like	NM_022090
AK022020	AK022020	Homo sapiens cDNA FLJ11958 fis, clone HEMBB1000996, [AK022020]	AK022020
MGAT4A	MGAT4A,GNT-IV,GNT-IVA	mannosyl (alpha-1,3-)-glycoprotein beta-1,4-N-acetylglucosaminyltransferase, isoenzyme A	NM_012214
THC2201936	THC2201936	Q8WSI5 (Q8WSI5) Prophenol oxidase, partial (3%) [THC2201936]	
THC2091303	THC2091303	GRI1_HUMAN (Q9Y3R0) Glutamate receptor-interacting protein 1 (GRIP1 protein), partial (82%) [THC2091303]	

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Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
FCRL2	FCRL2	Homo sapiens hypothetical protein FLJ31052 (FLJ31052), mRNA [NM_152378]	NM_152378
PDE4DIP	PDE4DIP,MMGL,CMYA2,MGC75440,DKFZp781J054	Homo sapiens phosphodiesterase 4D interacting protein (myomegalin) (PDE4DIP), transcript variant 5, mRNA [NM_001002811]	NM_001002811
WBP5	WBP5,DKFZp313K1940	pp21 homolog	NM_016303
PIM1	PIM1,PIM	pim-1 oncogene	NM_002648
NUPR1	NUPR1,P8,COM1	nuclear protein 1	NM_012385
SMAD7	SMAD7,MADH7,MADH8,FLJ 16482	MAD (mothers against decapentaplegic, Drosophila) homolog 7	NM_005904
LOC63920	LOC63920	transposon-derived Buster3 transposase-like	NM_022090
STAT1	STAT1,ISGF-3,STAT91,DKFZp686B04100	signal transducer and activator of transcription 1, 91kD	NM_007315
AK057151	AK057151	Homo sapiens cDNA FLJ32589 fis, clone SPLEN2000443, [AK057151]	AK057151
PPM1H	PPM1H,ARHCL1,FLJ13253, KIAA1157	KIAA1157 protein	AB032983
BM806490	BM806490	AGENCOURT_6553853 NIH_MGC_71 Homo sapiens cDNA clone IMAGE:5555887 5', mRNA sequence [BM806490]	BM806490
AL390181	AL390181	Homo sapiens mRNA; cDNA DKFZp547J125 (from clone DKFZp547J125), [AL390181]	AL390181
LOC150356	LOC150356	hypothetical protein BC012882	BC012882
WNT7B	WNT7B	Homo sapiens wingless-type MMTV integration site family, member 7B (WNT7B), mRNA [NM_058238]	NM_058238
BF210146	BF210146	601874052F1 NIH_MGC_54 Homo sapiens cDNA clone IMAGE:4098852 5', mRNA sequence [BF210146]	BF210146
LRP11	LRP11,MANSC3,FLJ14735, MGC39092, bA350J20,3	hypothetical protein FLJ14735	NM_032832

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
FGFR2	FGFR2,BEK,JWS,CEK3,CFD1,ECT1,KGFR,TK14,TK25, BFR-1,K-SAM	fibroblast growth factor receptor 2 (bacteria-expressed kinase, keratinocyte growth factor receptor, craniofacial dysostosis 1, Crouzon syndrome, Pfeiffer syndrome, Jackson-Weiss syndrome)	NM_022972
MT2A	MT2A,MT2	metallothionein 2A	NM_005953
BF378046	BF378046	BF378046 RC1-TN0151-270900-013-b06 TN0151 Homo sapiens cDNA, mRNA sequence [BF378046]	BF378046
MT2A	MT2A,MT2	metallothionein 2A	BC007034
BC037328	BC037328	Homo sapiens cDNA clone IMAGE:5263455, partial cds, [BC037328]	BC037328
MT2A	MT2A,MT2	metallothionein 2A	NM_005953
ZNF323	ZNF323,ZNF310P,FLJ23407 ,ZNF20-Lp, dJ874C20,2	hypothetical protein FLJ23407	NM_030899
TBC1D8	TBC1D8,AD3,VRP,HBLP1	vascular Rab-GAP/TBC-containing	NM_007063
BLVRA	BLVRA,BLVR,BVRA	biliverdin reductase A	NM_000712
FGFR2	FGFR2,BEK,JWS,CEK3,CFD1,ECT1,KGFR,TK14,TK25, BFR-1,K-SAM	fibroblast growth factor receptor 2 (bacteria-expressed kinase, keratinocyte growth factor receptor, craniofacial dysostosis 1, Crouzon syndrome, Pfeiffer syndrome, Jackson-Weiss syndrome)	NM_022973

Table 2 : List of the under-expressed genes (at least two-fold) in the docetaxel resistant cell-lines.

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
PIK3C3	PIK3C3,Vps34,MGC61518	phosphoinositide-3-kinase, class 3	NM_002647
SCARB1	SCARB1,CLA1,SRB1,CLA-1,SR-BI,CD36L1,MGC138242	CD36 antigen (collagen type I receptor, thrombospondin receptor)-like 1	NM_005505
ASGR1	ASGR1,ASGPR,CLEC4H1,H s, 12056	asialoglycoprotein receptor 1	M_001671
FLJ22659	FLJ22659	hypothetical protein FLJ22659	AK026312
ASMTL	ASMTL,ASTML,ASMTLX,AS MTLY	acetylserotonin O-methyltransferase-like	NM_004192

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
ARHGAP10 0	ARHGAP10,GRAF2,PS-GAP, FLJ20896,FLJ41791	hypothetical protein FLJ20896	NM_024605
EDG6	EDG6,LPC1,S1P4,SLP4,S1P R4	endothelial differentiation, G-protein-coupled receptor 6	NM_003775
KCNC3	KCNC3,KV3,3,SCA13,KSHIII D	potassium voltage-gated channel, Shaw-related subfamily, member 3	NM_004977
MGC11332	MGC11332	hypothetical protein MGC11332	M_032718
NRG1	NRG1,GGF,HGL,HRG,NDF, ARIA,GGF2,HRG1,HRGA,S MDF	neuregulin 1	NM_004495
LOC339240	LOC339240	Homo sapiens keratin pseudogene (LOC339240) on chromosome 17 [NRL_ 001443]	NR_001443
PTPN3	PTPN3,PTPH1,DKFZp686N0 569	Homo sapiens mRNA; cDNA DKFZp686N0569 (from clone DKFZp686N0569), [CR749204]	CR749204
AK123483	AK123483	Homo sapiens cDNA FLJ41489 fis, clone BRTHA2004582, [AK123483]	AK123483
NRG1	NRG1,GGF,HGL,HRG,NDF, ARIA,GGF2,HRG1,HRGA,S MDF	neuregulin 1	NM_013962
FAM80A	FAM80A, MGC47816,RP11-157D18,1	Homo sapiens hypothetical protein MGC47816 (MGC47816), mRNA [NM_ 173642]	NM_173642
BAMBI	BAMBI,NMA	putative transmembrane protein	NM_012342
PTPN3	PTPN3,PTPH1,DKFZp686N0 569	protein tyrosine phosphatase, non-receptor type 3	NM_002829
SAMD8	SAMD8,FLJ25082	Homo sapiens sterile alpha motif domain containing 8 (SAMD8), mRNA [NM_ 144660]	NM_144660
KCNMB4	KCNMB4	potassium large conductance calcium-activated channel, subfamily M, beta member 4	NM_014505
SPG20	SPG20,SPARTIN,TAHCCP1, KIAA0610	Homo sapiens spastic paraplegia 20, spartin (Troyer syndrome) (SPG20), mRNA [NM_015087]	NM_015087
RGS16	RGS16,RGS-R,A28-RGS14,A28-RGS14P	regulator of G-protein signalling 16	NM_002928

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
UGT2B7	UGT2B7,UGT2B9	UDP glycosyltransferase 2 family, polypeptide B7	NM_001074
LMBRD2	LMBRD2,MGC125692,DKFZ p434H2226,DKFZp686G105 7	Homo sapiens hypothetical protein DKFZp434H2226 (DKFZp434H2226), mRNA [NM_001007527]	NM_001007 527
TMPRSS4	TMPRSS4,MT-SP2,TMPRSS3	transmembrane protease, serine 4	NM_019894
PDK1	PDK1	pyruvate dehydrogenase kinase, isoenzyme 1	NM_002610
RAB39B	RAB39B	Homo sapiens RAB39B, member RAS oncogene family (RAB39B), mRNA [NM_171998]	NM_171998
HSPH1	HSPH1,HSP105A,HSP105B, KIAA0201,NY-CO-25, DKFZp686M05240	heat shock 105kD	NM_006644
PSCDBP	PSCDBP,HE,B3-1,CASP, CYBR,CYTIP	pleckstrin homology, Sec7 and coiled/coil domains, binding protein	NM_004288
UGT2B11	UGT2B11,MGC129611,MGC 129612	UDP glycosyltransferase 2 family, polypeptide B11	NM_001073
ZNF516	ZNF516,HsT287	KIAA0222 gene product	D86975
CKB	CKB,B-CK,CKBB	creatine kinase, brain	NM_001823
SLC7A8	SLC7A8,LAT2,LPI-PC1	Homo sapiens solute carrier family 7 (cationic amino acid transporter, y+ system), member 8 (SLC7A8), transcript variant 2, mRNA [NM_182728]	NM_182728
UGT2B28	UGT2B28	Homo sapiens UDP glycosyltransferase 2 family, polypeptide B28 (UGT2B28), mRNA,	NM_053039
SEMA3C	SEMA3C,SemE,SEMAE	sema domain, immunoglobulin domain (Ig), short basic domain, secreted, (semaphorin) 3C	NM_006379
PDK1	PDK1	pyruvate dehydrogenase kinase, isoenzyme 1	NM_002610
GATA2	GATA2,NFE1B,MGC2306	hypothetical protein MGC2306	NM_032638
THC2064535	THC2064535	Unknown	
PDLIM5	PDLIM5,L9,ENH,LIM	LIM protein (similar to rat protein kinase C-binding enigma)	NM_006457

