Non-Confidential

May 2014

Cautionary Statement Regarding Forward-Looking Information

This material includes forward-looking statements based on assumptions and beliefs regarding the information currently available to CanBas and subject to significant risks and uncertainties.

The forward-looking statements contain information on pharmaceuticals (including compounds under development), but this information is not intended to make any promise or guarantee with respect to the efficacy or effectiveness of these preparations nor provide medical advice of any kind.

Actual results may differ materially from those indicated in the forward-looking statements based on a number of factors such as adverse legislative and regulatory developments, patient conditions, dosage and administration, etc.

The audience is cautioned not to place undue reliance on these forward-looking statements, which speak only as of the date of this presentation. CanBas undertakes no obligation to update any forward-looking statement to reflect new information, events or circumstances after the date of this presentation or to reflect the occurrence of unanticipated events.

CanBas Co., Ltd.

Founded: Jan. 2000

IPO: Sep. 2009

Stock Symbol: M-4575

Headquarters: 2-2-1 Otemachi, Numazu City
Shizuoka 410-0801 Japan

Employees: 19, including 6 board members

Management:

- Chief Executive Officer: Takumi Kawabe, MD, PhD
- Chief Finance Officer: Makoto Katozumi
- Corporate Planning: Kazuyoshi Sakamoto

Scientific Advisory Board

Daniel D. Von Hoff, MD, FACP (Chairman)

Professor of Medicine, Pathology, Molecular and Cellular Biology, and Director, the Arizona Health Sciences Center's Cancer Therapeutics Program. Dr. Von Hoff's major interest is in the development of new anticancer agents, both in the clinic and in the laboratory (including mitoxantrone, fludarabine, paclitaxel, docetaxel, gemcitabine, CPT-11). He has published more than 515 papers, 127 book chapters, and more than 850 abstracts, and is former President of AACR and former board member of ASCO. He is a founder of ILEX Oncology, Inc. (acquired by Genzyme). He is founder and Editor Emeritus of Investigational New Drugs - The Journal of New Anticancer Agents; and, Editor-in-Chief of Molecular Cancer Therapeutics.

Donald W. Kufe, MD

Professor of Medicine, Harvard; Vice Director, Dana-Farber Cancer Institute. Dr. Kufe received his MD in 1970 from the University of Rochester School of Medicine, and postgraduate training at Harvard's Beth Israel Hospital. Subsequently, he undertook extensive laboratory-based research in molecular virology at Columbia's Institute of Cancer Research. In 1979, he joined Dana-Farber. He has served as Chief of the Division of Cancer Pharmacology, Deputy Director of the Dana-Farber Cancer Center, Director of the Harvard Phase I Oncology Group and Leader of the Experimental Therapeutics Program. He has served as the senior editor of Cancer Medicine, one of the major text books in oncology, and on the editorial board of multiple international cancer research journals.

William G. Dunphy, PhD

Professor, California Institute of Technology, Researcher, Howard Hughes Medical Institute and Adjunct Assistant Professor of Biochemistry and Molecular Biology at the USC School of Medicine. He holds an A.B. degree in Biochemical Sciences from Harvard College and Ph.D. in biochemistry from Stanford University. He was a Helen Hay Whitney postdoctoral fellow at the University of California, San Diego.

