OncoGenex Pharmaceuticals, Inc.
(NASDAQ: OGXI)

OncoGenex ASCO Reception: Key Opinion Leader Panel

Committed to the development and commercialization of new cancer therapies that address treatment resistance in cancer patients.

Forward-Looking Statements

This presentation contains forward-looking statements, including statements concerning anticipated clinical development activities, the potential benefits of product candidates and anticipated market opportunities. All statements other than statements of historical fact are statements that could be deemed forward-looking statements. These statements are based on management's current expectations and beliefs and are subject to a number of risks, uncertainties and assumptions that could cause actual results to differ materially from those described in the forward-looking statements. These risks and uncertainties include, among others, the possibility that interim clinical trial results will not be maintained or will become less substantial as patient survival follow up continues, risks that clinical trials will not be successful or confirm earlier clinical trial results, risks associated with obtaining funding from third parties, risks related to the timing and costs of clinical trials and the receipt of regulatory approvals, and the risk factors set forth in the company's filings with the Securities and Exchange Commission, including its Annual Report on Form 10-K for fiscal year 2008. The company undertakes no obligation to update the forward-looking statements contained herein or to reflect events or circumstances occurring after the date hereof.

Key Opinion Leader Panel & Agenda

Oliver Sartor, M.D.
Piltz Professor of Cancer Research
Depts. of Medicine and Urology
Tulane Medical School

Tomasz Beer, M.D.
Grover C. Bagby Endowed Chair for Prostate Cancer Research
Associate Professor of Medicine
Oregon Health & Science University

Martin Gleave, M.D.
Distinguished Professor , Department of Urologic Sciences, University of British Columbia
Director of the Vancouver Prostate Center
Director of Research for the Department of Urologic Sciences at UBC
Chair of the Genito-Urinary Tumour Group
Chief Scientific Officer, OncoGenex Pharmaceuticals, Inc.

Kim Chi, MD, FRCPC
Medical Oncologist,
BC Cancer Agency
Principal Investigator of Randomized OGX-011 CRPC Phase 2 Study

Brent Blumenstein, Ph.D.
Independent Statistician
Trial Architecture Consulting

Agenda

1. Treatment Strategies in Prostate Cancer
   (Dr. Oliver Sartor)
2. Clinical Trial Endpoints in Prostate Cancer
   (Dr. Tomasz Beer)
3. Clusterin as a Therapeutic Target
   (Dr. Martin Gleave)
4. Randomized Phase 2 Study Evaluating OGX-011 as 1st Line Therapy in CRPC
   (Dr. Kim Chi)
5. Additional Analysis of OGX-011 Phase 2 Data
   (Dr. Brent Blumenstein)
6. OGX-011 Phase 3 and Regulatory Plans
   (Scott Cormack)

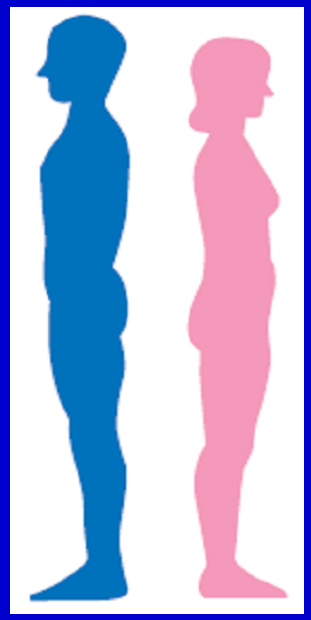
Prostate Cancer Overview

Oliver Sartor, MD

Piltz Professor of Cancer Research
Departments of Medicine and Urology
Tulane Medical School
New Orleans, Louisiana

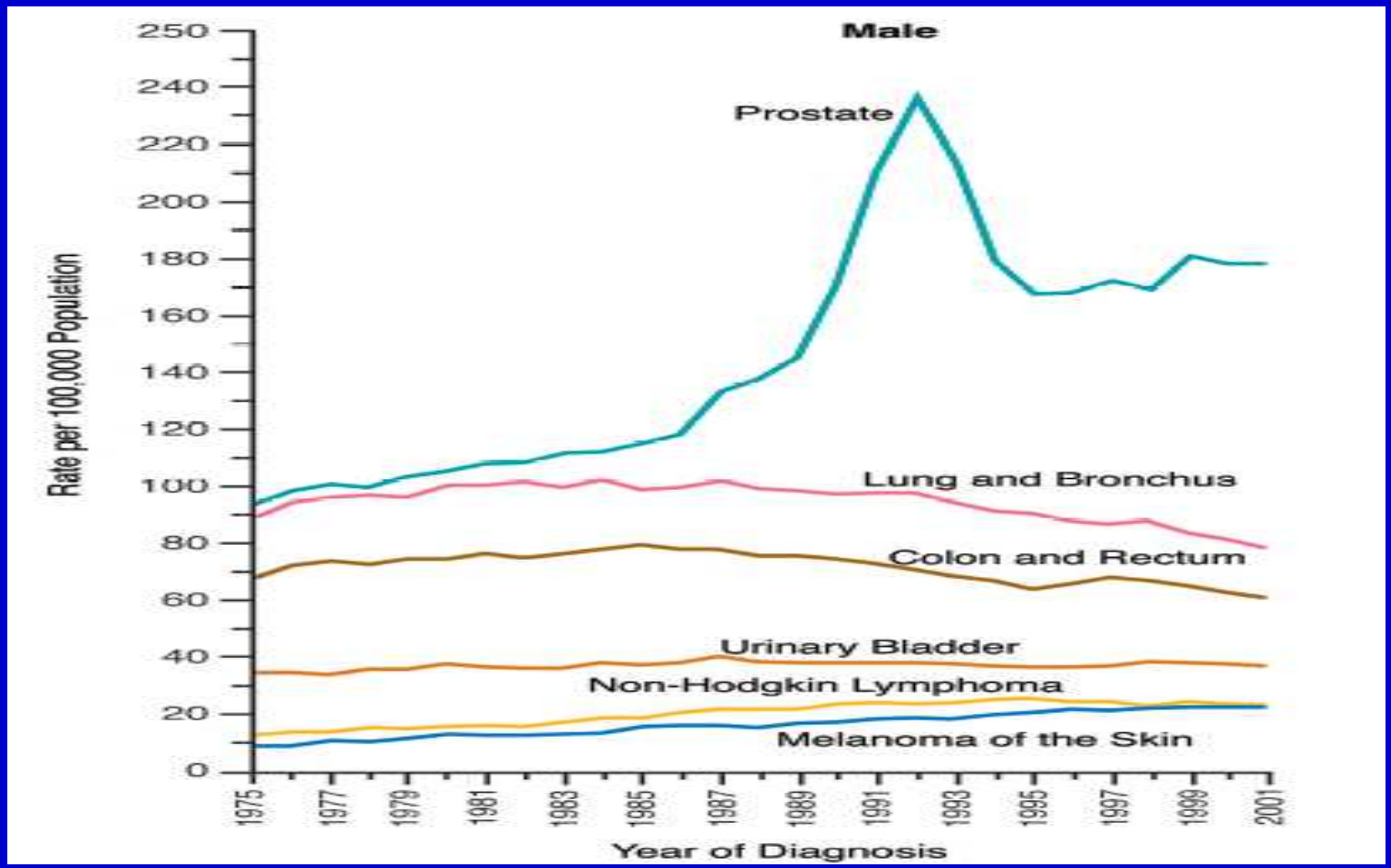
Prostate Cancer Currently Represents 25% of All Cancers Diagnosed in US Men

Estimated New Cases

Males				Females		
Prostate	186,320	25%		Breast	182,460	26%
Lung & bronchus	114,690	15%		Lung & bronchus	100,330	14%
Colon & rectum	77,250	10%		Colon & rectum	71,560	10%
Urinary bladder	51,230	7%		Uterine corpus	40,100	6%
Non-Hodgkin lymphoma	35,450	5%		Non-Hodgkin lymphoma	30,670	4%
Melanoma of the skin	34,950	5%		Thyroid	28,410	4%
Kidney & renal pelvis	33,130	4%		Melanoma of the skin	27,530	4%
Oral Cavity & pharynx	25,310	3%		Ovary	21,560	3%
Leukemia	25,180	3%		Kidney & renal pelvis	21,260	3%
Pancreas	18,770	3%		Leukemia	19,090	3%
All Sites	**745,180**	**100%**		**All Sites**	**692,000**	**100%**

American Cancer Society. *Cancer Facts and Figures 2008.* Atlanta: American Cancer Society, 2008.

Clinical Incidence of Prostate Cancer in the United States Has Changed Dramatically Over Time



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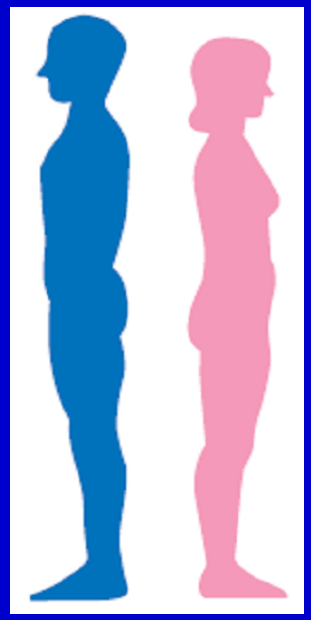
Prostate Cancer is the Second Leading Cause of Male Cancer Death

Estimated Deaths

Males				Females		
Lung & bronchus	90,810	31%		Lung & bronchus	71,030	26%
Prostate	28,660	10%		Breast	40,480	15%
Colon & rectum	24,260	8%		Colon & rectum	25,700	9%
Pancreas	17,500	6%		Pancreas	16,790	6%
Liver & intrahepatic bile duct	12,570	4%		Ovary	15,2520	6%
Leukemia	12,460	4%		Non-hodgkin's lymphoma	9,370	3%
Esophagus	11,250	4%		Leukemia	9,250	3%
Urinary bladder	9,950	3%		Uterine corpus	7,470	3%
Non-Hodgkin lymphoma	9,790	3%		Liver & intrahepatic bile duct	5,840	2%
Kidney & renal pelvis	8,100	3%		Brain & other nervous system	5,650	2%
All Sites	**294,120**	**100%**		**All Sites**	**271,530**	**100%**

American Cancer Society. *Cancer Facts and Figures 2008.* Atlanta: American Cancer Society, 2008.