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Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
BX538293	BX538293	Homo sapiens mRNA; cDNA DKFZp686F09157 (from clone DKFZp686F09157), [BX538293]	BX538293
HYAL1	HYAL1,NAT6,LUCA1,HYAL-1,MGC45987	Homo sapiens hyaluronoglucosaminidase 1 (HYAL1), transcript variant 4, mRNA [NM_153284]	NM_153284
UGT2B4	UGT2B4,UGT2B11	UDP glycosyltransferase 2 family, polypeptide B4	NM_021139
THC2185385	THC2185385	Unknown	
FLJ10159	FLJ10159	hypothetical protein FLJ10159	AK001021
UGT2B10	UGT2B10,MGC142209	UDP glycosyltransferase 2 family, polypeptide B10	NM_001075
PRTFDC1	PRTFDC1,HHGP,FLJ11888	HHGP protein	NM_020200
A_24_P57526 7	A_24_P575267	Unknown	
PTGES	PTGES,PGES,PIG12,PP102, PP1294,MGST-IV,MGST1 L1,TP53112,MGC1 0317,MGST1-L1	prostaglandin E synthase	NM_004878
DNAJC15	DNAJC15,MCJ, HSD18,DNAJD1	DNAJ domain-containing	NM_013238
NPAS1	NPAS1,MOP5,PASD5	neuronal PAS domain protein 1	NM_002517
A_24_P47894 0	A_24_P478940	Unknown	
FRMD4B	FRMD4B,GRSP1,KIAA1013, 6030440G05Rik	Homo sapiens FERM domain containing 4B, mRNA (cDNA clone IMAGE:4508579), partial cds, [BC028291]	BC028291
SLC7A11	SLC7A11,xCT,CCBR1	Homo sapiens solute carrier family 7, (cationic amino acid transporter, y+ system) member 11, mRNA (cDNA clone IMAGE:5300264), partial cds, [BC041925]	BC041925
AF132203	AF132203	Homo sapiens PRO1933 mRNA, complete cds, [AF132203]	AF132203
FLJ43855	FLJ43855	similar to sodium- and chloride-dependent creatine transporter	NM_198857
TFDP2	TFDP2,DP2,Dp-2	transcription factor Dp-2 (E2F dimerization partner 2)	NM_006286
PFKFB4	PFKFB4	6-phosphofructo-2-kinase/ fructose-2,6-biphosphatase 4	NM_004567

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Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
TAC3	TAC3,NKB,NKNB,PRO1155, ZNEUROK1	tachykinin 3 (neuromedin K, neurokinin beta)	NM_013251
ENC1	ENC1,NRPB,CCL28,ENC-1,PIG10,TP53I10,FLJ39259	ectodermal-neural cortex (with BTB-like domain)	NM_003633
EPAS1	EPAS1,HLF,MOP2,HIF2A,PASD2	endothelial PAS domain protein 1	NM_001430
NRP2	NRP2,NP2,NPN2,PRO2714, MGC126574,VEGF165R2	Homo sapiens neuropilin 2 (NRP2), transcript variant 6, mRNA [M_201264]	NM_201264
MFHAS1	MFHAS1,MASL1,FLJ23354	MFH-amplified sequences with leucine-rich tandem repeats 1	NM_004225
AK024680	AK024680	Homo sapiens cDNA: FLJ21027 fis, clone CAE07110, [AK024680]	AK024680
SYTL3	SYTL3,SLP3,MGC105130,MGC118883,MGC118884,MGC118885	Homo sapiens synaptotagmin-like 3 (SYTL3), mRNA [NM_001009991]	NM_001009 991
BDNF	BDNF,MGC34632	Homo sapiens brain-derived neurotrophic factor (BDNF), transcript variant 6, mRNA [NM_170734]	NM_170734
AL137342	AL137342	Homo sapiens mRNA; cDNA DKFZp761G1111 (from clone DKFZp761G 1111 [AL137342]	AL137342
D4S234E	D4S234E,P21,NSG1,D4S234 , NEEP21	DNA segment on chromosome 4 (unique) 234 expressed sequence	NM_014392
LCP1	LCP1,CP64,PLS2,LC64P,FLJ25423,FLJ26114,FLJ39956, L-PLASTIN,DKFZp781A23186	lymphocyte cytosolic protein 1 (L-plastin)	NM_002298
A_32_P95067	A_32_P95067	Unknown	
THC2038567	THC2038567	Q7UFH6 (Q7UFH6) Acetyl-CoA carboxylase (Biotin carboxyl carrier subunit) accB, partial (16%) [THC2038567]	
LMO1	LMO1,TTG1,RBTN1,RHOM1, MGC116692	LIM domain only 1 (rhombotin 1)	NM_002315
BSPRY	BSPRY,FLJ20150	hypothetical protein FLJ20150	M_017688
BDNF	BDNF,MGC34632	Homo sapiens brain-derived neurotrophic factor (BDNF), transcript variant 1, mRNA [NM_170735]	NM_170735

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Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
UGT8	UGT8,CGT	UDP glycosyltransferase 8 (UDP-galactose ceramide galactosyltransferase)	NM_003360
LIPG	LIPG,EL,EDL,PR0719	lipase, endothelial	NM_006033
AW205591	AW205591	UI-H-BI1-afr-b-02-0-UI,s1 NCI_CGAP_Sub3 Homo sapiens cDNA clone IMAGE: 2722515 3', mRNA sequence [AW205591]	AW205591
AKAP12	AKAP12,AKAP250,DKFZp68 6M0430, DKFZp686O0331	A kinase (PRKA) anchor protein (gravin) 12	NM_005100
B3GALT1	B3GALT1,MGC126594,beta3 Gal-T1	UDP-Gal:betaGlcNAc beta 1,3-galactosyltransferase, polypeptide 1	NM_020981
CHST7	CHST7,C6ST-2	carbohydrate (N-acetylglucosamine 6-O) sulfotransferase 7	NM_019886
NMNAT2	NMNAT2,PNAT2,PNAT-2,C1orf15,MGC2756,KIAA04 79	Homo sapiens nicotinamide nucleotide adenylyltransferase 2 (NMNAT2), transcript variant 1, mRNA [NM_015039]	NM_015039
SLC12A3	SLC12A3,TSC,NCCT	solute carrier family 12 (sodium/chloride transporters), member 3	NM_000339
SLC22A2	SLC22A2,OCT2,MGC32628	solute carrier family 22 (organic cation transporter), member 2	NM_003058
LMBRD2	LMBRD2,MGC125692,DKFZ p434H2226,DKFZp686G105 7	Homo sapiens hypothetical protein DKFZp434H2226 (DKFZp434H2226), mRNA [NM_001007527]	NM_001007 527
SCD	SCD,SCD1,FADS5,PRO0998	stearoyl-CoA desaturase (delta-9-desaturase)	NM_005063
AMPH	AMPH,AMPH1	amphiphysin (Stiff-Mann syndrome with breast cancer 128kD autoantigen)	NM_001635
ANKRD37	ANKRD37,Lrp2bp,MGC1115 07	Homo sapiens low density lipoprotein receptor-related protein binding protein (Lrp2bp), mRNA[NM_ 181726]	NM_181726
LIN7A	LIN7A,LIN7,VELI1,LIN-7A, MALS-1,TIP-33,MGC148143	Vertebrate LIN7 homolog 1, Tax interaction protein 33	NM_004664

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
PHEX	PHEX,HYP,PEX,XLH,HPDR, HYP1,HPDR1	phosphate regulating gene with homologies to endopeptidases on the X chromosome (hypophosphatemia, vitamin D resistant rickets)	NM_000444
C1QL1	C1QL1,CRF,C1QRF	C1q-related factor	NM_006688
EPAS1	EPAS1,HLF,MOP2,HIF2A,PA SD2	endothelial PAS domain protein 1	NM_001430
KCNC4	KCNC4,KV3,4,KSHIIIC,HKS HIIIC,MGC126818	Homo sapiens potassium voltage-gated channel, Shaw-related subfamily, member 4 (KCNC4), transcript variant 2, mRNA [NM_153763]	NM_153763
FOXL2	FOXL2,BPES,PFRK,POF3,B PES1,PINTO	forkhead box L2	NM_023067
SCD	SCD,SCD1,FADS5,PRO0998	stearoyl-CoA desaturase (delta-9-desaturase)	NM_005063
FGFBP1	FGFBP1,FGFBP,HBP17 7	heparin-binding growth factor binding protein	M_005130
CASP8	CASP8,CAP4,MACH,MCH5, FLICE,MGC78473	caspase 8, apoptosis-related cysteine protease	NM_033356
LZTS1	LZTS1,F37,FEZ1	leucine zipper, putative tumor suppressor1	NM_021020
SYTL3	SYTL3,SLP3,MGC105130,M GC118883,MGC118884,MG C118885	Homo sapiens synaptotagmin-like 3 (SYTL3), mRNA [NM_ 001009991]	NM_001009 991
HSHPX5	HSHPX5	PREDICTED: Homo sapiens HPX-5 (HSHPX5), mRNA [XM_496232]	XM_496232
NLGN1	NLGN1,KIAA1070,MGC4511 5	neuroligin 1	NM_014932
MGST1	MGST1,MGST,GST12,MGST -I,MGC14525	Homo sapiens microsomal glutathione S-transferase 1 (MGST1), transcript variant 1c, mRNA [NM_145791]	NM_145791
PLXNA2	PLXNA2,OCT,PLXN2,FLJ11 751,FLJ30634,KIAA0463	plexin A2	NM_025179
ST8SIA4	ST8SIA4,PST,PST1,SIAT8D, MGC34450,MGC61459,ST8 SIA-IV	sialyltransferase 8 (alpha-2, 8-polysialytransferase) D	NM_005668
THC2050576	THC2050576	Unknown	