CanBas
Cancer therapy by Basic research

Product Pipeline

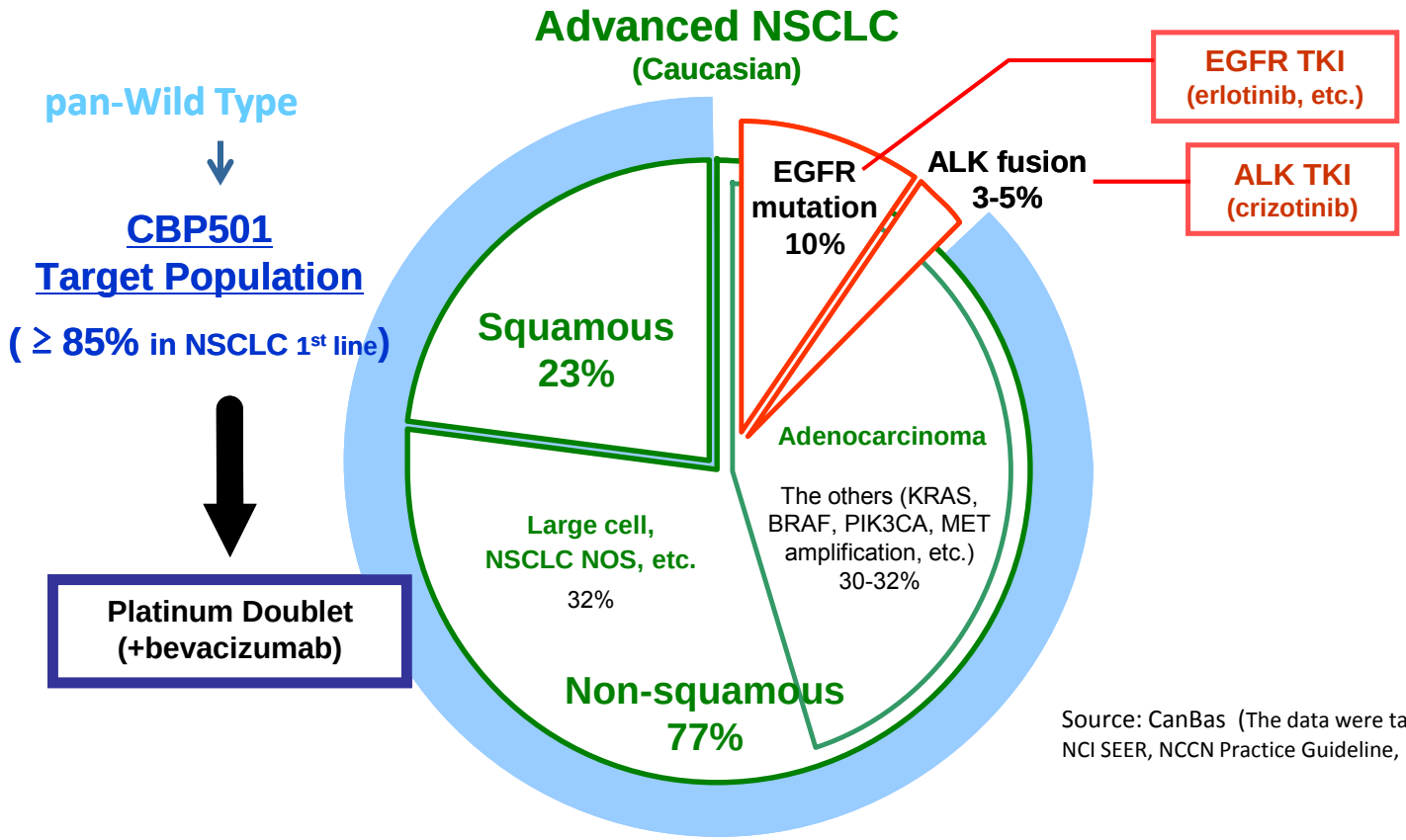
Product	Indication	Preclinical	Phase I	Phase II
<CBP501>				
Single Agent	Solid tumor	→		
Cisplatin adjunct	Ovarian	→		
Cisplatin/pemetrexed adjunct	MPM	→	→	→
	NSCLC	→	→	→
<CBS9106>	Hematopoietic, Lung etc.	→		