Natural History of Prostate Cancer: A Heterogeneous Disease

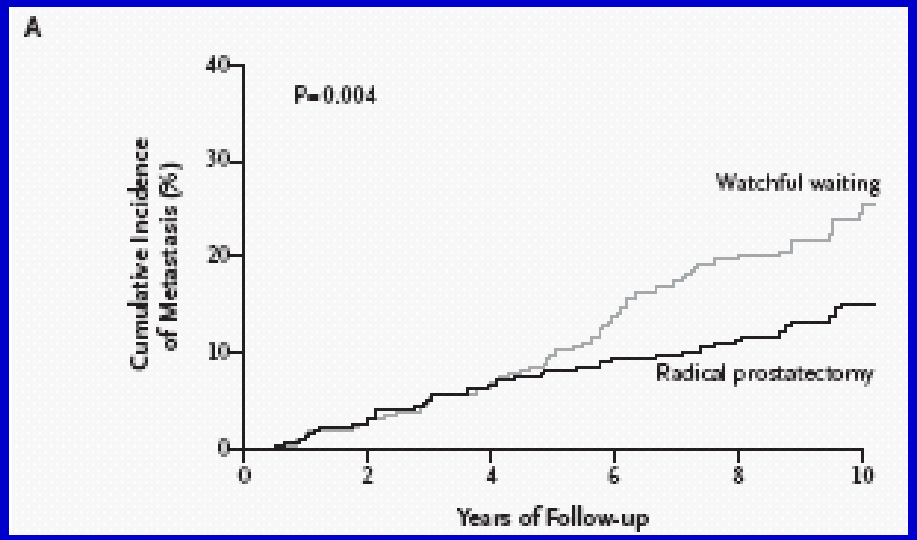
- Few cancers have such heterogeneity in their natural history
  - Not all prostate cancers are created equal
- Initial prognosis is driven by *Stage, Gleason, and PSA*
- Treatments are driven by prognosis but judgments occur regarding *Age and Co-morbidities* of the patient

Initial Treatment Varies by Stage

- Localized Prostate Cancer
  - Surveillance, Surgery, Radiation (External or Seeds)
- Locally Advanced
  - External Beam Radiation + Androgen Deprivation
- Metastatic
  - Androgen Deprivation

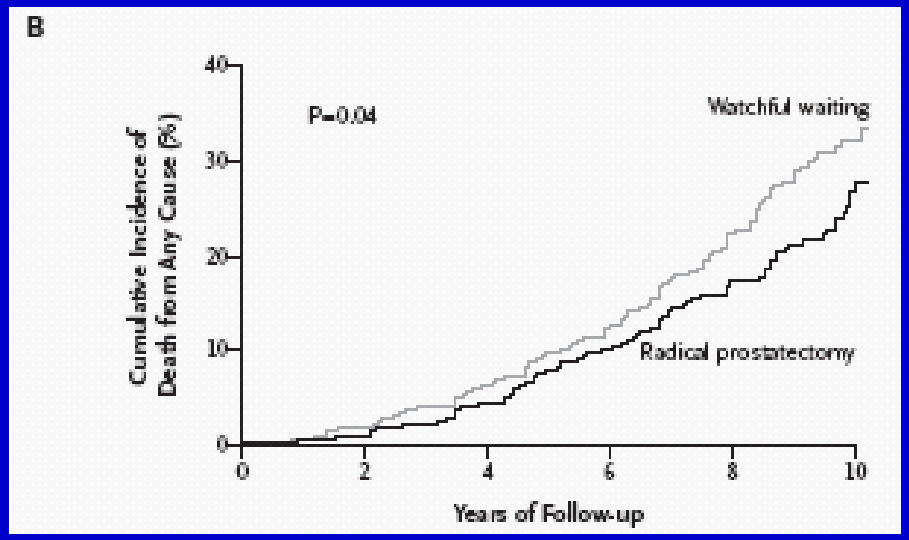
Randomized Trials of Surgery vs. Observation Demonstrate Advantages for Surgery in Localized Disease, But Many Fail

Cumulative Incidence of Distant Metastasis



No. at Risk						
Radical prostatecomy	347	333	306	254	181	87
Watchful waiting	348	332	310	243	156	73

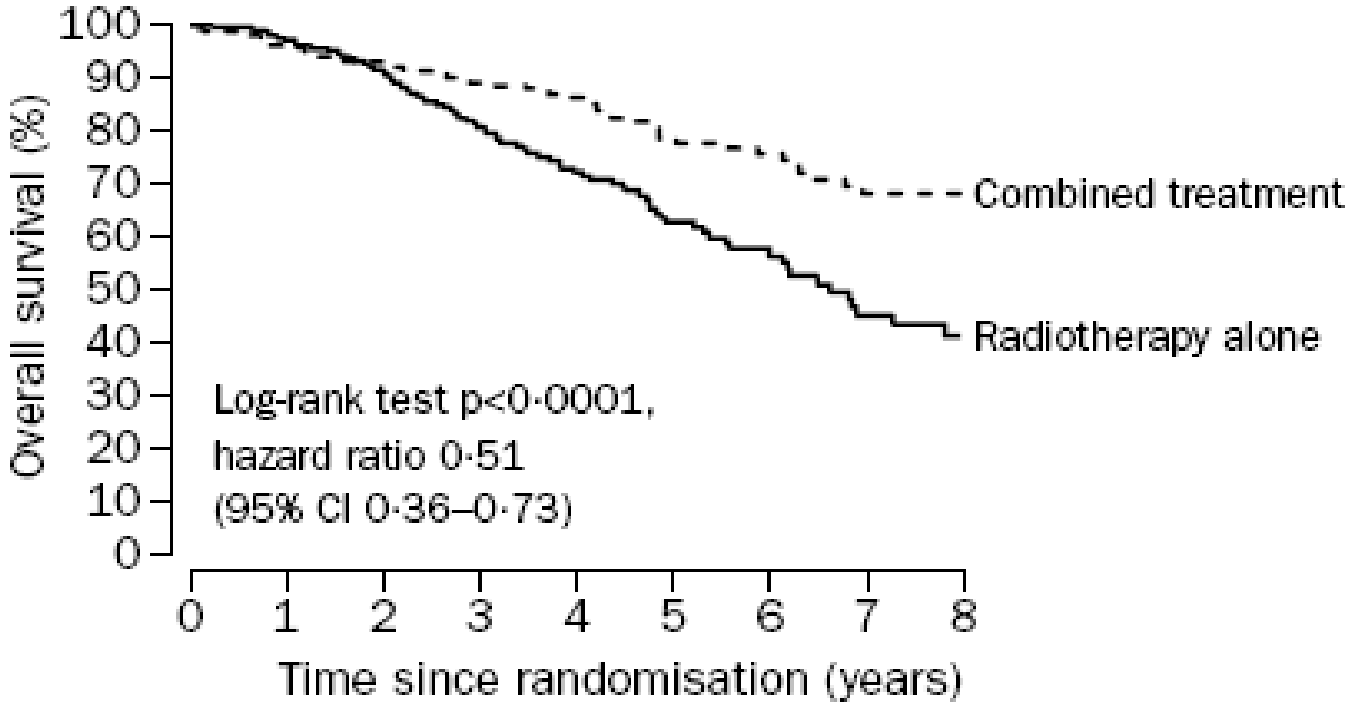
Cumulative Incidence of Death from Any Cause



No. at Risk						
Radical prostatecomy	347	343	332	284	210	118
Watchful waiting	348	341	326	279	198	104

Bill-Axelson A, et al. *N Engl J Med.* 2005;352:1977-1984

Radiation + Androgen Deprivation Therapy (ADT) Saves Lives in Locally Advanced Prostate Cancer, But Many Fail



Bolla et al. Lancet 360:103-108, 2002

What to Do After Surgery or Radiation Fail?

- Failures after Surgery
  - Surveillance, salvage radiation, ADT
- Failures after Radiation
  - Surveillance, ADT
- Together ADT is the common pathway for both radiation and surgery failures
  - But ADT is not curative; what do we do with ADT failures?
  - What is the natural history of these patients?

ADT Failures: The Face of Change

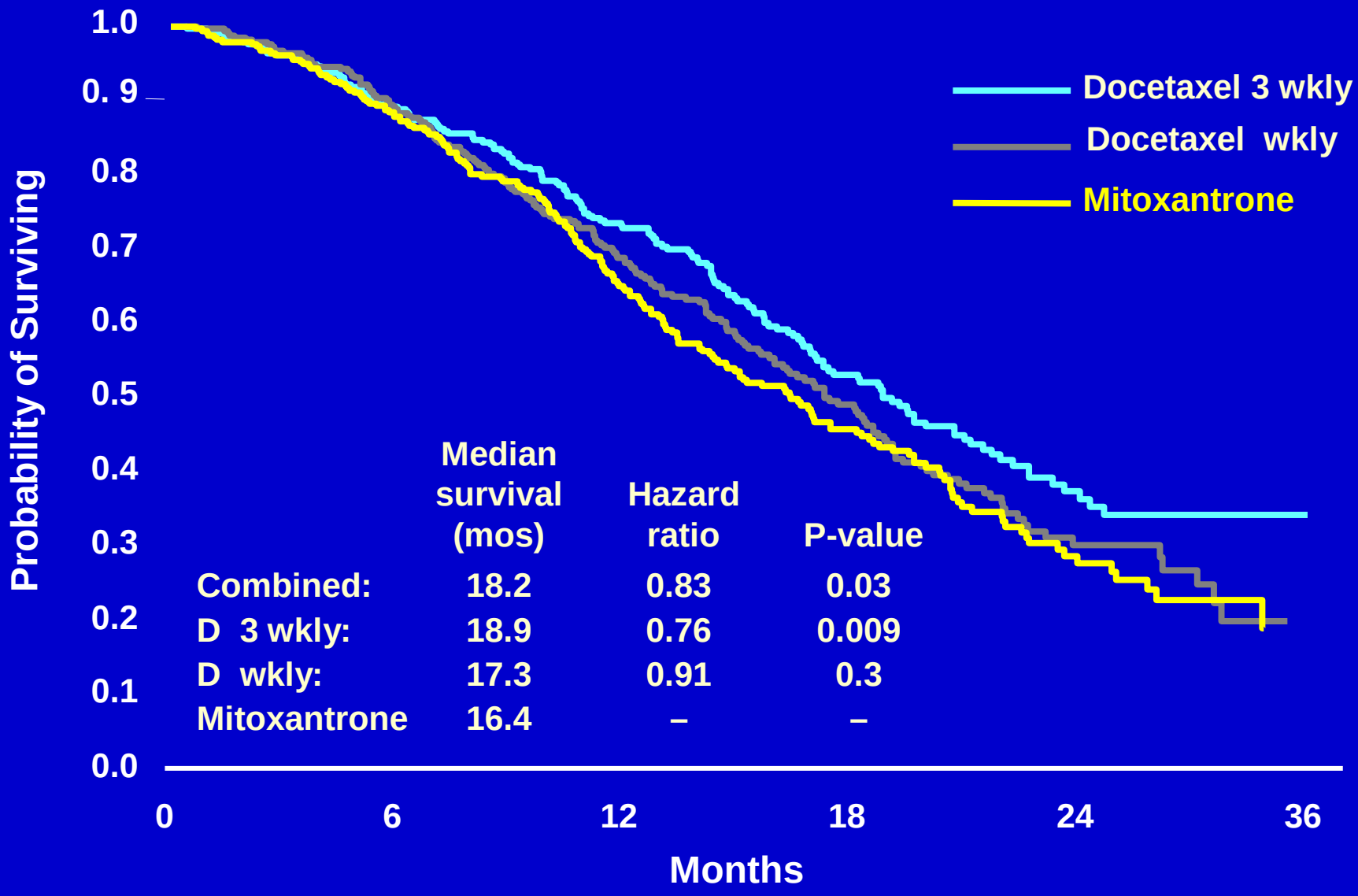
- Many changes have occurred in our understanding of this disease
  - Pathophysiology
    - The evolution in terminology from “hormone-refractory” and “androgen-independent”, to “castrate-refractory”
  - Natural history
    - Tremendous changes in our understanding of natural history, in significant part attributable to PSA testing
  - Therapeutic options
    - Multiple new paradigms on the rise