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Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
SLC3A1	SLC3A1,D2H,ATR1,NBAT,RBAT,CSNU1,FLJ34681	solute carrier family 3 (cystine, dibasic and neutral amino acid transporters, activator of cystine, dibasic and neutral amino acid transport), member 1	NM_000341
TNRC9	TNRC9,CAGF9	trinucleotide repeat containing 9	U80736
AK022997	AK022997	Homo sapiens cDNA FLJ12935 fis, clone NT2RP2004982, [AK022997]	AK022997
UGT8	UGT8,CGT	UDP glycosyltransferase 8 (UDP-galactose ceramide galactosyltransferase)	U62899
LETM2	LETM2,FLJ25409	Homo sapiens leucine zipper-EF-hand containing transmembrane protein 2 (LETM2), mRNA [NM_144652]	NM_144652
PLAC8	PLAC8,C15,onzin	hypothetical protein	NM_016619
DIAPH2	DIAPH2,DIA,POF,DIA2,POF2,FLJ11167	diaphanous (Drosophila, homolog) 2	NM_007309
BC014452	BC014452	Homo sapiens cDNA clone IMAGE:4903661, complete cds, [BC014452]	BC014452
SUNC1	SUNC1,MGC33329	Homo sapiens Sad1 and UNC84 domain containing 1 (SUNC1),mRNA [NM_152782]	NM_152782
DUSP13	DUSP13,BEDP,MDSP,TMDP , SKRP4,FLJ32450	protein phosphatase	NM_016364
AUTS2	AUTS2,KIAA0442,MGC1314 0	Homo sapiens autism susceptibility candidate 2 (AUTS2), mRNA [M_015570]	NM_015570
PLAC8	PLAC8,C15,onzin	hypothetical protein	NM_016619
THC2210862	THC2210862	Unknown	
MSX2	MSX2,FPP,MSH,PFM,CRS2,HOX8,PFM1	msh (Drosophila) homeo box homolog 2	NM_002449
SMAD9	SMAD9,MADH6,MADH9,SMAD8A,SMAD8B	MAD (mothers against decapentaplegic, Drosophila) homolog 9	NM_005905
TTN	TTN,TMD,CMH9,CMD1G,CMPD4,HMERF,LGMD2J,FLJ26020,FLJ26409,FLJ32040,FLJ34413,FLJ39564,FLJ43066,DKFZp451N061	Homo sapiens titin (TTN), transcript variant N2-A, mRNA [NM_133378]	NM_133378

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
LRRN6C	LRRN6C,LERN3,LINGO2,FLJ31810 0	Homo sapiens hypothetical protein FLJ31810 (FLJ31810), mRNA [NM_152570]	NM_152570
MEIS2	MEIS2,MRG1,MGC2820,HsT18361	Homo sapiens Meis1, myeloid ecotropic viral integration site 1 homolog 2 (mouse) (MEIS2), transcript variant a, mRNA [NM_170677]	NM_170677
DHRS3	DHRS3,SDR1,RDH17,Rsdr1,retSDR1	short-chain dehydrogenase/reductase 1	M_004753
OLR1	OLR1,LOX1,CLEC8A,SCAR E1	oxidised low density lipoprotein (lectin-like) receptor 1	NM_002543
NSBP1	NSBP1	nucleosomal binding protein 1	NM_030763
MOXD1	MOXD1,MOX,PRO5780,dJ248E1,1,DKFZP564G202	Homo sapiens monooxygenase, DBH-like 1 (MOXD1), mRNA [NM_015529]	NM_015529
DCAMKL1	DCAMKL1,DCLK,KIAA0369	doublecortin and CaM kinase-like 1	NM_004734
C12orf59	C12orf59,FLJ31166,MGC111385	hypothetical protein FLJ31166	NM_153022
A_23_P13685 7	A_23_P136857	Unknown	
BF675806	BF675806	602083723F1 NIH_MGC_83 Homo sapiens cDNA clone IMAGE:4248004 5', mRNA sequence [BF675806]	BF675806
SLC44A5	SLC44A5,CTL5,FLJ34081,MGC34032	Homo sapiens hypothetical protein MGC34032 (MGC34032), mRNA [NM_152697]	NM_152697
SALL1	SALL1,TBS,HSAL1,ZNF794	sal (Drosophila)-like 1	NM_002968
GPR177	GPR177,MRP,WLS,C1orf139 , FLJ23091,MGC14878,MGC131760	hypothetical protein FLJ23091	NM_024911
AK3L1	AK3L1,AK3,AK4	Homo sapiens cDNA: FLJ23313 fis, clone HEP11919	AK026966
AK3L1	AK3L1,AK3,AK4	adenylate kinase 3	NM_013410
FZD8	FZD8,FZ-8,hFZ8	frizzled (Drosophila) homolog 8	NM_031866
THC2088463	THC2088463	ALU7_HUMAN (P39194) Alu subfamily SQ sequence contamination warning entry, partial (8%) [THC2088463]	

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
FLJ39502	FLJ39502,FLJ26337,MGC13 4803	hypothetical protein FLJ39502	AK096821
PROS1	PROS1,PSA,PROS,PS21,PS 22,PS23,PS24,PS25,PS 26,Protein S,protein Sa	protein S (alpha)	NM_000313
PTPRD	PTPRD,HPTP,PTPD,HPTPD, MGC119750,MGC119751,M GC119752,MGC119753,HPT P-DELTA,R-PTP-DELTA	protein tyrosine phosphatase, receptor type, D	NM_002839
PDE1A	PDE1A,HCAM1,HSPDE1A,M GC26303	phosphodiesterase 1A, calmodulin-dependent	NM_005019
MYB	MYB,efg,c-myb,c-myb_CDS	v-myb avian myeloblastosis viral oncogene homolog	NM_005375
SLC16A10	SLC16A10,TAT1,PR00813	hypothetical protein PRO0813	NM_018593
GJA7	GJA7,CX45,DKFZp686P073 8	gap junction protein, alpha 7, 45kD (connexin 45)	NM_005497
GAL	GAL,GALN,GLNN,MGC4016 7	galanin-related peptide	NM_015973
CPM	CPM	carboxypeptidase M	NM_001874
PDE1A	PDE1A,HCAM1,HSPDE1A,M GC26303	phosphodiesterase 1A, calmodulin-dependent	NM_005019
PLXNA2	PLXNA2,OCT,PLXN2,FLJ11 751,FLJ30634,KIAA0463	Homo sapiens cDNA FLJ30634 fis, clone CTONG2002453,	AK055196
PDE1A	PDE1A,HCAM1,HSPDE1A,M GC26303	Homo sapiens phosphodiesterase 1A, calmodulin-dependent (PDE1A), transcript variant 2, mRNA [NM_001003683]	NM_001003 683
AW467174	AW467174	AW467174 ha35g06,x1 NCI_ CGAP_Kid12 Homo sapiens cDNA clone IMAGE:2875738 3' similar to gb:X60673_rna1 GTP:AMP PHOSPHOTRANSFERASE MITOCHONDRIAL (HUMAN);, mRNA sequence [AW467174]	AW467174
PLAT	PLAT,TPA,T-PA, DKFZp686103148	plasminogen activator, tissue	NM_000930
LOC441047	LOC441047	similar to Adenylate kinase isoenzyme 4, mitochondrial (ATP-AMP transphosphorylase)	XM_496720
CXCR4	CXCR4,FB22,HM89,LAP3,L CR1,NPYR,WHIM,CD184,LE STR,NPY3R,NPYRL,HSY3R R, NPYY3R,D2S201 E	chemokine (C-X-C motif), receptor 4 (fusin)	NM_003467

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
AK3L1	AK3L1,AK3,AK4	adenylate kinase 3	NM_013410
SMPDL3A	SMPDL3A,ASM3A,ASML3a, FLJ20177,yR36GH4,1	Homo sapiens sphingomyelin phosphodiesterase, acid-like 3A (SMPDL3A), mRNA [M_ 006714]	M_006714
KIAA0960	KIAA0960	KIAA0960 protein	AB023177
LHFP	LHFP,MGC22429	lipoma HMGIC fusion partner	NM_005780
CPM	CPM	carboxypeptidase M	NM_001874
A_24_P34529	A_24_P345290	Unknown	
RXFP1	RXFP1,LGR7,LGR7,1,LGR7, 2,LGR7,10,MGC138347,MG C142177	leucine-rich repeat-containing G protein-coupled receptor 7	M_021634
PNOC	PNOC,PPNOC	prepronociceptin	NM_006228
GALNT14	GALNT14,GALNT15,FLJ126 91,FLJ13977,GalNac-T10,GalNac-T14	hypothetical protein FLJ12691	NM_024572
TM4SF1	TM4SF1,L6,H-L6,M3S1,TAAL6	Homo sapiens transmembrane 4 superfamily member 1 (TM4SF1), mRNA [NM_014220]	M_014220
ZAR1	ZAR1	Homo sapiens zygote arrest 1 (ZAR1), mRNA[NM_175619]	NM_175619
A_23_P10091	A_23_P10091	Unknown	
GLT8D2	GLT8D2,FLJ31494	gycosyltransferase	M_031302
RXFP1	RXFP1,LGR7,LGR7,1,LGR7, 2,LGR7,10,MGC138347,MG C142177	Relaxin receptor 1 (Leucine-rich repeat-containing G protein-coupled receptor 7), [Source:Uniprot/ SWISSPROT;Acc:Q9H BX9] [ENST00000343542]	BX647985
CGNL1	CGNL1,JACOP,FLJ14957,KI AA1749,MGC138254	hypothetical protein FLJ14957	NM_032866
AK094972	AK094972	Homo sapiens cDNA FLJ37653 fis, clone BRHIP2010217, [AK094972]	AK094972
LRCH2	LRCH2,KIAA1495,dA204F4,4	Homo sapiens leucine-rich repeats and calponin homology (CH) domain containing 2 (LRCH2), mRNA [NM_020871]	NM_020871
BM930757	BM930757	UI-E-EJ1-ajm-I-20-0-UI,r1 UI-E-EJ1 Homo sapiens cDNA clone UI-E-EJ1-ajm-I-20-0-UI 5', mRNA sequence [BM930757]	BM930757