Summary of the clinical studies on CBP501

Stage	Indication	regimem	High lights
Phase I	Solid tumors (30 pts)	**Single agent** (qw x3 with a week off = 28days/cycle)	・Recommended dose（CBP501 25mg/m^2+Cis 75mg/m^2） Easily manageable histamine release syndrome is the only additional AE at RD
	Solid tumors (48 pts) Incl. Exp Cohort <Platinum Resist Ovarian>	**Combo: CBP501+Cisplatin** (q3w)	・Sign of activity on MPM and Platinum resistant ovarian carcinoma ・《Results on heavily pretreated platinum ref/resist ovarian carcinoma》 ORR(RECIST)：PR 14%(2pts), SD>3M 36%(5pts) Response by CA-125：PR 36%(5pts) PFS: 5.2M
	Solid tumors (6 pts)	**Triplet: Pemetrexed,Cisplatin,CBP501 (Phase I part of a Phase I/II study)** (q3w)	・Recommended dose CBP501 25mg/m^2+Cis 75mg/m^2+Pem 500mg/m^2 ・Sing of activity in MPM PR 1/4 SD 3/4
Phase II	Malignant pleural mesothelioma <first line> (65 pts)	**Triplet: Pemetrexed, Cisplatin ± CBP501 (Phase II part)** (q3w)	・Primary endpoint achieved PFS rate at 4M was 68%, the success criteria was >55% ・No addition of ≥G3 AEs to Chemotherapy （Experimental arm 53%(21/40) vs Chemo alone 61%(14/23)）
	Non-squamous NSCLC <first line> (192 pts)	**Triplet: Pemetrexed, Cisplatin ± CBP501** (q3w)	・Primary endpoint NOT achieved, however, OS prolonged in a major subgroup of patients which lead to the identification of additional MoA ・No addition of ≥G3 AEs to Chemotherapy

CBP501

- Improving standard of care
  - The only approved drugs targeting mutations on NSCLC are EGFR and ALK TKIs
  - “pan-Wild Type” patients, who have no actionable mutations, can only rely on “Platinum Doublets”



Source: CanBas (The data were taken from NCI SEER, NCCN Practice Guideline, etc.)

CBP501

- Excellent safety profile in combination therapies - NO additional AEs≥G3 to the standard of care

< MPM >

Name	MOA	Target	Regimen	Additional AEs (?G3)	Stage
CBP501	Calmodulin modulation	Unselected	CDDP+PEM **+CBP501** CDDP+PEM	**None** (P2)	P2 completed
Bevacizumab	Anti-VEGF mAb	Unselected	CDDP+PEM **+BEV** CDDP+PEM	PE(6%) (P2)	P2/3 ongoing

< NSCLC >

Name	MOA	Target	Regimen	Additional AEs(?G3)	Stage
CBP501	Calmodulin modulation	Non-squamous	CDDP+PEM **+CBP501** CDDP+PEM	**None** (P2)	P2 completed
Bevacizumab	Anti-VEGF mAb	Non-squamous	CARB+PAC **+BEV** CARB+PAC	Bleeding event (4%) (P3)	Approved
Necitumumab	Anti-EGFR mAb	Squamous	CDDP+GEM **+NEC** CDDP+GEM	— (No information)	P3 ongoing
Ipilimumab	Anti-CTLA 4 mAb	Squamous	CARB+PAC **+IPI** CARB+PAC+Placebo	Immune-related AEs(15%) (P2)	P3 ongoing

CBP501, a unique calmodulin inhibitory peptide

Originally expected mode of action was
"increase platinum concentration in CBP501 sensitive tumors"
without increasing adverse activity of platinums or other cytotoxics
(Please refer to the appendix)
Now, additional expected mode of action identified

Expected to all patients:

1. Inhibit tumor cell migration (←CBP501 can work alone) ➡ Suppress metastasis

Expected to "non-inflamed" patients

1. Inhibition of M2 (=pro-tumor) macrophages (←CBP501 can work alone) ➡ Suppress metastasis

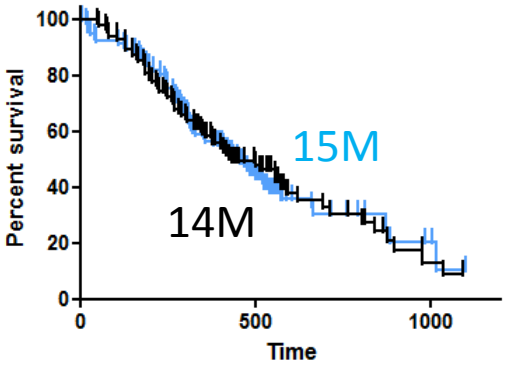
Anticipated in "inflamed" patients

1. Inhibition of M1 (=anti-tumor) macrophages (←CBP501 can work alone) ➡ Tumor growth & metastasis
2. Increase neutrophil extracellular traps (NETs) (←CBP501 can work alone) ➡ Thrombosis & metastasis