Current Progression and Survival of PSA Rising but "Non-Metastatic"CRPC

- PSA rise post-ADT to occurrence of metastatic disease: 22-30 months
  - Smith et al. JCO 23:2918, 2005 and Nelson et al. ASCO 2007, Abstract 5018
- Time from post-ADT PSA rise to death: 40-68 months
  - Oefelin et al. J Urol. 171:1525, 2004 , Nelson et al. ASCO 2007, Abstract 5018, and Sartor et al. Cancer 112:2393, 2008

Overall Survival in Metastatic CRPC

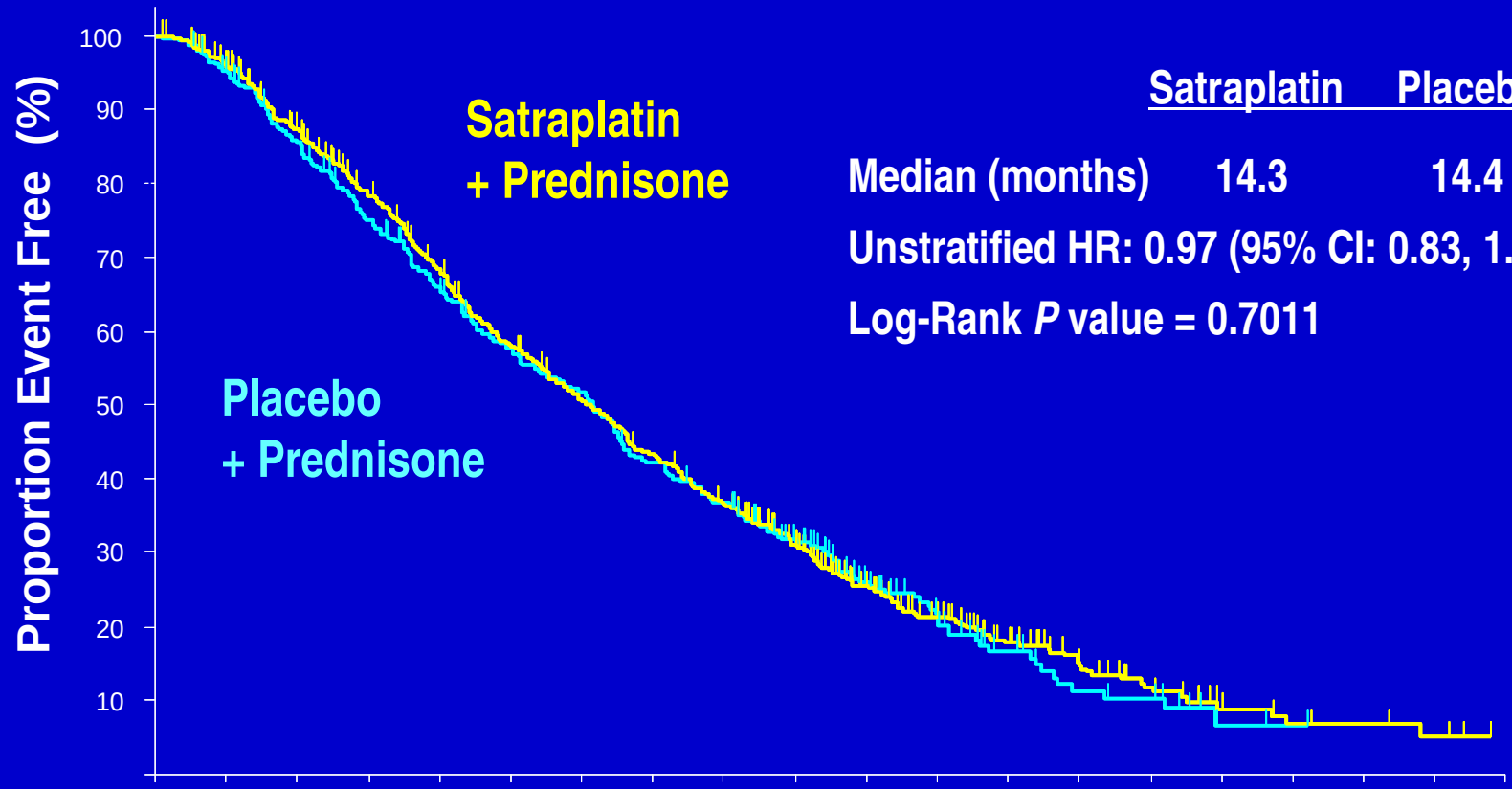
Tannock et al. N Engl J Med 2004:351;1502-1512



	Median survival (mos)	Hazard ratio	P-value
Combined:	18.2	0.83	0.03
D 3 wkly:	18.9	0.76	0.009
D wkly:	17.3	0.91	0.3
Mitoxantrone	16.4	–	–

Survival Curves for Second-Line Chemotherapy in Metastatic CRPC

Sartor et al, ASCO 2008



	Satraplatin	Placebo
Median (months)	14.3	14.4

Unstratified HR: 0.97 (95% CI: 0.83, 1.13)

Log-Rank *P* value = 0.7011

Satraplatin	635	528	398	288	208	103	50	20	6
Placebo	315	262	194	150	105	53	21	10	1

Therapeutic Options for CRPC Today

- Secondary Hormonal Manipulations
  - Antiandrogen withdrawal, antiandrogen administration, adrenal suppressives (ketoconazole), corticosteroids (prednisone, dexamethasone, etc.), estrogens (DES, etc.)
- External Beam Radiation Therapy for Palliation
- Intravenous Bone-seeking Radioisotopes for Palliation
  - Samarium-153 EDTMP, Strontium-89 (FDA approvals)
- Bisphosphonates
  - Zoledronate (FDA approval)
- Chemotherapy
  - Mitoxantrone, docetaxel, estramustine (FDA approvals)
- Experimental Therapies

Endpoints in Prostate Cancer

Tomasz M. Beer, M.D.

Knight Cancer Institute

at Oregon Health & Science University

Goals of Therapy in Advanced Prostate Cancer

- Control, relieve, or eliminate disease manifestations that are present
- Prevent or delay disease manifestations expected to occur
- Disease manifestations
  - Death
  - Symptoms and complications
    - Pain
    - Skeletal complications
  - Radiographic evidence of disease
  - Serum PSA

Overall Survival

- Advantages
  - Definitive measure of clinical benefit
  - Unambiguous ascertainment
- Disadvantages
  - Prolonged time to event
    - Especially in earlier stages of disease
  - Large, long studies necessary to demonstrate
  - Susceptible to dilution by subsequent therapy
  - Drives drug development to very late stage disease

Progression-free Survival

Advantages

- Ascertained early
- Not susceptible to dilution by subsequent therapy
  - Important in earlier stages of disease
- Study designs with cross-over possible
  - Enhanced accrual

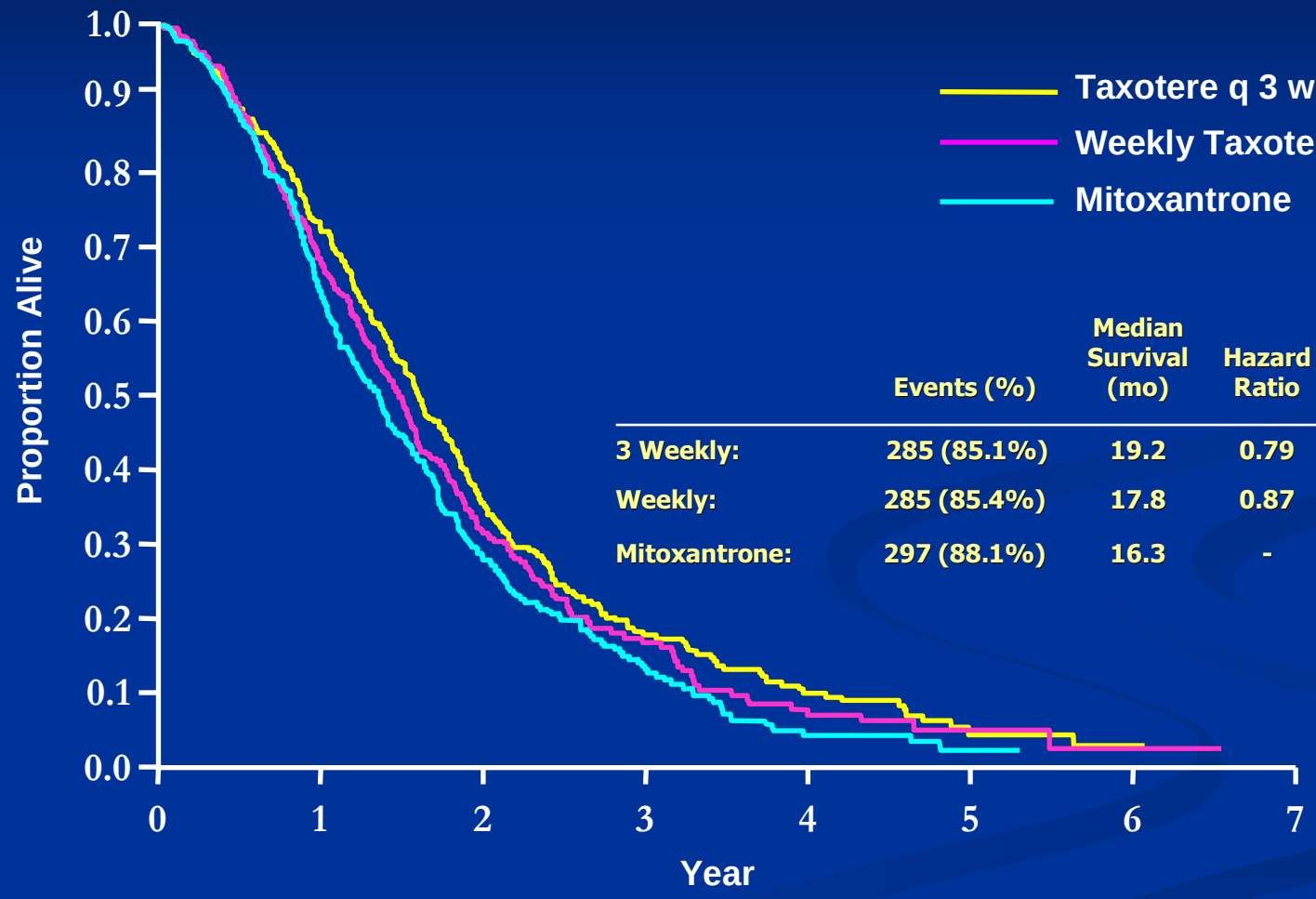
Disadvantages

- Ascertained early
  - May miss treatment effect
- Complex design
  - Broad range of disease manifestations
    - Radiographic disease in bone and soft tissue, symptoms, PSA, clinical decline
  - Not uniformly defined or recognized by the FDA as a measure of clinical benefit
- Susceptible to biases related to frequency and completeness of ascertainment
- Requires frequent, comprehensive monitoring
- Loosely correlated with overall survival