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
ATP8A1	ATP8A1,ATPIA,ATPP2,ATPASEII,MGC26327,MGC130042 , MGC130043	Homo sapiens ATPase, aminophospholipid transporter (APLT), Class I, type 8A, member 1 (ATP8A1), mRNA [NM_006095]	NM_006095
SOX9	SOX9,CMD1,SRA1,CMPD1	SRY (sex determining region Y)-box 9 (campomelic dysplasia, autosomal sex-reversal)	NM_000346
SLC39A8	SLC39A8,BIGM103,LZT-Hs6	up-regulated by BCG-CWS	NM_022154
TMEM47	TMEM47,BCMP1,TM4SF10, MGC32949,DKFZp564E153, DKFZP761J17121	brain cell membrane protein 1	M_031442
SLC10A4	SLC10A4,P4,MGC29802	Homo sapiens solute carrier family 10 (sodium/bile acid cotransporterfamily), member 4 (SLC10A4), mRNA [NM_152679]	NM_152679
SLC1A3	SLC1A3,EA6,EAAT1,GLAST, GLAST1,FLJ25094	solute carrier family 1 (glial high affinity glutamate transporter), member 3	M_004172
EDG7	EDG7	Homo sapiens cDNA FLJ34412 fis, clone HEART2002432, [AK091731]	AK091731
ITGA2	ITGA2,BR,GPIa,CD49B,VLA-2,VLAA2	integrin, alpha 2 (CD49B, alpha 2 subunit of VLA-2 receptor)	NM_002203
SLC1A3	SLC1A3,EA6,EAAT1,GLAST, GLAST1,FLJ25094	solute carrier family 1 (glial high affinity glutamate transporter), member 3	M_004172
PLCXD3	PLCXD3	Homo sapiens phosphatidylinositol-specific phospholipase C, X domain containing 3 (PLCXD3), mRNA [M_001005473]	NM_001005 473
BF514799	BF514799	UI-H-BW1-anj-a-01-0-UI,s1 NCI_CGAP_Sub7 Homo sapiens cDNA clone IMAGE: 3082272 3', mRNA sequence [BF514799]	BF514799
SLC16A12	SLC16A12,MCT12	Homo sapiens cDNA FLJ42911 fis, clone BRHIP3024118, weakly similar to Monocarboxylate transporter 4, [AK124901]	AK124901
THC2208430	THC2208430	Unknown	

(continued)

Primary Sequence Name	Sequence Name(s)	Sequence Description	Accession #
THC2182743	THC2182743	EDG7_HUMAN (Q9UBY5) Lysophosphatidic acid receptor Edg-7 (LPA receptor 3) (LPA-3), partial (36%) [THC2182743]	
C4orf18	C4orf18,AD021,AD036,FLJ38155,DKFZp434L142	AD021 protein	NM_016613
ANKRD38	ANKRD38,FLJ10884,KIAA0172,dJ1078M7,1,RP5-1155K23,5	Homo sapiens hypothetical protein LOC163782 (LOC163782), mRNA [NM_181712]	NM_181712
CALCRL	CALCRL,CRLR,CGRPR	calcitonin receptor-like	NM_005795
hSHISA3	LOC152573	hypothetical protein BC012029	BC012029

Table 3 : List of the over-expressed genes by at least two fold in the docetaxel resistant cell-lines.

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	**50nM**	**25nM**	**12nM**	**5nM**	**Mean**	**Mean**	**Mean**
RPIB9	1,49	1,51	1,55	1,59	1,53	34,22	34,22
CXCL2	1,45	1,28	1,96	1,28	1,49	30,98	30,98
AL137761	1,18	1,13	1,53	1,54	1,35	22,17	22,17
TFPI2	1,61	1,65	1,48	0,61	1,34	21,84	21,84
THC2051204	1,32	1,21	1,34	1,34	1,30	20,11	20,11
TNF	1,22	1,28	1,52	1,04	1,26	18,36	18,36
ABCB1	1,21	1,19	1,20	1,20	1,20	15,83	15,83
PURG	1,34	1,23	1,11	1,07	1,19	15,39	15,39
ADAMTS5	1,22	1,34	1,03	0,96	1,14	13,75	13,75
MCTP1	1,01	1,14	1,28	1,00	1,11	12,79	12,79
SPTLC2L	0,88	1,22	1,36	0,92	1,10	12,46	12,46
OAS3	1,15	1,13	1,09	0,97	1,08	12,16	12,16
MCTP1	1,00	1,12	1,15	0,97	1,06	11,45	11,45
GAS1	1,22	1,30	0,98	0,59	1,02	10,47	10,47
BIRC3	0,96	1,04	1,28	0,79	1,02	10,40	10,40
BQ186674	0,76	0,95	1,30	1,03	1,01	10,28	10,28
MAL	0,88	1,00	1,10	0,98	0,99	9,79	9,79
UBXD3	1,23	1,12	0,88	0,74	0,99	9,78	9,78
WNT2B	0,95	0,96	0,97	1,01	0,97	9,42	9,42
BM716045	1,06	1,02	0,98	0,82	0,97	9,36	9,36
SFRP1	1,04	1,01	0,91	0,91	0,97	9,30	9,30
PLEKHH2	1,12	0,92	0,86	0,84	0,93	8,61	8,61

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
GNG11	1,10	1,15	0,81	0,53	0,90	7,88	7,88
CDH16	1,27	1,15	0,71	0,44	0,89	7,84	7,84
AKR1C1	1,00	1,10	0,82	0,66	0,89	7,84	7,84
MGC42367	1,23	1,12	0,77	0,46	0,89	7,83	7,83
SFRP1	0,93	0,92	0,86	0,86	0,89	7,82	7,82
AQP1	0,90	0,87	0,90	0,90	0,89	7,82	7,82
RAFTLIN	0,83	0,94	1,01	0,76	0,89	7,69	7,69
FAM111A	0,90	0,82	0,86	0,93	0,88	7,53	7,53
FAM111A	0,86	0,88	0,92	0,83	0,87	7,46	7,46
ADAMTS1	1,06	0,93	0,83	0,65	0,87	7,39	7,39
FHOD3	0,93	0,91	0,82	0,77	0,86	7,20	7,20
DUSP23	0,93	0,89	0,75	0,81	0,85	7,00	7,00
ITGB8	0,87	0,88	0,89	0,74	0,85	7,00	7,00
ADAMTS1	1,02	0,92	0,80	0,64	0,84	6,99	6,99
THC2134488	0,97	0,89	0,78	0,73	0,84	6,92	6,92
IL15	0,87	0,92	0,89	0,64	0,83	6,75	6,75
PLEKHH2	1,01	0,83	0,68	0,78	0,83	6,69	6,69
AKR1C1	0,90	1,01	0,76	0,62	0,82	6,61	6,61
IGFBP3	0,66	0,99	0,90	0,69	0,81	6,45	6,45
CYP1A1	0,83	0,78	0,72	0,82	0,79	6,14	6,14
PHLDA1	0,75	0,72	0,76	0,83	0,77	5,86	5,86
TLR3	0,68	0,75	1,07	0,55	0,76	5,79	5,79
GPC3	0,86	0,81	0,64	0,72	0,76	5,74	5,74
AHRR	0,83	0,79	0,67	0,74	0,76	5,73	5,73
CACNG6	0,96	0,96	0,58	0,54	0,76	5,72	5,72
AQP1	0,76	0,72	0,75	0,75	0,75	5,58	5,58
AKR1C3	0,79	0,93	0,66	0,59	0,74	5,53	5,53
FSTL1	0,87	0,74	0,81	0,53	0,74	5,48	5,48
AHR	0,82	0,81	0,74	0,57	0,73	5,40	5,40
C1orf88	1,05	0,91	0,49	0,48	0,73	5,39	5,39
CDKN1C	0,68	0,82	0,70	0,72	0,73	5,38	5,38
A_32_P32463	0,79	0,76	0,65	0,72	0,73	5,37	5,37
PCDH9	0,53	0,77	0,78	0,81	0,72	5,31	5,31
NTN4	0,88	0,75	0,60	0,67	0,72	5,29	5,29
MID1	0,60	0,74	0,93	0,61	0,72	5,28	5,28
CSPG2	0,80	0,80	0,77	0,50	0,72	5,22	5,22

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
AL133090	0,96	0,89	0,66	0,35	0,71	5,15	5,15
DDC	0,76	1,01	0,50	0,55	0,71	5,08	5,08
AKR1C1	0,78	0,89	0,63	0,52	0,70	5,07	5,07
PHLDA1	0,68	0,67	0,64	0,79	0,70	4,98	4,98
KCNH2	0,38	0,83	0,90	0,65	0,69	4,86	4,86
ITGB8	0,67	0,62	0,75	0,66	0,68	4,74	4,74
ST6GAL1	0,78	0,91	0,69	0,32	0,67	4,72	4,72
TMEFF1	0,75	0,79	0,64	0,51	0,67	4,70	4,70
FBXL16	0,96	0,88	0,46	0,38	0,67	4,69	4,69
ATP8A2	0,79	0,76	0,76	0,34	0,66	4,60	4,60
CART1	0,69	0,76	0,78	0,40	0,66	4,57	4,57
C9orf150	0,57	0,62	0,69	0,75	0,66	4,53	4,53
PHLDA1	0,63	0,59	0,66	0,72	0,65	4,47	4,47
HDAC9	0,77	0,55	0,71	0,55	0,64	4,41	4,41
GLS	0,65	0,62	0,74	0,56	0,64	4,39	4,39
GATS	0,79	0,73	0,55	0,46	0,63	4,30	4,30
CNTNAP3	0,72	0,70	0,71	0,40	0,63	4,30	4,30
PDE4B	0,84	0,61	0,62	0,43	0,63	4,23	4,23
DKFZp586I1420	0,63	0,66	0,65	0,56	0,62	4,20	4,20
ZNRF2	0,64	0,60	0,66	0,58	0,62	4,18	4,18
FGF2	0,73	0,79	0,57	0,39	0,62	4,17	4,17
SP5	0,72	0,65	0,64	0,47	0,62	4,17	4,17
LAMA2	0,46	0,63	0,77	0,59	0,61	4,08	4,08
THC2056328	0,59	0,57	0,76	0,51	0,61	4,05	4,05
KIAA1666	0,75	0,68	0,53	0,46	0,61	4,03	4,03
A_23_P103951	0,73	0,67	0,61	0,41	0,60	4,02	4,02
IL1R1	0,39	0,62	0,77	0,63	0,60	4,00	4,00
CD55	0,74	0,66	0,59	0,39	0,59	3,93	3,93
MANEAL	0,75	0,70	0,55	0,37	0,59	3,93	3,93
AK026140	0,68	0,61	0,52	0,57	0,59	3,93	3,93
FXYD2	0,43	0,58	0,70	0,64	0,59	3,88	3,88
CD40	0,60	0,65	0,56	0,54	0,59	3,87	3,87
KIAA1505	0,76	0,65	0,62	0,31	0,59	3,86	3,86
DEPDC6	0,65	0,59	0,68	0,42	0,59	3,85	3,85
GLS	0,58	0,54	0,71	0,52	0,59	3,85	3,85
PPP2R2C	0,45	0,51	0,69	0,68	0,58	3,82	3,82