➡ = "would/could lead to"

Results of CBP08-02 NSCLC study & a subgroup analysis on OS

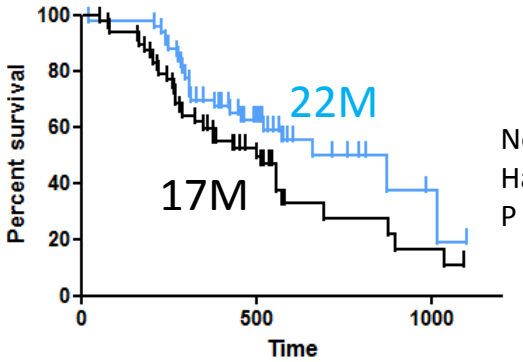
OS of all treated population



Number of patients: 192
Hazard ratio: 1.0

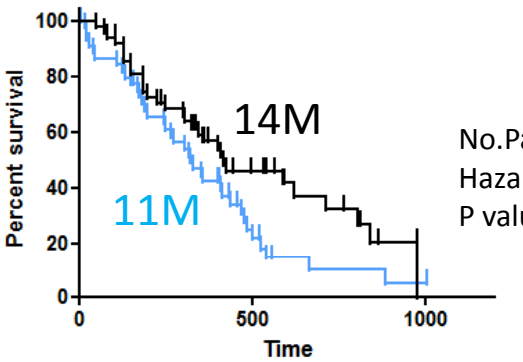
Non-inflamed vs inflamed patients (intergroup comparison)

Non-inflamed



No.Patients 96
Hazard ratio 0.6
P value 0.08

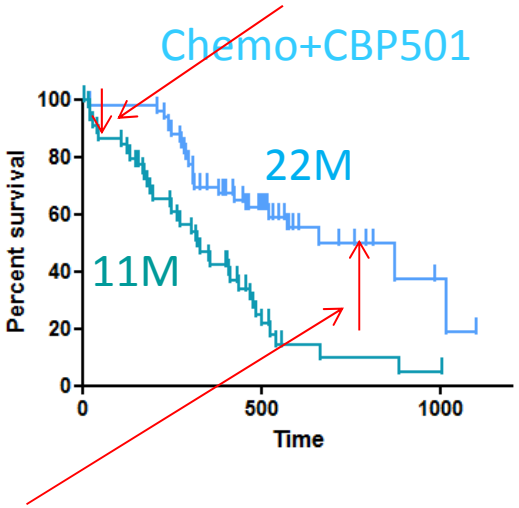
inflamed



No.Patients 96
Hazard ratio 1.6
P value 0.47

Non-inflamed vs inflamed at screening
(intragroup comparison)

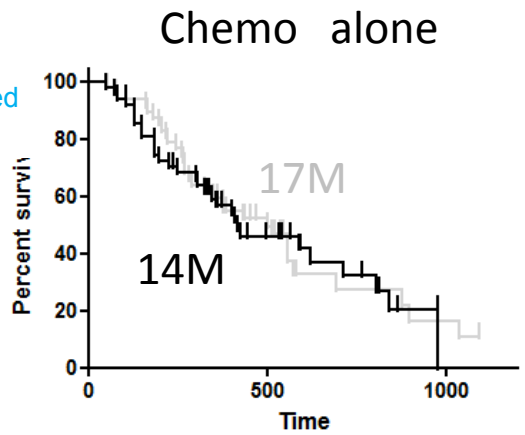
Point 1: Inhibition of M1 macrophage in inflamed