Serum PSA decline

- Advantages
  - Inexpensively and easily ascertained
  - Frequently utilized by patient and physicians
  - Correlated with overall survival
- Disadvantages
  - Not a direct measure of clinical benefit
  - Not an accepted surrogate for overall survival
  - Not acceptable for drug approval
  - May not equally reflect burden of disease with different classes of drugs

TAX 327 Long-Term Overall Survival

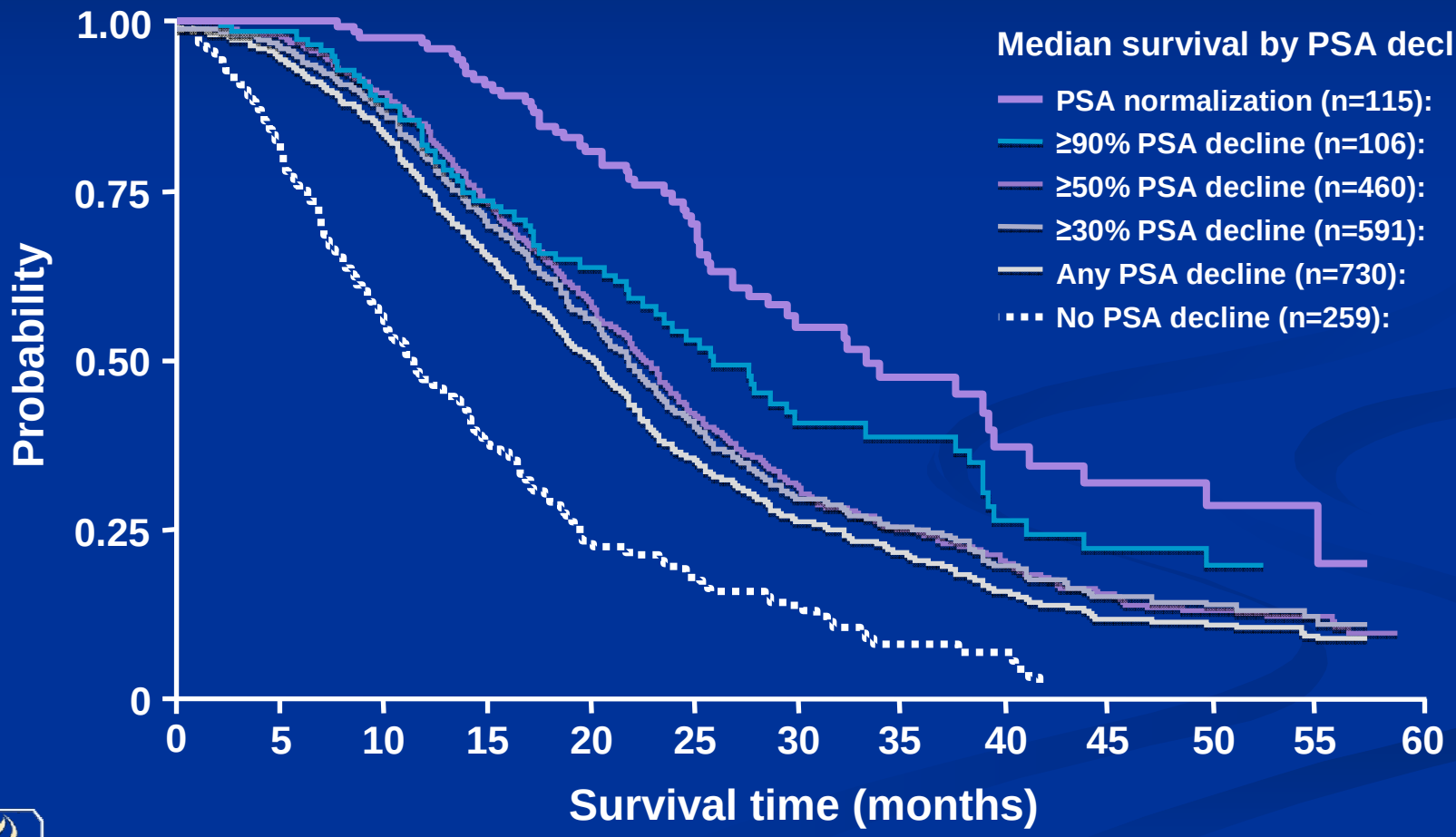


	Events (%)	Median Survival (mo)	Hazard Ratio	*P*-Value
3 Weekly:	285 (85.1%)	19.2	0.79	.004
Weekly:	285 (85.4%)	17.8	0.87	.09
Mitoxantrone:	297 (88.1%)	16.3	-	-

Knight Cancer Institute
at Oregon Health & Science University

Berthold DR, et al. J Clin Oncol 2007;25 (18S Part I of II):236s (abstract and oral presentation 5005).

Survival by PSA Decline



Median survival by PSA decline

PSA normalization (n=115):	33.3 months
≥90% PSA decline (n=106):	26.6 months
≥50% PSA decline (n=460):	22.4 months
≥30% PSA decline (n=591):	21.6 months
Any PSA decline (n=730):	20.7 months
No PSA decline (n=259):	11.7 months

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at Oregon Health & Science University

Armstrong AJ, JCO 2007;25:3965

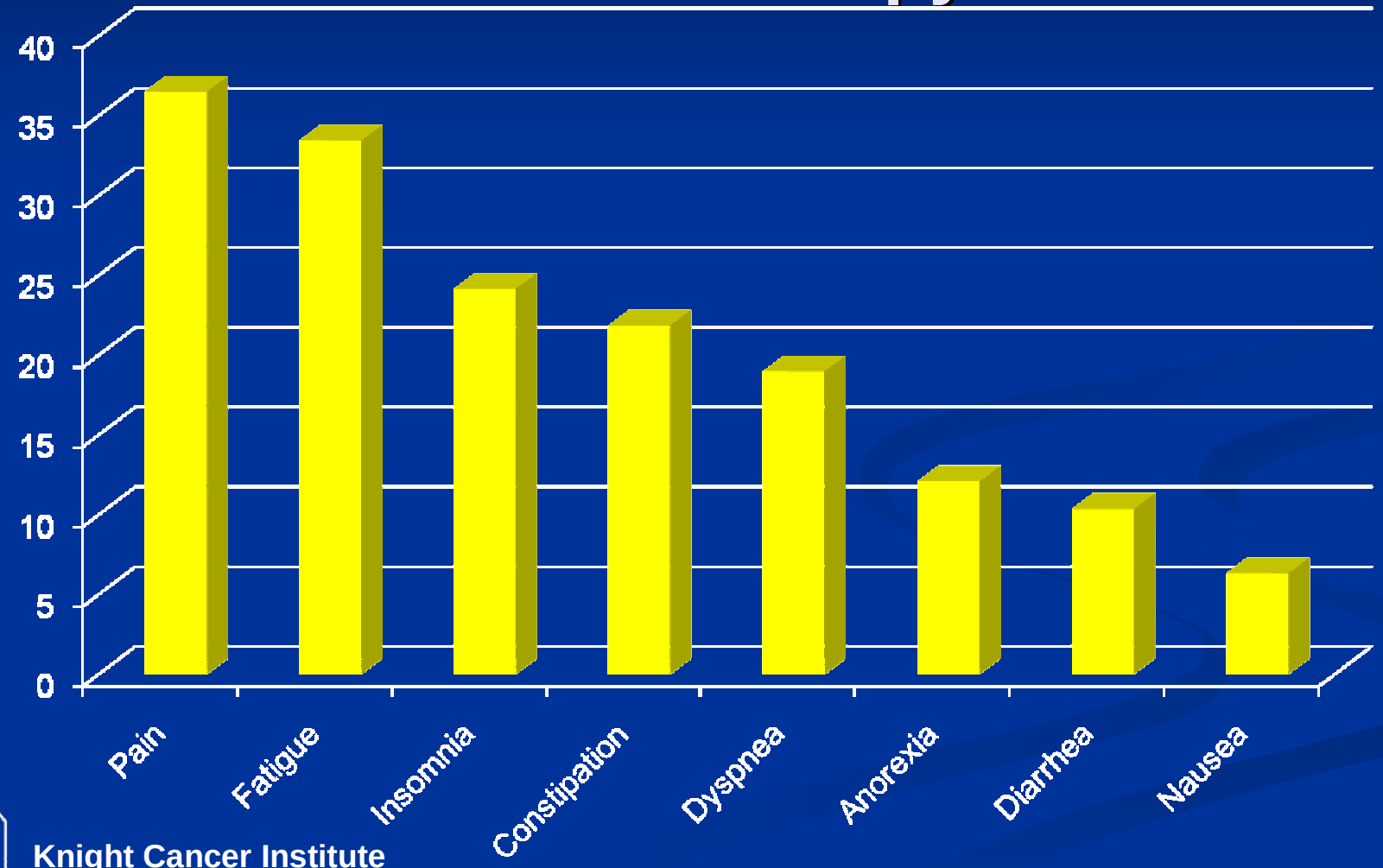
Skeletal-related Event-free Survival

- Advantages
  - A measure of clinical benefit
    - Delay of a recognized complication of prostate cancer
  - Recognized by the FDA for drug approval
- Disadvantages
  - Ambiguous ascertainment
    - May include asymptomatic events
  - Focused on a single organ system
  - May not measure anti-neoplastic treatment effects

Symptom Relief

- Advantages
  - Measure of clinical benefit
  - Previously recognized by the FDA for drug approval
  - Ascertained quickly
  - Smaller studies may be sufficient
- Disadvantages
  - Complex ascertainment
  - Meticulous compliance with patient-reported surveys necessary
  - Accounting of potential confounding effects of concomitant treatments necessary
  - Blinded design necessary
  - Design requires that a clinically meaningful benefit is demonstrated, subject to disagreement