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
NRP1	0,44	0,60	0,70	0,58	0,58	3,80	3,80
SP5	0,67	0,64	0,56	0,43	0,58	3,77	3,77
ARHGDIB	0,46	0,56	0,72	0,56	0,57	3,75	3,75
RA12	0,84	0,64	0,34	0,47	0,57	3,74	3,74
LOC284262	0,70	0,74	0,52	0,31	0,57	3,69	3,69
TXNRD3	0,51	0,58	0,63	0,54	0,57	3,68	3,68
HIVEP1	0,54	0,54	0,63	0,56	0,57	3,68	3,68
BC042017	0,38	0,65	0,72	0,51	0,57	3,67	3,67
ABCB2	0,56	0,58	0,64	0,48	0,56	3,66	3,66
GLS	0,48	0,63	0,65	0,48	0,56	3,65	3,65
RASSF8	0,64	0,59	0,52	0,49	0,56	3,63	3,63
CR622072	0,58	0,47	0,64	0,54	0,56	3,62	3,62
LITAF	0,68	0,59	0,50	0,45	0,55	3,56	3,56
IGF2	0,67	0,67	0,49	0,38	0,55	3,56	3,56
CYR61	0,61	0,49	0,51	0,59	0,55	3,55	3,55
PHF15	0,55	0,55	0,56	0,54	0,55	3,54	3,54
ProSAPiP1	0,63	0,51	0,51	0,54	0,55	3,51	3,51
THC2227602	0,76	0,63	0,43	0,35	0,54	3,49	3,49
LOC389722	0,61	0,59	0,53	0,43	0,54	3,46	3,46
ANKRD18A	0,66	0,53	0,58	0,35	0,53	3,38	3,38
FBN1	0,71	0,54	0,55	0,31	0,53	3,37	3,37
RPS6KA2	0,39	0,47	0,62	0,62	0,52	3,34	3,34
GRB10	0,61	0,47	0,53	0,47	0,52	3,33	3,33
AY336981	0,51	0,45	0,54	0,59	0,52	3,31	3,31
RASSF8	0,61	0,56	0,49	0,42	0,52	3,31	3,31
SLPI	0,39	0,67	0,63	0,38	0,52	3,31	3,31
SLPI	0,37	0,69	0,64	0,37	0,52	3,30	3,30
THC2095463	0,61	0,51	0,53	0,40	0,52	3,27	3,27
COL16A1	0,57	0,63	0,50	0,35	0,51	3,26	3,26
GRAMD3	0,55	0,52	0,55	0,43	0,51	3,26	3,26
FXYD2	0,38	0,50	0,60	0,56	0,51	3,24	3,24
PDGFB	0,45	0,46	0,54	0,59	0,51	3,24	3,24
FAM107B	0,47	0,45	0,53	0,59	0,51	3,23	3,23
LOC440934	0,41	0,58	0,65	0,40	0,51	3,22	3,22
VCX	0,37	0,54	0,56	0,56	0,51	3,21	3,21
LAMB3	0,49	0,56	0,58	0,38	0,50	3,18	3,18

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
CYR61	0,54	0,46	0,46	0,53	0,50	3,14	3,14
NCALD	0,60	0,50	0,42	0,47	0,50	3,14	3,14
WNT5A	0,58	0,41	0,47	0,52	0,49	3,12	3,12
ABCC3	0,69	0,54	0,41	0,33	0,49	3,10	3,10
GLIS1	0,61	0,50	0,45	0,40	0,49	3,09	3,09
JAG1	0,60	0,50	0,45	0,39	0,49	3,06	3,06
NRL	0,48	0,51	0,52	0,41	0,48	3,03	3,03
AGT	0,43	0,38	0,42	0,68	0,48	3,01	3,01
TMSB4X	0,38	0,42	0,59	0,48	0,47	2,93	2,93
CCPG1	0,53	0,49	0,44	0,38	0,46	2,90	2,90
ADRA2C	0,45	0,44	0,51	0,45	0,46	2,88	2,88
BM665539	0,39	0,39	0,53	0,50	0,45	2,84	2,84
TEX15	0,44	0,41	0,53	0,41	0,45	2,82	2,82
SEMA3B	0,42	0,44	0,48	0,45	0,45	2,81	2,81
NYD-SP18	0,51	0,51	0,38	0,39	0,45	2,79	2,79
ASNS	0,50	0,37	0,48	0,43	0,44	2,79	2,79
NFKBIZ	0,43	0,31	0,54	0,49	0,44	2,77	2,77
AK096677	0,53	0,38	0,49	0,37	0,44	2,76	2,76
CA313037	0,52	0,50	0,41	0,33	0,44	2,76	2,76
PTPRM	0,45	0,43	0,45	0,42	0,44	2,75	2,75
SLC1A1	0,52	0,44	0,36	0,43	0,44	2,74	2,74
GRB10	0,40	0,35	0,50	0,50	0,44	2,73	2,73
NQO1	0,46	0,41	0,45	0,42	0,44	2,73	2,73
A_24_P401051	0,36	0,38	0,51	0,48	0,43	2,72	2,72
GPR161	0,50	0,47	0,39	0,37	0,43	2,71	2,71
A_32_P32905	0,58	0,41	0,43	0,30	0,43	2,70	2,70
LOC389652	0,47	0,35	0,46	0,42	0,43	2,68	2,68
SRGAP3	0,36	0,35	0,59	0,41	0,43	2,67	2,67
PDE4D	0,44	0,42	0,34	0,49	0,42	2,64	2,64
THC2055165	0,49	0,40	0,43	0,33	0,41	2,58	2,58
LOC63920	0,51	0,37	0,37	0,39	0,41	2,56	2,56
AK022020	0,52	0,44	0,34	0,33	0,41	2,55	2,55
MGAT4A	0,41	0,38	0,37	0,44	0,40	2,53	2,53
THC2201936	0,47	0,36	0,42	0,36	0,40	2,52	2,52
THC2091303	0,47	0,48	0,33	0,32	0,40	2,51	2,51
FCRL2	0,49	0,40	0,38	0,32	0,40	2,51	2,51

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
PDE4DIP	0,41	0,37	0,44	0,38	0,40	2,51	2,51
WBP5	0,41	0,45	0,39	0,35	0,40	2,50	2,50
PIM1	0,53	0,43	0,32	0,31	0,40	2,50	2,50
NUPR1	0,45	0,34	0,43	0,37	0,40	2,49	2,49
SMAD7	0,51	0,39	0,33	0,34	0,39	2,48	2,48
LOC63920	0,47	0,37	0,36	0,37	0,39	2,47	2,47
STAT1	0,45	0,49	0,30	0,33	0,39	2,47	2,47
AK057151	0,51	0,35	0,36	0,34	0,39	2,46	2,46
PPM1H	0,46	0,39	0,35	0,36	0,39	2,45	2,45
BM806490	0,40	0,39	0,40	0,36	0,39	2,45	2,45
AL390181	0,44	0,40	0,37	0,31	0,38	2,41	2,41
LOC150356	0,40	0,36	0,41	0,34	0,38	2,38	2,38
WNT7B	0,37	0,36	0,40	0,37	0,38	2,37	2,37
BF210146	0,41	0,32	0,37	0,39	0,37	2,36	2,36
LRP11	0,42	0,41	0,32	0,33	0,37	2,33	2,33
FGFR2	0,37	0,38	0,33	0,33	0,36	2,27	2,27
MT2A	0,44	0,34	0,32	0,32	0,35	2,26	2,26
BF378046	0,38	0,31	0,37	0,33	0,35	2,25	2,25
MT2A	0,42	0,33	0,32	0,32	0,35	2,22	2,22
BC037328	0,38	0,32	0,37	0,31	0,35	2,21	2,21
MT2A	0,43	0,33	0,31	0,31	0,34	2,21	2,21
ZNF323	0,33	0,35	0,34	0,35	0,34	2,20	2,20
TBC1D8	0,41	0,31	0,31	0,33	0,34	2,18	2,18
BLVRA	0,34	0,34	0,31	0,35	0,33	2,16	2,16
FGFR2	0,36	0,36	0,30	0,31	0,33	2,15	2,15

Table 4 : List of the under-expressed genes by at least two fold in the docetaxel resistant cell-lines.

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
PIK3C3	-0,32	-0,32	-0,33	-0,34	-0,33	0,47	-2,12
SCARB1	-0,39	-0,32	-0,35	-0,35	-0,35	0,45	-2,24
ASGR1	-0,32	-0,39	-0,40	-0,39	-0,38	0,42	-2,38
FLJ22659	-0,42	-0,31	-0,37	-0,40	-0,38	0,42	-2,38
ASMTL	-0,32	-0,39	-0,46	-0,35	-0,38	0,42	-2,40
ARHGAP10	-0,46	-0,37	-0,31	-0,39	-0,38	0,41	-2,41

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
EDG6	-0,44	-0,43	-0,36	-0,34	-0,39	0,41	-2,45
KCNC3	-0,46	-0,43	-0,32	-0,35	-0,39	0,41	-2,45
MGC11332	-0,48	-0,39	-0,36	-0,34	-0,39	0,41	-2,47
NRG1	-0,50	-0,39	-0,37	-0,32	-0,39	0,40	-2,47
LOC339240	-0,41	-0,45	-0,30	-0,42	-0,39	0,40	-2,48
PTPN3	-0,43	-0,36	-0,36	-0,43	-0,39	0,40	-2,48
AK123483	-0,39	-0,38	-0,42	-0,41	-0,40	0,40	-2,51
NRG1	-0,52	-0,40	-0,37	-0,32	-0,40	0,40	-2,51
FAM80A	-0,33	-0,31	-0,46	-0,52	-0,41	0,39	-2,55
BAMBI	-0,55	-0,36	-0,32	-0,40	-0,41	0,39	-2,56
PTPN3	-0,49	-0,42	-0,33	-0,43	-0,42	0,38	-2,62
SAMD8	-0,56	-0,44	-0,32	-0,36	-0,42	0,38	-2,62
KCNMB4	-0,58	-0,39	-0,37	-0,36	-0,43	0,38	-2,67
SPG20	-0,53	-0,45	-0,35	-0,40	-0,43	0,37	-2,70
RGS16	-0,58	-0,43	-0,34	-0,38	-0,43	0,37	-2,71
UGT2B7	-0,65	-0,45	-0,31	-0,34	-0,44	0,37	-2,74
LMBRD2	-0,39	-0,42	-0,48	-0,48	-0,44	0,36	-2,77
TMPRSS4	-0,68	-0,39	-0,35	-0,37	-0,45	0,36	-2,79
PDK1	-0,46	-0,46	-0,48	-0,39	-0,45	0,36	-2,80
RAB39B	-0,34	-0,54	-0,42	-0,50	-0,45	0,36	-2,81
HSPH1	-0,68	-0,50	-0,31	-0,35	-0,46	0,35	-2,89
PSCDBP	-0,44	-0,46	-0,50	-0,46	-0,46	0,34	-2,91
UGT2B11	-0,69	-0,45	-0,34	-0,38	-0,46	0,34	-2,91
ZNF516	-0,55	-0,46	-0,39	-0,49	-0,47	0,34	-2,95
CKB	-0,58	-0,49	-0,36	-0,46	-0,47	0,34	-2,98
SLC7A8	-0,34	-0,42	-0,61	-0,56	-0,48	0,33	-3,05
UGT2B28	-0,76	-0,50	-0,34	-0,37	-0,49	0,32	-3,10
SEMA3C	-0,59	-0,56	-0,35	-0,47	-0,49	0,32	-3,11
PDK1	-0,52	-0,50	-0,50	-0,45	-0,49	0,32	-3,11
GATA2	-0,55	-0,50	-0,50	-0,44	-0,50	0,32	-3,13
THC2064535	-0,52	-0,45	-0,51	-0,51	-0,50	0,32	-3,16
PDLIM5	-0,60	-0,53	-0,42	-0,45	-0,50	0,32	-3,16
BX538293	-0,54	-0,56	-0,39	-0,52	-0,50	0,31	-3,18
HYAL1	-0,70	-0,56	-0,36	-0,40	-0,50	0,31	-3,20
UGT2B4	-0,74	-0,55	-0,34	-0,39	-0,51	0,31	-3,20
THC2185385	-0,54	-0,48	-0,39	-0,62	-0,51	0,31	-3,21