Point 2: Inhibition of M2 macrophage in non-inflamed

Chemo+CBP501 non-inflamed
Chemo+CBP501 inflamed

No.Patients 96
Hazard ratio 0.3
P value <0.0001



Chemo_ non-inflamed
Chemo_ inflamed

No. Patients 96
Hazard ratio 0.9
P value 0.8

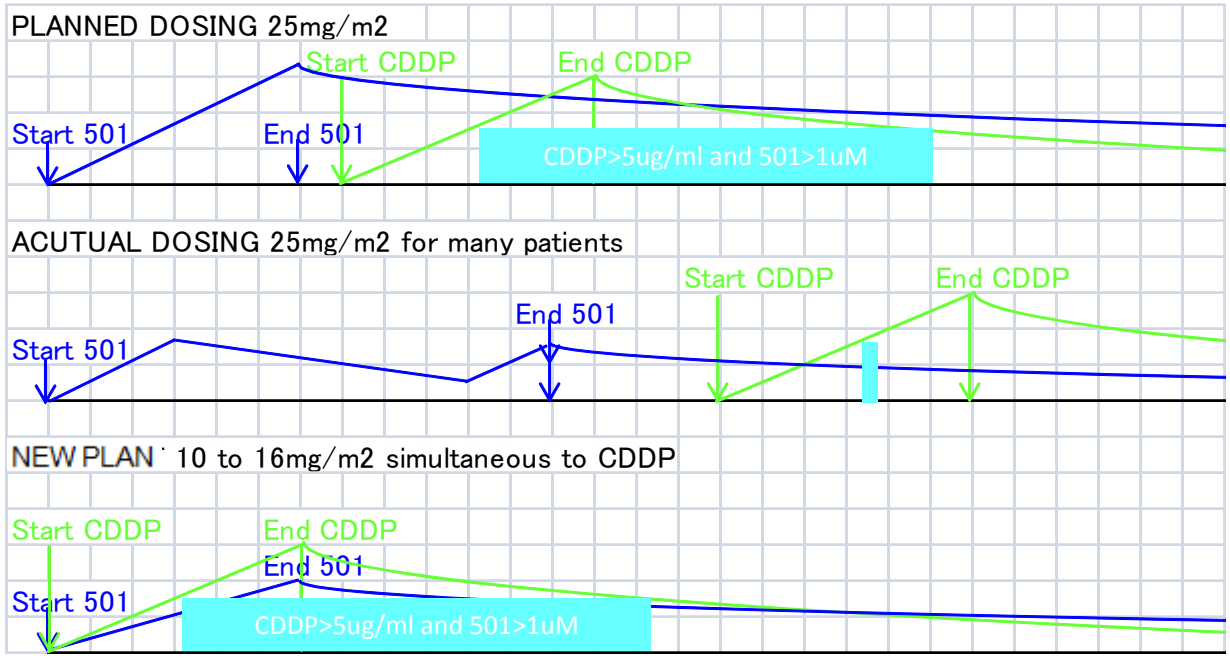
CanBas
Cancer therapy by Basic research

Reconsideration of infusion schedule and dosage

Only 30% of patients in the experimental arms of the two Ph-II trials received study drugs within "+ 30 minutes" of scheduled infusion duration ('1:00+30=1:30' for CBP501 and '1:10+30=1:40' for CBP501 Cmax to cisplatin Cmax), presumably due to infusion reaction.