QLQ-C30 baseline data from CRPC patients entering docetexel-based chemotherapy



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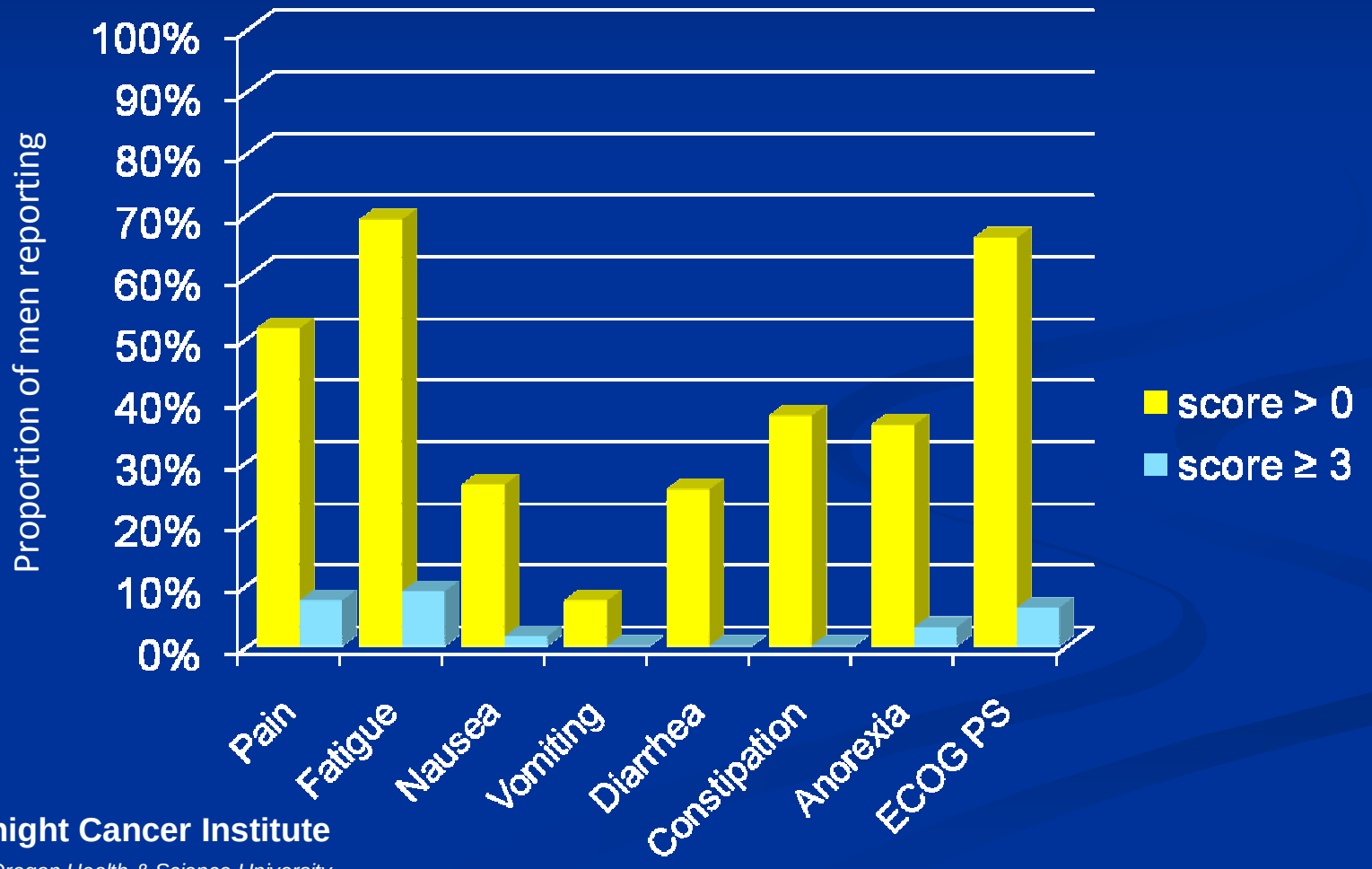
Cancer 2004;100:758–63.

Incidence of Self-reported CTCAE Symptom Grades at Baseline

N = 100 men with metastatic prostate cancer starting chemotherapy



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at Oregon Health & Science University

Ethan Basch, MSKCC

Pain Palliation Endpoint Model

Key Design Elements

- Patients
  - Must have stable baseline pain
- Measures
  - Pain measure must be valid and reliable
  - Narcotic use must be controlled and documented
- Responder definition
  - Pain reduction without increase in analgesic use
  - Based on average of multiple daily measures at time points of interest (e.g., every 12 weeks)
  - Must be supported by related outcomes
    - Clinical, biomarker, radiographic, HRQL, patient global impression of improvement

Basch: GU ASCO, 2009
FDA: PRO Draft Guidance, 2006

Hsp's in Oncogenesis and Treatment Stress

- HS response is a highly conserved adaptive response evolved to safeguard organisms or cells against stress (eg hyperthermia, oxidative stress, and toxins).
- Critical role in Darwinian fitness, adaptation, evolvability, ageing
- Essential chaperoning functions are subverted during oncogenesis to facilitate malignant transformation and rapid somatic evolution
  - biochemical buffers for many genetic lesions within tumors
  - allow mutant proteins to retain or gain function while permitting cancer cells to tolerate imbalanced signaling that oncoproteins create

Background: Clusterin

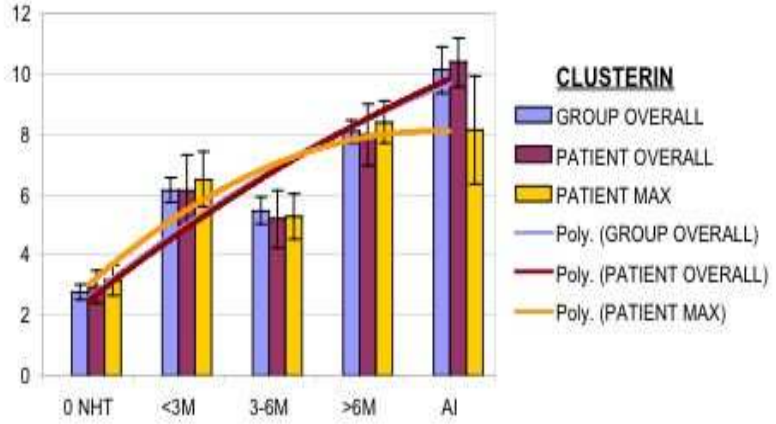
- Heterodimeric glycoprotein highly conserved across species
- Transcriptionally regulated by HSF-1
- Chaperone protein function similar to heat shock proteins
- Secretory and nuclear forms
  - sCLU - Anti-apoptotic
    - Prevents protein aggregation
    - Inhibits activated Bax
    - Increases NF-kB activity through I-kB degradation
  - nCLU - Pro-apoptotic

Michel Biochem J, 1997; Steinberg, Clin Cancer Res, 1997; Humphreys, JBC, 1999; Miyake, Can Res, 2000; Miyake Clin Can Res, 2000; Zhang, Nat Cell Biol, 2005

Background: Clusterin

- Expressed in a number of cancers
- Expression induced by standard anti-cancer therapies
- Prostate Cancer
  - Increased expression correlates with higher Gleason Grade
  - Increases after castration therapy and in CRPC tissues
- Overexpression in pre-clinical models confers resistance to hormone, radiation and chemotherapy

IMAGE PRO-PLUS SCORING



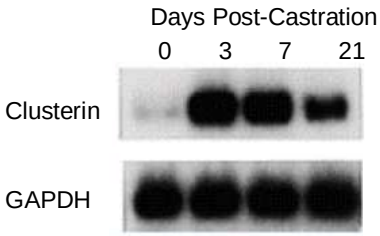
Gleave, Urology, 2002; Zellweger, Clin Can Res, 2002; July, Prostate, 2002; Redondo, Am J Path, 2000; Miyake, Urology, 2000; Parczyk, J Can Res Clin Oncol, 1994; July, Mol Can Thera, 2004; Redondo, Am J Path, 2000

Clusterin – Mechanism of Action

1. Expression is induced by standard anti-cancer therapies

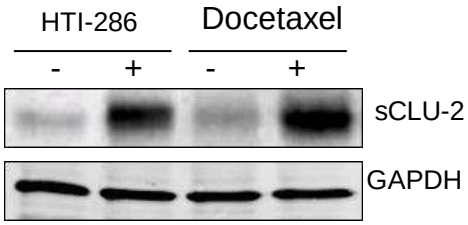
2. Increased expression confers broad spectrum treatment-resistance
   - Chemotherapy
   - Proteasome inhibition
   - Radiation
   - Heat shock
   - Hormone Ablation
   - Etc.

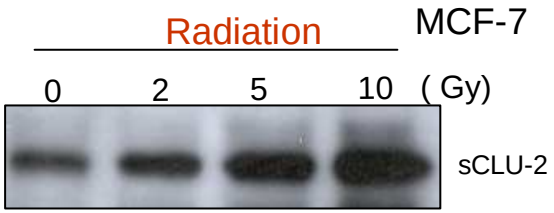
Androgen Ablation
Shionogi Tumors



Cancer Research 60; 170, 2000

Microtubular Inhibitors
PC-3 Cells



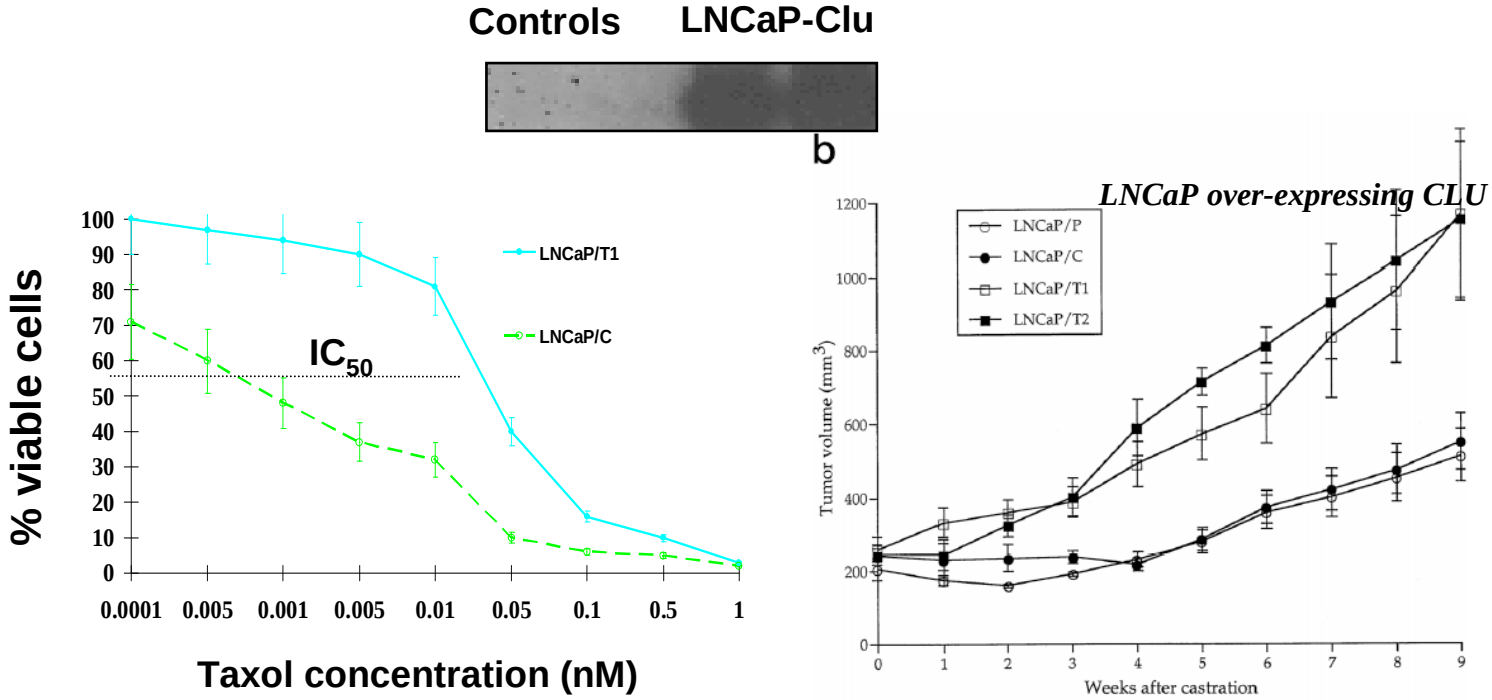


Clusterin - Stress-induced Cytoprotective Chaperone

sCLU Function in Cell Stress and Survival:

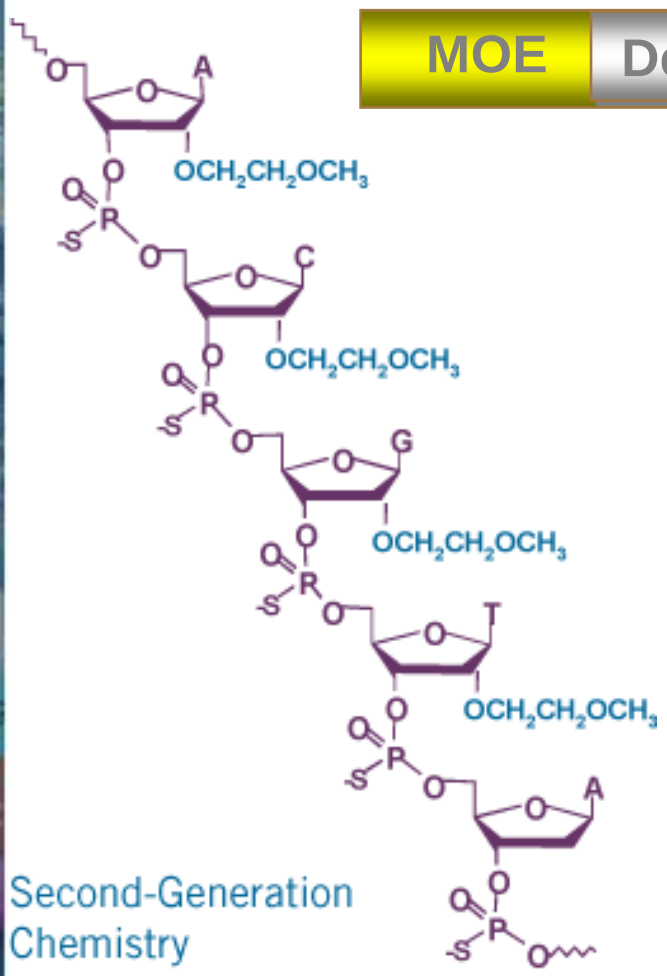
1. Potent inhibitor of protein aggregation under stress conditions
2. Interacts with and inhibits activated Bax (Zhang et al, Nature Cell Biology, 2005)
3. Enhances Ik-B degradation to increase NF-kB transcriptional activity

- **sCLU overexpression confers broad spectrum treatment resistance**



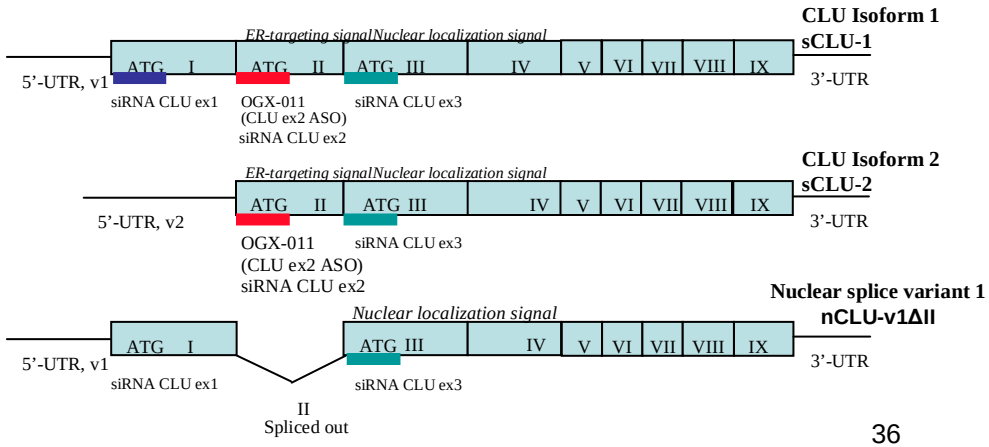
Cancer Research 60;170, 2000; *Cancer Research 60;2547, 2000;* *Clin Can Res 8:3276-84, 2002*

OGX-011 Molecule: 2nd Generation Phosphorothioate MOE Gapmer



Advantages of 2'MOE analogues

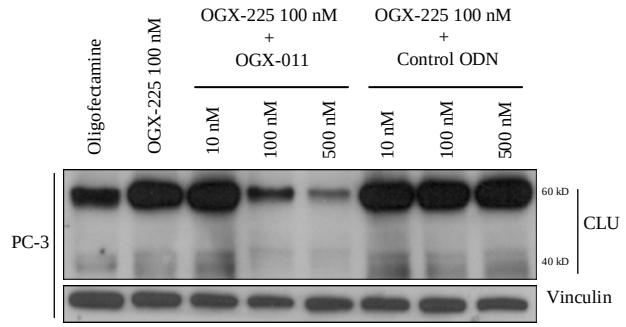
- Increased resistance to enzymatic degradation
- Primate tissue half-life of 7-11 days
- Once-weekly 2-hour infusion
- Higher doses administered
- Improved & favorable safety profile
- Lower cost of goods



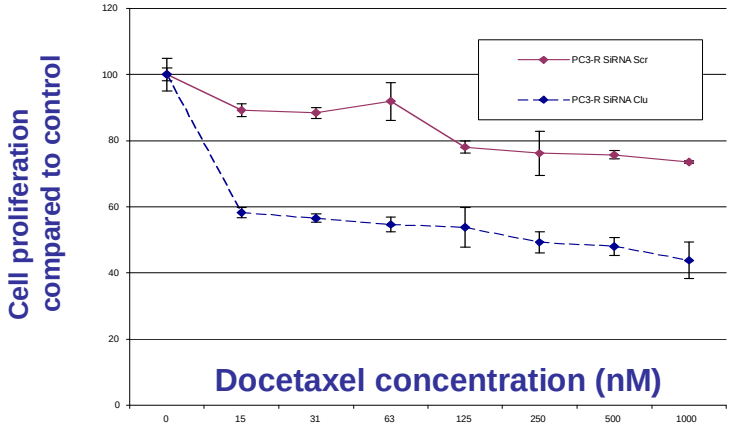
J Pharmacol Exp Ther.; 298(3):934-40, 2001

Clusterin Knockdown Enhances Activity of Chemotherapy in Prostate Cancer Cells

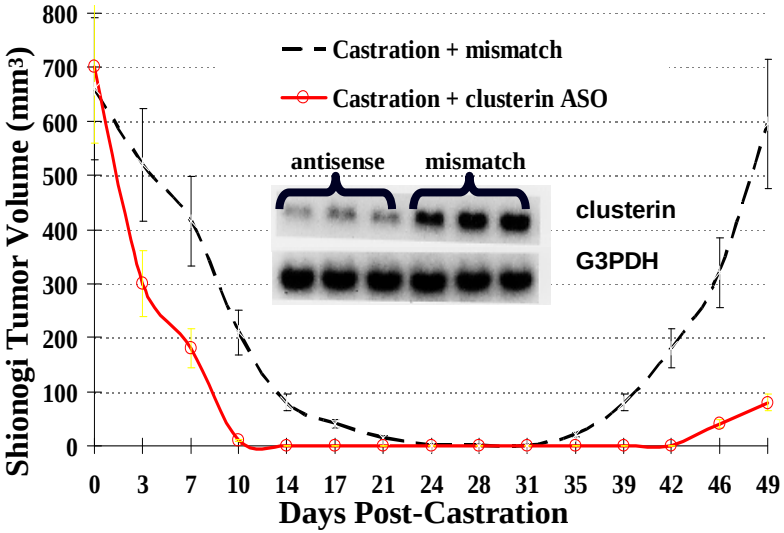
Clu Knockdown by CLU ASO (OGX-011)



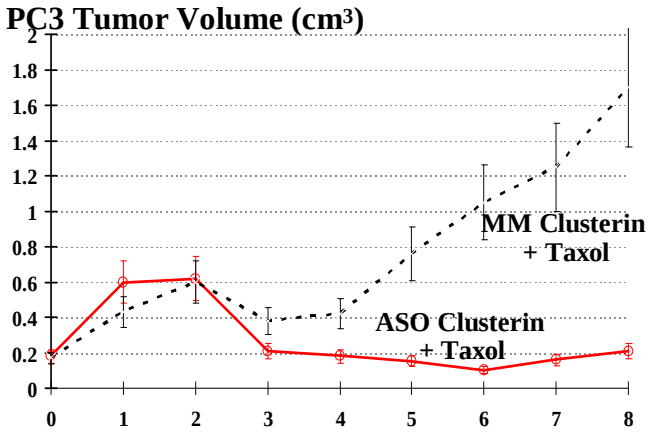
OGX-011 Chemosensitizes PC-3 Cells to Docetaxel



Clusterin ASO Enhance Castration-induced Apoptosis in Shionogi Tumors

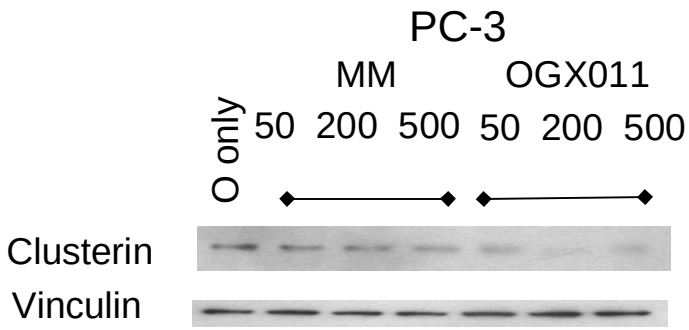


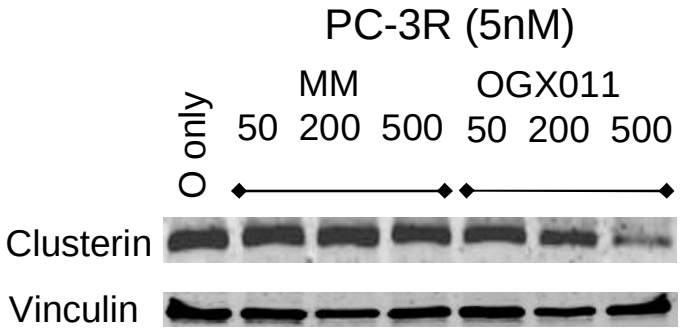
OGX-011 Enhances Taxol Activity in PC3 Tumors in vivo