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
FLJ10159	-0,56	-0,49	-0,46	-0,53	-0,51	0,31	-3,24
UGT2B10	-0,75	-0,54	-0,35	-0,41	-0,51	0,31	-3,26
PRTFDC1	-0,49	-0,50	-0,64	-0,42	-0,51	0,31	-3,26
A_24_P575267	-0,78	-0,50	-0,36	-0,42	-0,51	0,31	-3,26
PTGES	-0,68	-0,59	-0,40	-0,38	-0,51	0,31	-3,27
DNAJC15	-0,78	-0,57	-0,35	-0,37	-0,52	0,30	-3,29
NPAS1	-0,67	-0,57	-0,46	-0,37	-0,52	0,30	-3,29
A_24_P478940	-0,70	-0,58	-0,40	-0,39	-0,52	0,30	-3,30
FRMD4B	-0,49	-0,50	-0,58	-0,52	-0,52	0,30	-3,34
SLC7A11	-0,58	-0,62	-0,39	-0,52	-0,53	0,30	-3,36
AF132203	-0,61	-0,47	-0,42	-0,61	-0,53	0,30	-3,36
FLJ43855	-0,61	-0,54	-0,50	-0,46	-0,53	0,30	-3,38
TFDP2	-0,67	-0,65	-0,46	-0,33	-0,53	0,30	-3,38
PFKFB4	-0,54	-0,56	-0,51	-0,50	-0,53	0,30	-3,38
TAC3	-0,55	-0,46	-0,50	-0,61	-0,53	0,30	-3,38
ENC1	-0,74	-0,50	-0,49	-0,40	-0,53	0,29	-3,40
EPAS1	-0,54	-0,50	-0,49	-0,61	-0,54	0,29	-3,43
NRP2	-0,72	-0,54	-0,49	-0,42	-0,54	0,29	-3,48
MFHAS1	-0,56	-0,56	-0,53	-0,53	-0,54	0,29	-3,50
AK024680	-0,68	-0,50	-0,47	-0,52	-0,54	0,29	-3,51
SYTL3	-0,64	-0,68	-0,45	-0,42	-0,55	0,28	-3,53
BDNF	-0,63	-0,46	-0,50	-0,62	-0,55	0,28	-3,57
AL137342	-0,80	-0,41	-0,41	-0,64	-0,56	0,27	-3,67
D4S234E	-0,69	-0,44	-0,46	-0,68	-0,57	0,27	-3,70
LCP1	-0,92	-0,52	-0,30	-0,54	-0,57	0,27	-3,72
A_32_P95067	-0,72	-0,63	-0,54	-0,39	-0,57	0,27	-3,73
THC2038567	-0,86	-0,75	-0,38	-0,32	-0,57	0,27	-3,75
LMO1	-0,61	-0,56	-0,61	-0,52	-0,58	0,27	-3,77
BSPRY	-0,72	-0,46	-0,52	-0,61	-0,58	0,26	-3,78
BDNF	-0,61	-0,49	-0,56	-0,67	-0,58	0,26	-3,81
UGT8	-0,78	-0,44	-0,48	-0,63	-0,58	0,26	-3,81
LIPG	-0,85	-0,67	-0,33	-0,51	-0,59	0,26	-3,89
AW205591	-0,74	-0,79	-0,52	-0,32	-0,59	0,26	-3,90
AKAP12	-0,94	-0,68	-0,32	-0,43	-0,59	0,25	-3,92
B3GALT1	-0,54	-0,53	-0,63	-0,67	-0,59	0,25	-3,93
CHST7	-0,79	-0,64	-0,47	-0,49	-0,59	0,25	-3,93

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
NMNAT2	-0,86	-0,74	-0,44	-0,36	-0,60	0,25	-3,96
SLC12A3	-0,86	-0,67	-0,31	-0,57	-0,60	0,25	-3,98
SLC22A2	-0,94	-0,67	-0,40	-0,43	-0,61	0,25	-4,05
LMBRD2	-0,67	-0,61	-0,55	-0,62	-0,61	0,24	-4,09
SCD	-0,68	-0,51	-0,53	-0,74	-0,61	0,24	-4,11
AMPH	-0,89	-0,57	-0,49	-0,53	-0,62	0,24	-4,17
ANKRD37	-0,76	-0,66	-0,48	-0,59	-0,62	0,24	-4,18
LIN7A	-0,98	-0,59	-0,39	-0,52	-0,62	0,24	-4,19
PHEX	-0,88	-0,76	-0,42	-0,44	-0,62	0,24	-4,22
C1QL1	-0,59	-0,55	-0,72	-0,64	-0,63	0,24	-4,24
EPAS1	-0,63	-0,57	-0,63	-0,72	-0,64	0,23	-4,34
KCNC4	-0,75	-0,67	-0,50	-0,64	-0,64	0,23	-4,36
FOXL2	-0,81	-0,68	-0,47	-0,61	-0,64	0,23	-4,38
SCD	-0,77	-0,58	-0,62	-0,61	-0,64	0,23	-4,40
FGFBP1	-0,99	-0,64	-0,47	-0,50	-0,65	0,22	-4,48
CASP8	-0,40	-0,77	-0,65	-0,80	-0,65	0,22	-4,51
LZTS1	-0,81	-0,71	-0,65	-0,46	-0,66	0,22	-4,53
SYTL3	-0,74	-0,81	-0,54	-0,53	-0,66	0,22	-4,54
HSHPX5	-0,80	-0,61	-0,63	-0,59	-0,66	0,22	-4,56
NLGN1	-0,41	-0,33	-0,86	-1,05	-0,66	0,22	-4,62
MGST1	-0,98	-0,68	-0,45	-0,58	-0,67	0,21	-4,70
PLXNA2	-0,73	-0,60	-0,49	-0,88	-0,68	0,21	-4,75
ST8SIA4	-0,75	-0,53	-0,59	-0,85	-0,68	0,21	-4,77
THC2050576	-1,00	-0,72	-0,50	-0,51	-0,68	0,21	-4,78
SLC3A1	-0,87	-0,74	-0,62	-0,51	-0,68	0,21	-4,82
TNRC9	-0,82	-0,62	-0,51	-0,79	-0,68	0,21	-4,84
AK022997	-1,23	-0,47	-0,36	-0,70	-0,69	0,20	-4,88
UGT8	-0,94	-0,51	-0,56	-0,76	-0,69	0,20	-4,93
LETM2	-1,04	-0,83	-0,41	-0,50	-0,70	0,20	-4,97
PLAC8	-1,07	-0,80	-0,39	-0,53	-0,70	0,20	-4,99
DIAPH2	-1,18	-0,71	-0,38	-0,54	-0,70	0,20	-5,05
BC014452	-0,91	-0,61	-0,56	-0,76	-0,71	0,20	-5,12
SUNC1	-0,95	-0,79	-0,60	-0,52	-0,72	0,19	-5,20
DUSP13	-0,84	-0,74	-0,61	-0,71	-0,73	0,19	-5,32
AUTS2	-0,59	-0,52	-0,84	-1,02	-0,74	0,18	-5,51
PLAC8	-1,15	-0,84	-0,43	-0,55	-0,74	0,18	-5,52