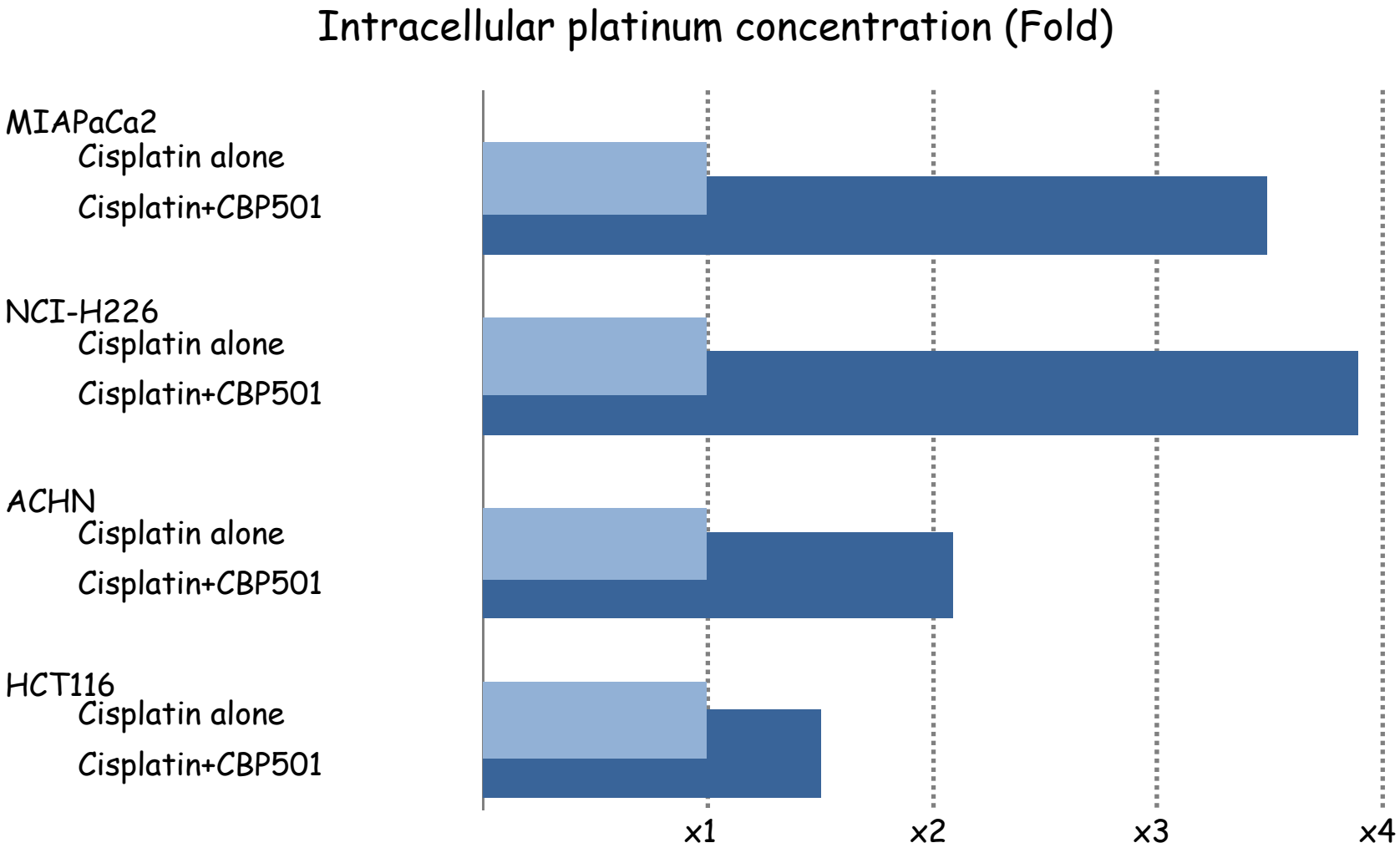
Compliance and efficacy should be improved
by changing infusion schedule and dosage of CBP501

Current plan: CBP501 (25mg/m^2) 1hr ⇒Pemetrexed 10min ⇒CDDP 1hr
New plan: Pemetrexed 10min ⇒ [CBP501 (10-16mg/m^2) + CDDP] 1hr



CBP501 1st MoA - Calmodurin Modulation (1)