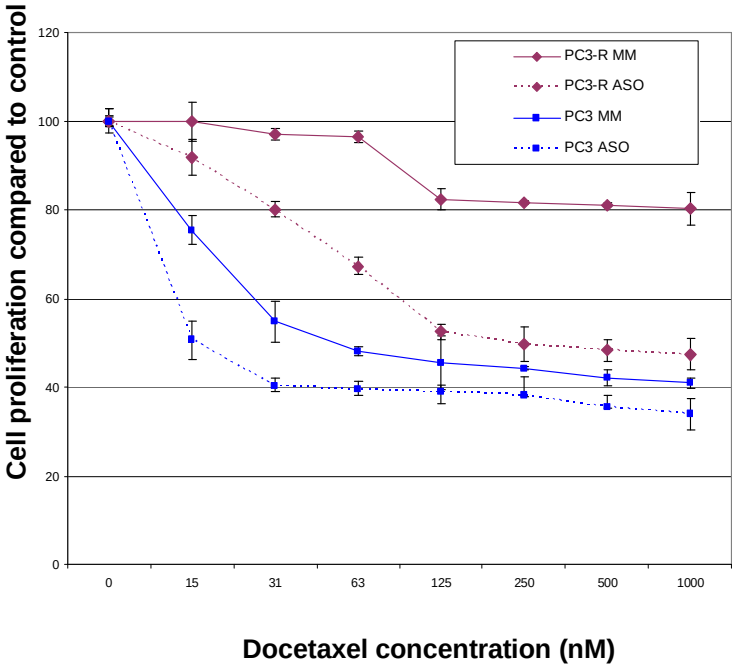


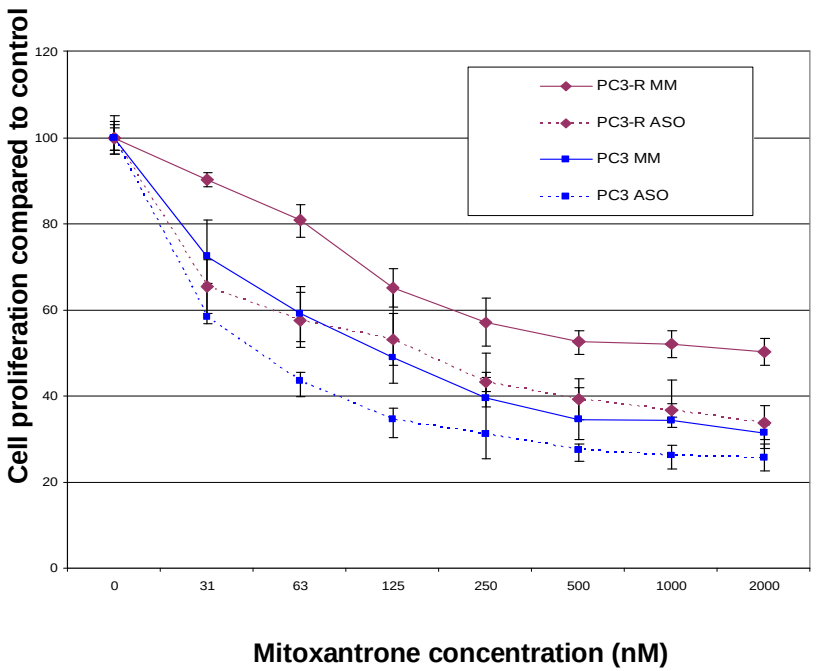
Cancer Research 60;170, 2000 *Clin Cancer Res 6:1655, 2000*

CLU Inhibition Enhances In Vitro Activity of Docetaxel & Mitoxantrone in Normal and Resistant Prostate Cancer Cells



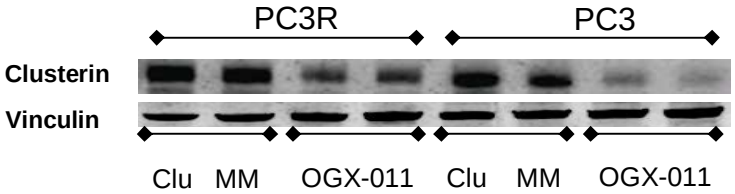




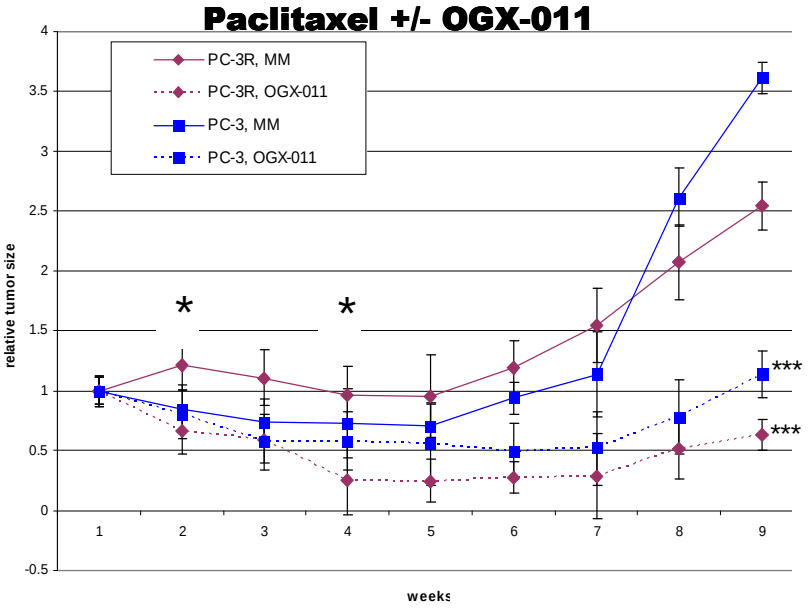


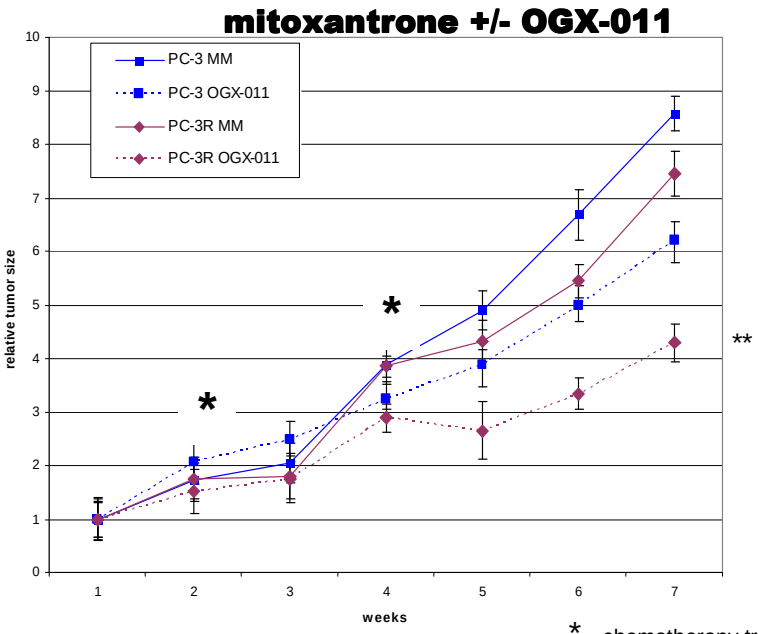
CLU Inhibition Enhances In Vivo Activity of Docetaxel & Mitoxantrone in Normal and Resistant Prostate Cancer Cells

Relative growth of PC3/PC3-R xenografts *in vivo* after taxane or mitoxantrone chemotherapy +/- OGX-011



***In vivo* target knockdown**





** P<0.05

*** P<0.01

* - chemotherapy treatment

* - chemotherapy treatment

Summary of Preclinical Findings

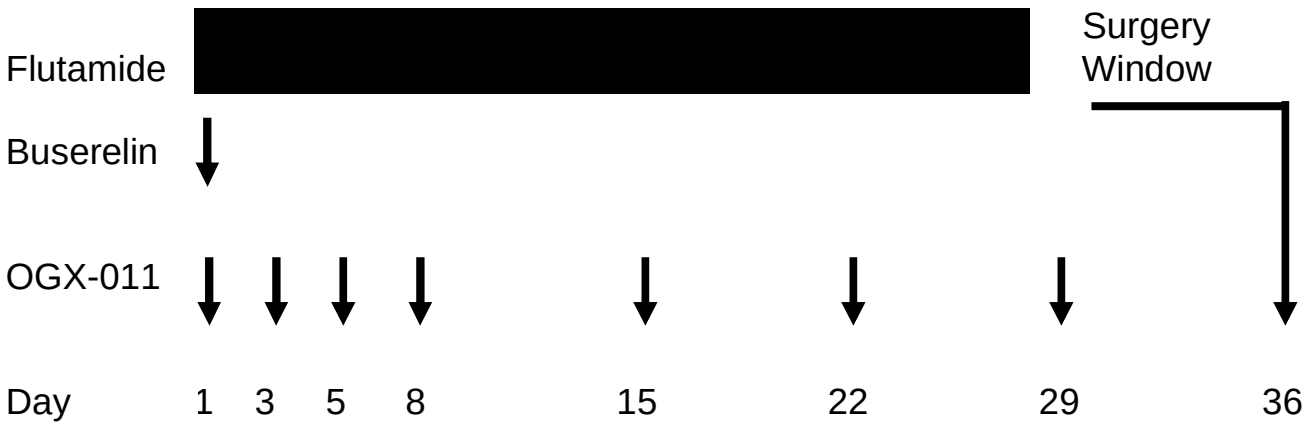
- Levels of sCLU are significantly increased in vitro and in vivo after chemotherapy or other anticancer treatment
- OGX-011 decreases sCLU-2 levels in a dose- and sequence-specific manner in vitro and in vivo
- Baseline levels of sCLU are significantly increased in docetaxel resistant PC-3 (PC3R) cell lines and these PC3R cell lines are multidrug resistant
- OGX-011 treatment suppresses sCLU protein levels and restores chemotherapy sensitivity both in vitro and in vivo, even in multidrug resistant PC3R cell lines
- When given in combination with chemotherapy, OGX-011 treatment showed a significant delay in in vivo tumor growth