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
THC2210862	-1,43	-0,77	-0,43	-0,36	-0,75	0,18	-5,61
MSX2	-0,96	-0,69	-0,72	-0,68	-0,76	0,17	-5,79
SMAD9	-0,79	-0,80	-0,77	-0,70	-0,76	0,17	-5,81
TTN	-0,86	-0,74	-0,55	-0,91	-0,77	0,17	-5,82
LRRN6C	-1,00	-0,73	-0,53	-0,82	-0,77	0,17	-5,88
MEIS2	-1,12	-0,92	-0,67	-0,38	-0,77	0,17	-5,91
DHRS3	-1,03	-0,75	-0,65	-0,67	-0,77	0,17	-5,95
OLR1	-1,34	-0,95	-0,31	-0,58	-0,80	0,16	-6,25
NSBP1	-1,18	-0,84	-0,55	-0,64	-0,80	0,16	-6,30
MOXD1	-0,90	-0,70	-0,80	-0,82	-0,80	0,16	-6,37
DCAMKL1	-1,11	-0,84	-0,65	-0,63	-0,81	0,16	-6,40
C12orf59	-1,01	-0,81	-0,64	-0,76	-0,81	0,16	-6,42
A_23_P136857	-1,10	-0,99	-0,71	-0,52	-0,83	0,15	-6,75
BF675806	-1,09	-0,87	-0,72	-0,65	-0,83	0,15	-6,82
SLC44A5	-1,28	-0,83	-0,73	-0,54	-0,84	0,14	-6,96
SALL1	-1,26	-1,04	-0,74	-0,36	-0,85	0,14	-7,03
GPR177	-1,27	-0,89	-0,62	-0,63	-0,85	0,14	-7,13
AK3L1	-1,27	-0,99	-0,70	-0,49	-0,86	0,14	-7,28
AK3L1	-1,14	-0,99	-0,76	-0,56	-0,86	0,14	-7,29
FZD8	-0,94	-0,79	-0,85	-0,93	-0,88	0,13	-7,52
THC2088463	-0,98	-0,95	-0,73	-0,85	-0,88	0,13	-7,55
FLJ39502	-0,87	-0,97	-0,69	-1,01	-0,88	0,13	-7,65
PROS1	-1,02	-0,87	-0,69	-0,97	-0,89	0,13	-7,76
PTPRD	-1,28	-0,82	-0,66	-0,85	-0,90	0,13	-7,97
PDE1A	-1,24	-1,13	-0,72	-0,53	-0,90	0,12	-8,01
MYB	-0,89	-0,81	-0,86	-1,05	-0,90	0,12	-8,01
SLC16A10	-1,19	-1,22	-0,74	-0,49	-0,91	0,12	-8,08
GJA7	-1,18	-0,87	-0,80	-0,80	-0,91	0,12	-8,12
GAL	-1,22	-0,96	-0,71	-0,81	-0,92	0,12	-8,40
CPM	-1,17	-0,95	-0,83	-0,79	-0,93	0,12	-8,57
PDE1A	-1,40	-1,12	-0,73	-0,52	-0,94	0,11	-8,75
PLXNA2	-1,10	-0,82	-0,58	-1,28	-0,95	0,11	-8,86
PDE1A	-1,21	-1,30	-0,76	-0,53	-0,95	0,11	-8,88
AW467174	-1,37	-1,23	-0,73	-0,49	-0,95	0,11	-8,95
PLAT	-1,34	-1,10	-0,43	-0,95	-0,95	0,11	-8,98
LOC441047	-1,28	-1,19	-0,77	-0,58	-0,95	0,11	-9,01

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	50nM	25nM	12nM	5nM	Mean	Mean	Mean
CXCR4	-1,59	-1,32	-0,53	-0,41	-0,96	0,11	-9,16
AK3L1	-1,35	-1,13	-0,82	-0,57	-0,97	0,11	-9,27
SMPDL3A	-1,46	-1,05	-0,67	-0,71	-0,97	0,11	-9,37
KIAA0960	-1,36	-0,63	-0,52	-1,38	-0,97	0,11	-9,39
LHFP	-1,26	-1,02	-0,97	-0,66	-0,98	0,11	-9,52
CPM	-1,17	-0,96	-0,92	-0,86	-0,98	0,11	-9,52
A_24_P345290	-1,33	-1,22	-0,85	-0,55	-0,99	0,10	-9,75
RXFP1	-1,56	-1,09	-0,69	-0,64	-1,00	0,10	-9,94
PNOC	-1,04	-1,15	-0,92	-0,91	-1,01	0,10	-10,18
GALNT14	-1,39	-1,16	-0,68	-0,81	-1,01	0,10	-10,26
TM4SF1	-0,98	-1,11	-1,03	-0,98	-1,02	0,09	-10,59
ZAR1	-1,08	-0,98	-1,03	-1,04	-1,03	0,09	-10,75
A_23_P10091	-1,12	-0,86	-1,33	-0,91	-1,05	0,09	-11,31
GLT8D2	-1,28	-1,21	-0,92	-0,84	-1,06	0,09	-11,53
RXFP1	-1,81	-1,24	-0,71	-0,55	-1,08	0,08	-11,90
CGNL1	-1,40	-1,26	-0,95	-0,73	-1,08	0,08	-12,08
AK094972	-1,43	-1,35	-0,75	-1,05	-1,14	0,07	-13,93
LRCH2	-1,27	-1,00	-1,15	-1,34	-1,19	0,06	-15,54
BM930757	-1,30	-1,11	-1,20	-1,20	-1,20	0,06	-15,91
ATP8A1	-1,34	-1,38	-1,15	-0,98	-1,21	0,06	-16,34
SOX9	-1,25	-1,31	-1,34	-0,97	-1,22	0,06	-16,47
SLC39A8	-1,36	-1,12	-1,32	-1,22	-1,25	0,06	-17,94
TMEM47	-1,59	-1,10	-1,13	-1,29	-1,28	0,05	-18,94
SLC10A4	-1,23	-1,12	-1,28	-1,51	-1,29	0,05	-19,33
SLC1A3	-1,49	-1,34	-1,01	-1,38	-1,30	0,05	-20,08
EDG7	-1,35	-1,32	-1,27	-1,36	-1,33	0,05	-21,23
ITGA2	-1,74	-1,39	-1,03	-1,45	-1,40	0,04	-25,31
SLC1A3	-1,75	-1,42	-1,09	-1,37	-1,41	0,04	-25,49
PLCXD3	-1,28	-1,22	-1,33	-1,93	-1,44	0,04	-27,49
BF514799	-1,37	-1,45	-1,38	-1,70	-1,47	0,03	-29,80
SLC16A12	-1,56	-1,34	-1,54	-1,50	-1,48	0,03	-30,49
THC2208430	-1,88	-1,83	-1,03	-1,39	-1,53	0,03	-34,08
THC2182743	-1,60	-1,49	-1,47	-1,67	-1,56	0,03	-36,19
C4orf18	-1,67	-1,49	-1,58	-1,57	-1,58	0,03	-37,64
ANKRD38	-1,71	-1,50	-1,53	-1,62	-1,59	0,03	-38,90
CALCRL	-1,74	-1,77	-1,92	-1,84	-1,82	0,02	-66,13

(continued)

	Log(Ratio) for each dose				Log (ratio)	Ratio	Fold change
Primary Sequence Name	**50nM**	**25nM**	**12nM**	**5nM**	**Mean**	**Mean**	**Mean**
LOC152573	-2,17	-2,80	-2,05	-1,79	-2,20	0,01	-159,40

Claims

1. An *in vitro* method for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family, wherein the method comprises:

   1) providing a biological sample from said subject; 2) determining in the biological sample the expression level of at least 5 genes selected from the group consisting of the genes listed in Tables 1 and 2, thereby predicting or monitoring whether a patient affected by a prostate cancer is responsive to a treatment with a molecule of the taxoid family.

2. The method according to claim 1, wherein the method further comprises comparing the expression level of said at least 5 genes to a reference expression level, the reference expression level being the expression level of the genes in cell-lines or patients sensitive to the treatment by the molecule of the taxoid family.

3. The method according to claim 2, wherein the over-expression of genes from Table 1 and/or the under-expression of genes from Table 2 are indicative of a resistance to the treatment by the molecule of the taxoid family.

4. The method according to anyone of claims 1-3, wherein the at least 5 genes are selected from one of the following groups or a combination thereof:

   a) RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, WNT2B, GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and LOC152573, preferably RPIB9, CXCL2, TFPI2, TNF, ABCB1, ADAMTS5, PURG, OAS3, GAS1, BIRC3, MAL, GALNT14, TM4SF1, RXFP1, ATP8A1, SOX9, SLC39A8, EDG7, ITGA2, SLC1A3, CALCRL and LOC152573;
   b) RPIB9, TFPI2, ABCB1, BIRC3, WNT2B, SFRP1, FSTL1, AHR, CDKN1C, ABCB2, CYR61, WNT5A, ABCC3, JAG1, STAT1, WNT7B, CASP8, LZTS1, FZD8, GALNT14, RXFP1 and LOC152573;
   c) ABCC3, CD55, COL16A1, DHRS3, FSTL1, GLS, HDL, HIVE1, LAMA2, LAMB3, LIPG, LITAF, MAL, MFHAS1, NFKBIZ, NRP1, NRP2, OAS3, OLR1, PSCDBP, RFTN1, SCARAB1, SEMA3B, SEMA3C, SFRP1, SLC1A3, ST6GAL1, TLR3, TM4SF1 and TNF;
   d) ADAMTS1, ADRA2C, AKAP12, CDKN1C, CYR61, FBN1, GAS1, GPC3, IGF2, IGFBP3, JAG1, MGST1, NTN4, PDE1A, PDE4B, PDE4D, PDE4DIP, PDGFB, PHLDA1, PIM1, PPP2R2C, RGS16, SCD, SLC1A1, SMPDL3A, TFP12 and VCAN;
   e) ABCB1, AHR, AHRR, AMPH, BIRC3, CXCL2, CYP1A1, IL1R1, NQO1, PLAT, PLXNA2, SLC16A10, SLC3A1, SLC7A8, SLPI, TAP1, UGT8, UGT2B4, UGT2B7, UGT2B10, UGT2B11 and UGT2B28;
   f) AQP1, ARHGDIB, BAMBI, CREB5, CXCR4, EPAS1, FGF2, FGFBP1, GRB10, IL15, MT2A, NUPR1, PDK1, PROS1, PTPN3, RPS6KA2, TFDP2, WNT2B, WNT5A and WNT7B;
   g) AGT, ATP8A2, BDNF, EDG6, GAL, GATA2, ITGA2, LRP11, LZTS1, MYB, NCALD, PNOC, PTGES, SRGAP3, TAC3 and TTN;
   h) AFF1, ASGR1, BLVRA, CASP8, CD40, KCNH2, NRG1, NRL, PHEX, PLAC8, SMAD7, SMAD9, SOX9, SPG20 and STAT1;
   i) TNF, ABCB1, CYP1A1, AHRR, AHR, PP2R2C, ABCC3, NQO1, PIK3C3, UGT2B7, UGT2B11, UGT2B28, UGT2B4, UGT2B10, CHST7, MGST1 and UGT8; and,
   j) Wnt2B, Wnt5A, Wnt7B, SFRP1, FSTL1, Jag1, Cyr61, LOC152573, FZD8 and FOXL2.

5. The method according to anyone of claims 1-4, wherein the molecule of the taxoid family is docetaxel, larotaxel, XRP6258, BMS-184476, BMS-188797, BMS-275183, ortataxel, RPR 109881A, RPR 116258, NBT-287, PG-pacl-

itaxel, ABRAXANE®, Tesetaxel, IDN 5390, Taxoprexin, DHA-paclitaxel, and MAC-321, more preferably docetaxel.