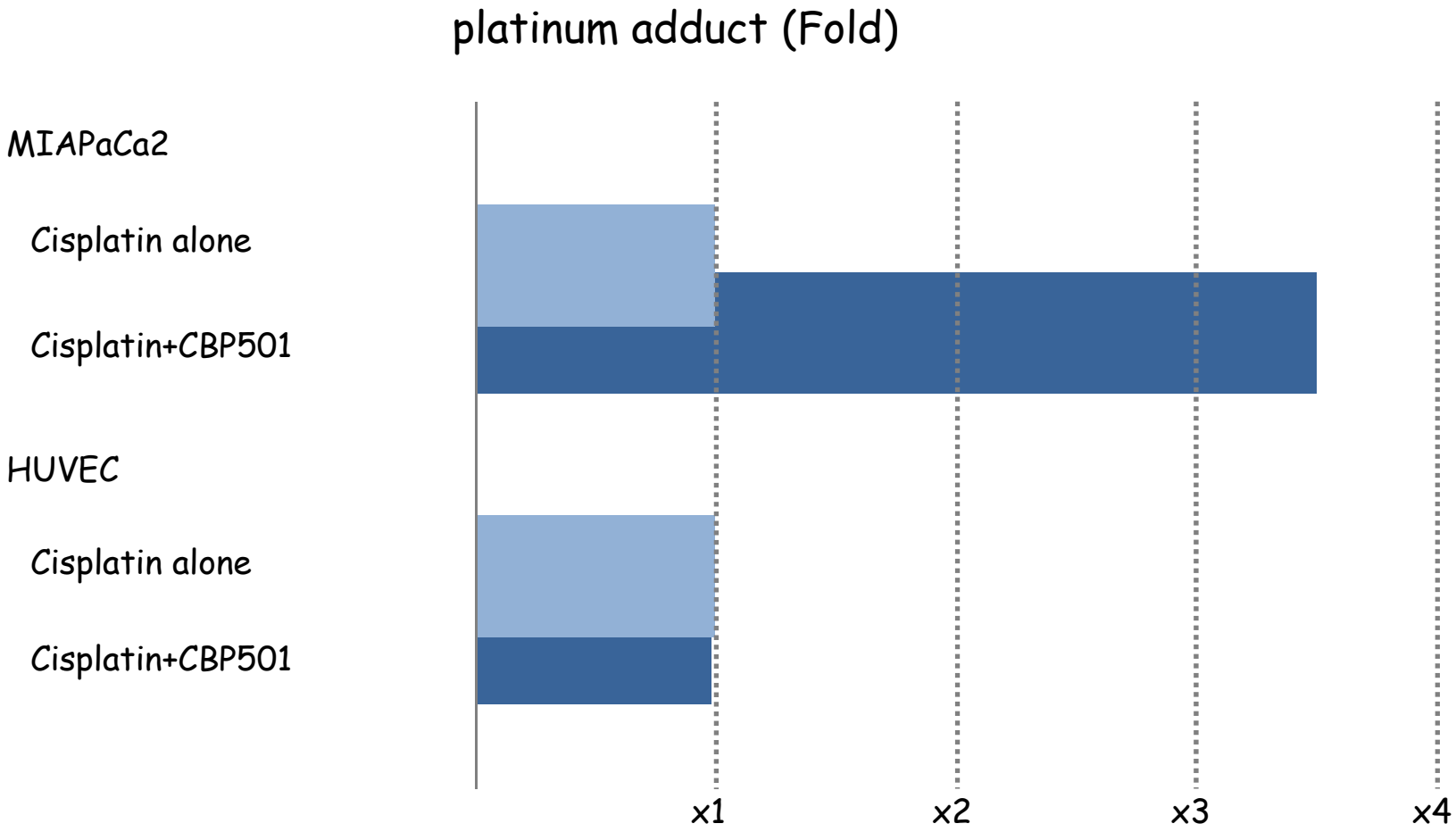
Increases intracellular platinum concentration



< Appendix >

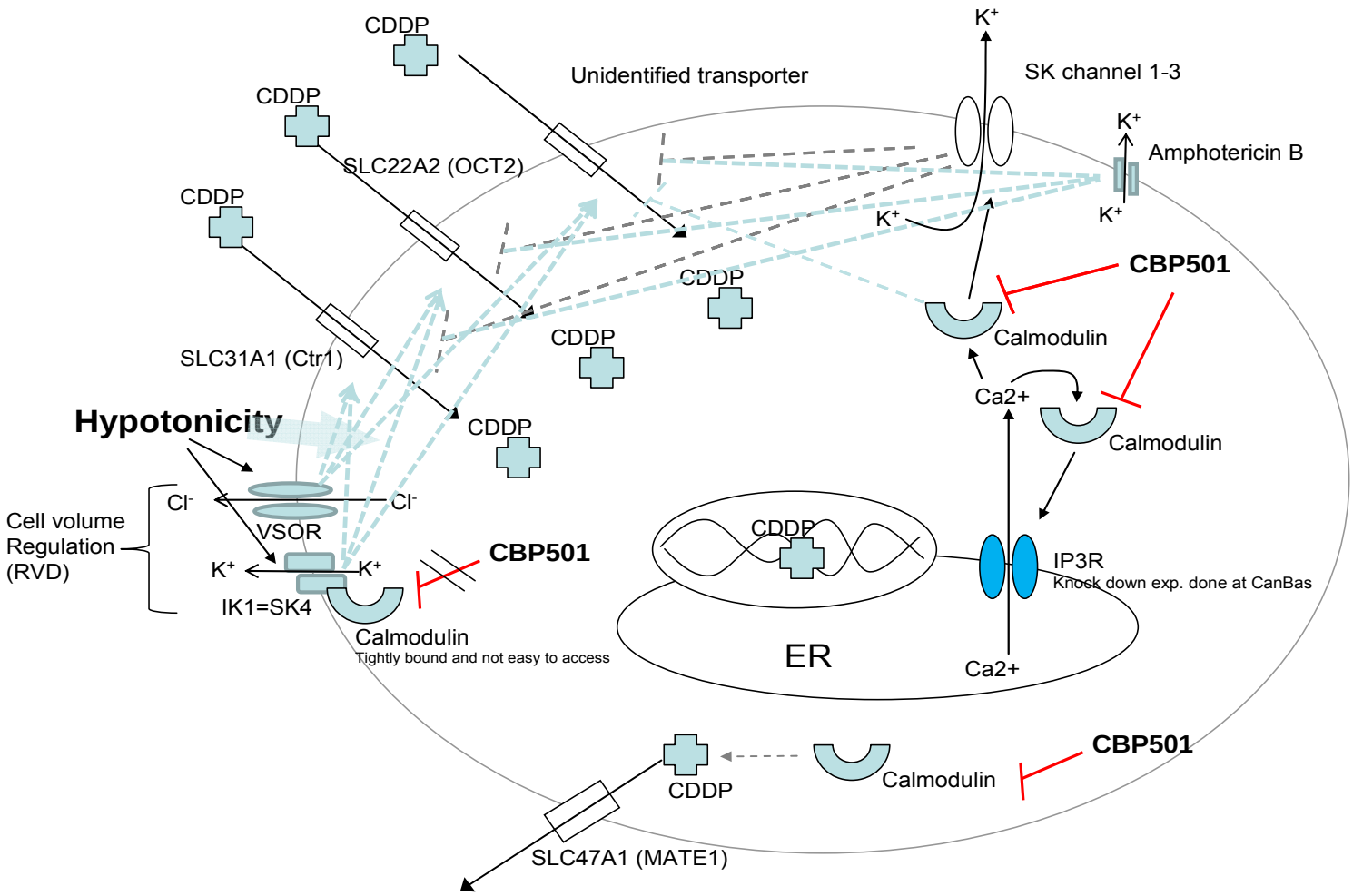
CBP501 1st MoA - Calmodurin Modulation (2)

Increases platinum adduct formation in tumor cells



CBP501 1st MoA - Calmodurin Modulation (3)

Working hypothesis on how CBP501 increases the platinum concentration in cancerous cell



CBS9106

- CRM1 inhibitor

  which revives multiple tumor suppressors in tumor cells
- IND/IMPD ready
- NCE (MW: 420, synthetic small molecule)
- Cancer-cell-specific cytotoxicity demonstrated *in vitro & vivo*
  - Identified and optimized through CanBas' proprietary cell cycle phenotypic screening method that yielded CBP501
  - Acts by inhibition and destabilization of CRM1
    (Ref: *Blood*, 2011)
  - Multiple xenograft models showed tumor growth suppression and prolonged survival without significant adverse effects
- A “hot” MoA (*e.g.* KPT-330, Phase I completed, ASCO2013)
- Ready for Phase I clinical trial