6. The method according to anyone of claims 1-5, wherein the method comprises determining the expression level of at least 10, 15, 20, 25, 30, 40, 50, 60, 70, 80, 90, 100, 150, 200 or 300 genes from those listed in Tables 1 and 2.

7. The method according to anyone of claims 1-6, wherein the expression level of genes is determined by the quantity of protein or mRNA encoded by said genes.

8. The method according to anyone of claims 1-7, wherein the biological sample is a cancer sample.

9. The method according to anyone of claims 1-8, wherein the cancer is selected from the group consisting of the breast cancer, the lung cancer, the prostate cancer, the gastric cancer and the head and neck cancer, more preferably a prostate cancer.

10. A kit for predicting or monitoring whether a patient affected by a cancer is responsive to a treatment with a molecule of the taxoid family, wherein the kit comprises detection means selected from the group consisting of a pair of primers, a probe and an antibody specific to at least 5 genes selected from the group consisting of the genes listed in Tables 1 and 2.

11. A DNA chip comprising a solid support which carries nucleic acids that are specific to at least 5 genes selected from the group consisting of the genes listed in Tables 1 and 2.

12. Kit according to claim 10 or DNA chip according to claim 11, wherein the at least 5 genes are selected from one of the following groups or a combination thereof:

a) RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, WNT2B, GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orfl8, ANKRD38, CALCRL, and LOC152573, preferably RPIB9, CXCL2, TFPI2, TNF, ABCB1, ADAMTS5, PURG, OAS3, GAS1, BIRC3, MAL, GALNT14, TM4SF1, RXFP1, ATP8A1, SOX9, SLC39A8, EDG7, ITGA2, SLC1A3, CALCRL and LOC152573;
b) RPIB9, TFPI2, ABCB1, BIRC3, WNT2B, SFRP1, FSTL1, AHR, CDKN1C, ABCB2, CYR61, WNT5A, ABCC3, JAG1, STAT1, WNT7B, CASP8, LZTS1, FZD8, GALNT14, RXFP1 and LOC152573;
c) ABCC3, CD55, COL16A1, DHRS3, FSTL1, GLS, HDL, HIVE1, LAMA2, LAMB3, LIPG, LITAF, MAL, MFHAS1, NFKBIZ, NRP1, NRP2, OAS3, OLR1, PSCDBP, RFTN1, SCARAB1, SEMA3B, SEMA3C, SFRP1, SLC1A3, ST6GAL1, TLR3, TM4SF1 and TNF;
d) ADAMTS1, ADRA2C, AKAP12, CDKN1C, CYR61, FBN1, GAS1, GPC3, IGF2, IGFBP3, JAG1, MGST1, NTN4, PDE1A, PDE4B, PDE4D, PDE4DIP, PDGFB, PHLDA1, PIM1, PPP2R2C, RGS16, SCD, SLC1A1, SMPDL3A, TFPI2 and VCAN;
e) ABCB1, AHR, AHRR, AMPH, BIRC3, CXCL2, CYP1A1, IL1R1, NQO1, PLAT, PLXNA2, SLC16A10, SLC3A1, SLC7A8, SLPI, TAP1, UGT8, UGT2B4, UGT2B7, UGT2B10, UGT2B11 and UGT2B28;
f) AQP1, ARHGDIB, BAMBI, CREB5, CXCR4, EPAS1, FGF2, FGFBP1, GRB10, IL15, MT2A, NUPR1, PDK1, PROS1, PTPN3, RPS6KA2, TFDP2, WNT2B, WNT5A and WNT7B;
g) AGT, ATP8A2, BDNF, EDG6, GAL, GATA2, ITGA2, LRP11, LZTS1, MYB, NCALD, PNOC, PTGES, SRGAP3, TAC3 and TTN;
h) AFF1, ASGR1, BLVRA, CASP8, CD40, KCNH2, NRG1, NRL, PHEX, PLAC8, SMAD7, SMAD9, SOX9, SPG20 and STAT1;
i) TNF, ABCB1, CYP1A1, AHRR, AHR, PP2R2C, ABCC3, NQO1, PIK3C3, UGT2B7, UGT2B11, UGT2B28, UGT2B4, UGT2B10, CHST7, MGST1 and UGT8; and,
j) Wnt2B, Wnt5A, Wnt7B, SFRP1, FSTL1, Jag1, Cyr61, LOC152573, FZD8 and FOXL2.

13. A method for screening or identifying a compound suitable for improving the treatment of a cancer with a molecule of the taxoid family or for reducing the resistance development during the treatment of a cancer with a molecule of the taxoid family, comprising

1) providing a cell-line with at least 5 genes over-expressed and/or under-expressed respectively selected from the group of over-expressed genes of Table 1 and under-expressed genes of Table 2; 2) contacting said cell-

line with a test compound; 3) determining the expression level of said at least 5 genes; and, 4) selecting the compound which decreases the expression level of the over-expressed genes and increases the expression level of the under-expressed genes.

14. A method for screening or identifying a compound suitable for improving the treatment of a cancer with a molecule of the taxoid family or for reducing the resistance development during the treatment of a cancer with the molecule of the taxoid family, comprising

1) providing a cell-line sensitive to the molecule of the taxoid family; 2) contacting said cell-line with a test compound and the molecule of the taxoid family; 3) determining the expression level of said at least 5 genes selected from the genes listed in Tables 1 and 2; and, 4) selecting the compound which inhibits the appearance of an over-expression and/or an under-expression of at least 5 genes respectively selected from the group of genes of Table 1 and genes of Table 2.

15. A method for screening or identifying a compound suitable for improving the treatment of a cancer with a molecule of the taxoid family or for reducing the resistance development during the treatment of a cancer with the molecule of the taxoid family, comprising

1) providing a cell-line with at least on gene over-expressed and/or under-expressed respectively selected from the group consisting of RPIB9, CXCL2, AL137761, TFPI2, THC2051204, TNF, ABCB1, PURG, ADAMTS5, MCTP1, SPTLC2L, OAS3, MCTP1, GAS1, BIRC3, BQ186674, MAL, UBXD3, and WNT2B for the over-expressed genes, and GALNT14, TM4SF1, ZAR1, A_23_P10091, GLT8D2, RXFP1, CGNL1, AK094972, LRCH2, BM930757, ATP8A1, SOX9, SLC39A8, TMEM47, SLC10A4, SLC1A3, EDG7, ITGA2, PLCXD3, BF514799, SLC16A12, THC2208430, THC2182743, C4orf18, ANKRD38, CALCRL, and LOC152573 for the under-expressed genes; 2) contacting said cell-line with a test compound; 3) determining the expression level of said at least one gene; and, 4) selecting the compound which decreases the expression level of over-expressed genes and increases the expression level of under-expressed genes.

16. The method according to any one of claims 13 to 15, wherein the cell-line is a cancer cell-line.

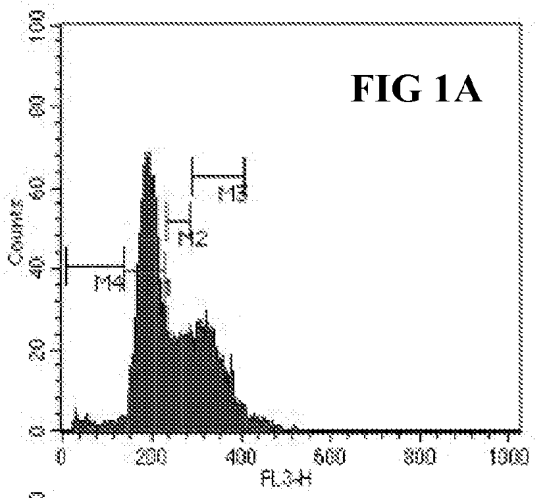

FIG 1A

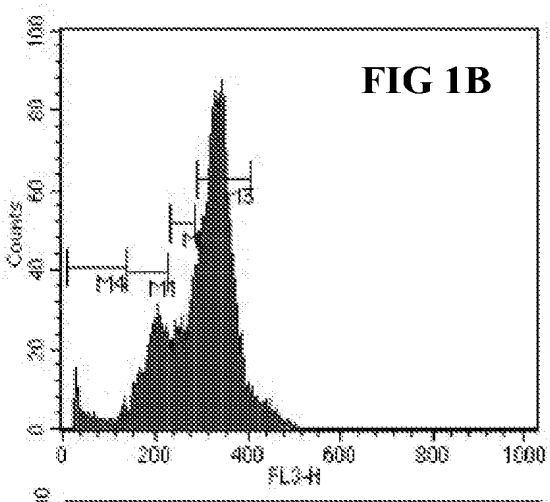

FIG 1B

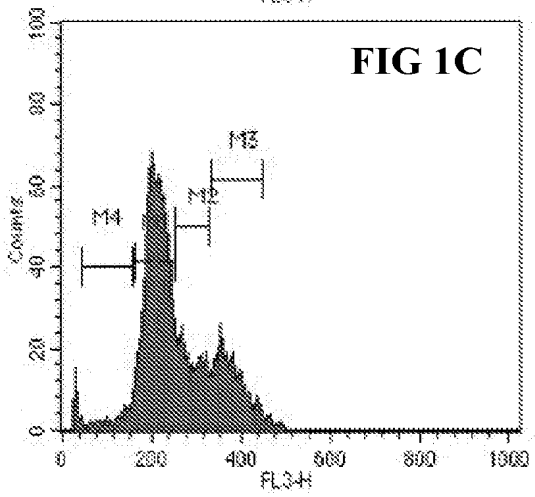

FIG 1C

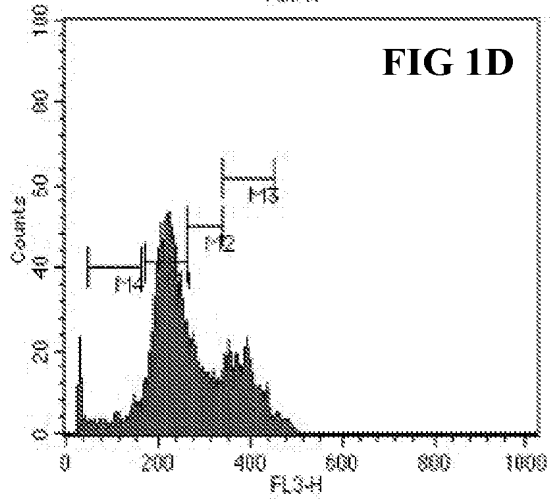

FIG 1D