Phase 1 Study Design To Establish Optimum Biologic Dose in Prostate Cancer

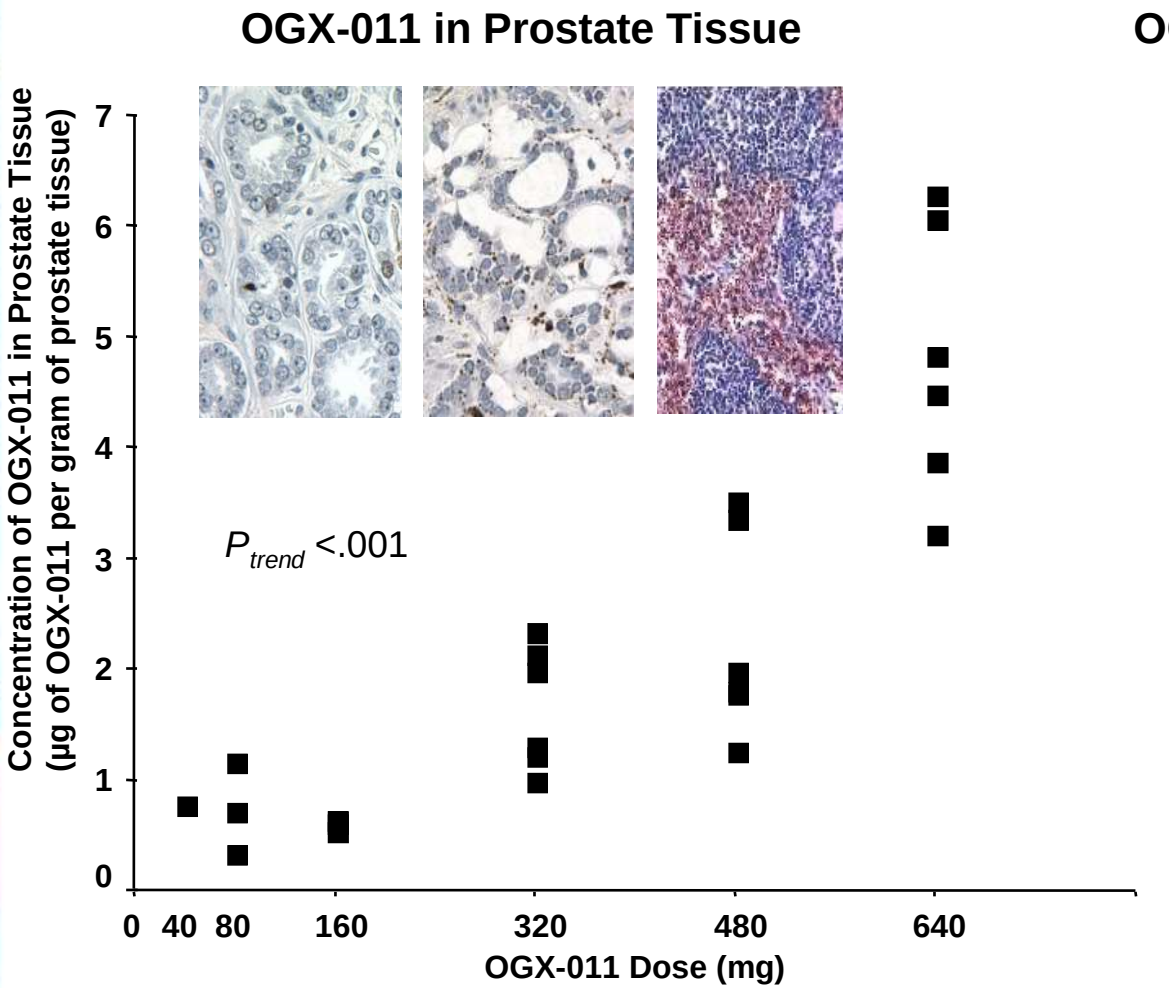
Objective: Determine Phase 2 dose from toxicity and biologic activity on target and surrogate tissues

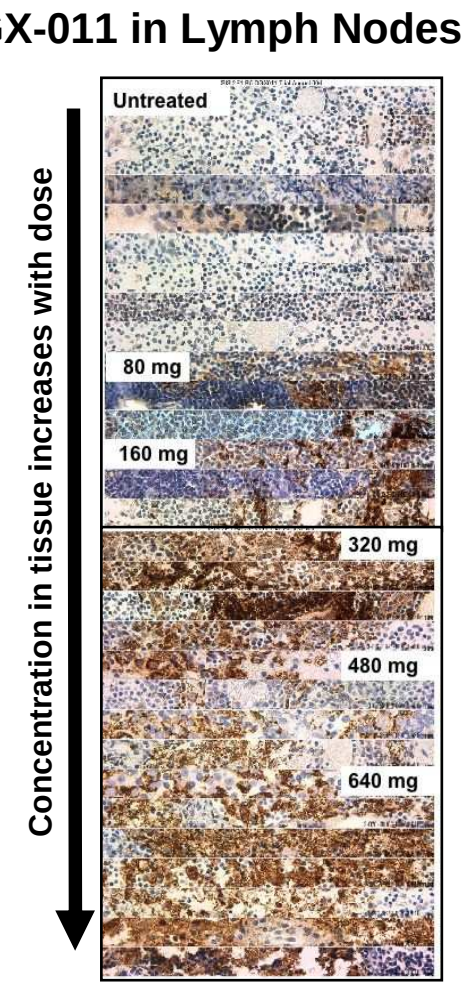
Patients: Localized prostate cancer with high risk features (PSA > 10, Gleason 7-10, T3, Gleason 6 and 3+ biopsies)

Treatment Schema:



OGX-011 Drug Concentration Measured in Prostate Tissue



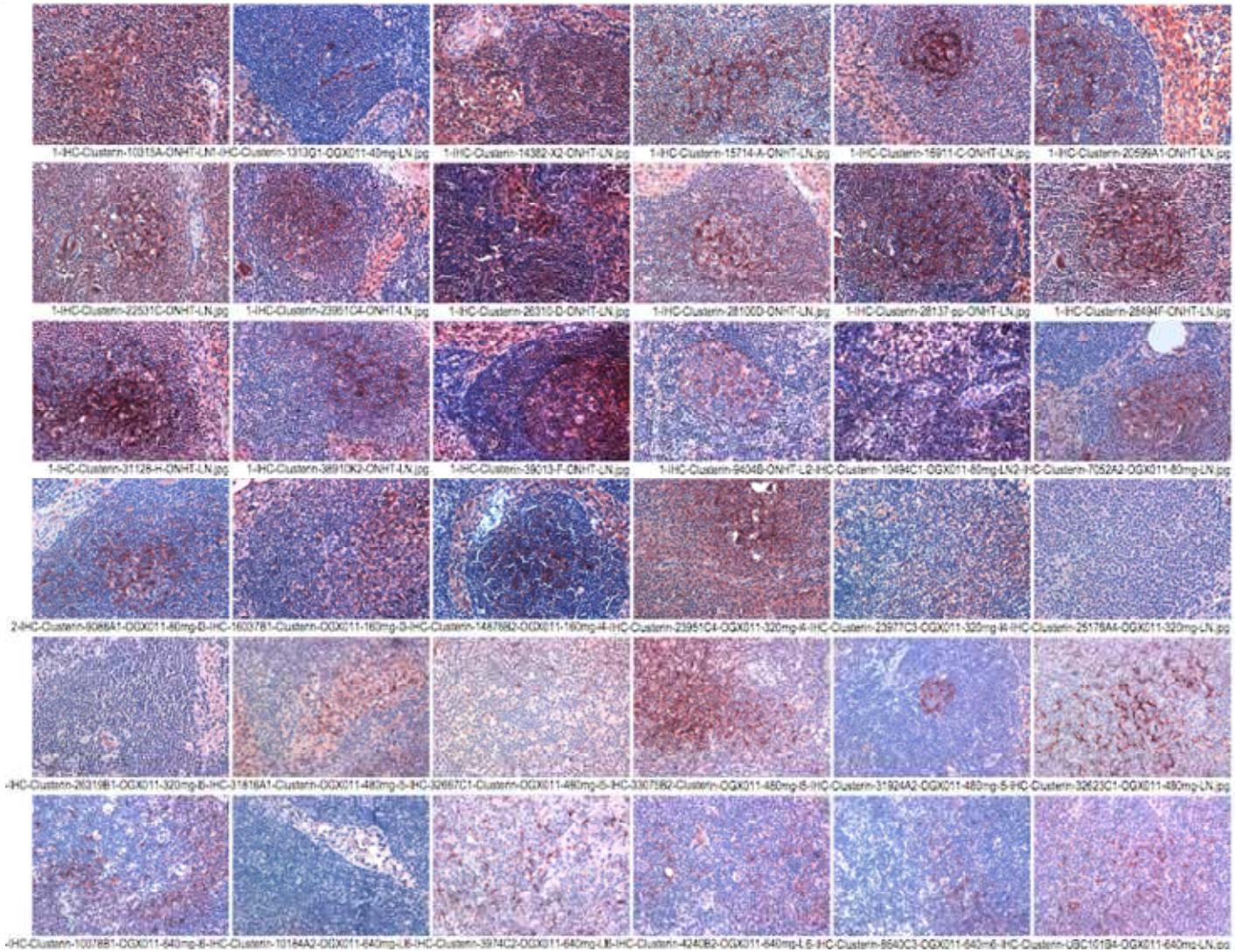


Chi *et al.*, JNCI. 97:1287-96, 2005

OGX-011 Target Regulation Dose-dependent Suppression of Clusterin in Regional Lymph Nodes

Untreated Controls

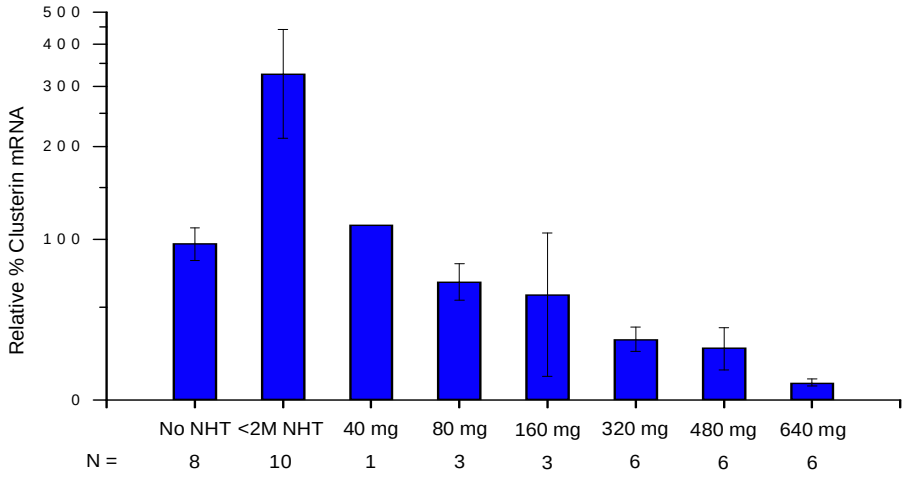
6 weeks of hormone ablation + OGX-011 (dose escalation)



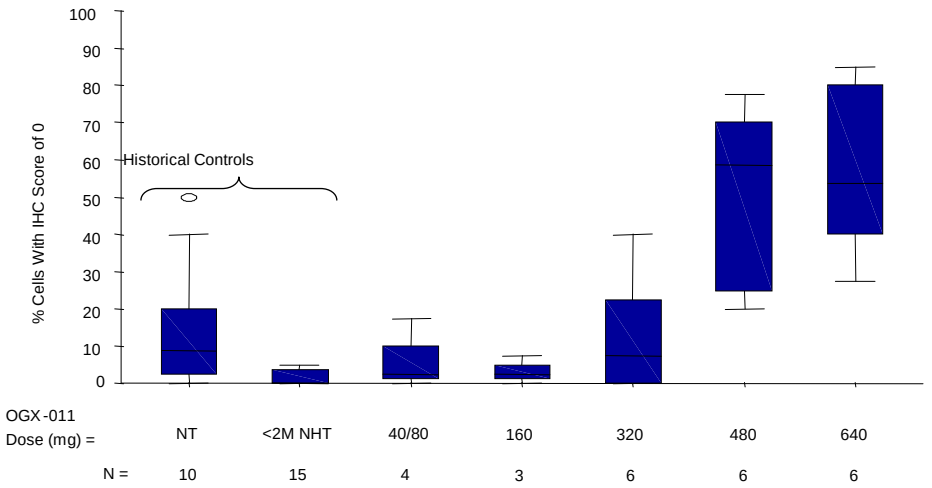
Chi *et al.*, JNCI. 97:1287-96, 2005

OGX-011: Dose Dependent Target Effects

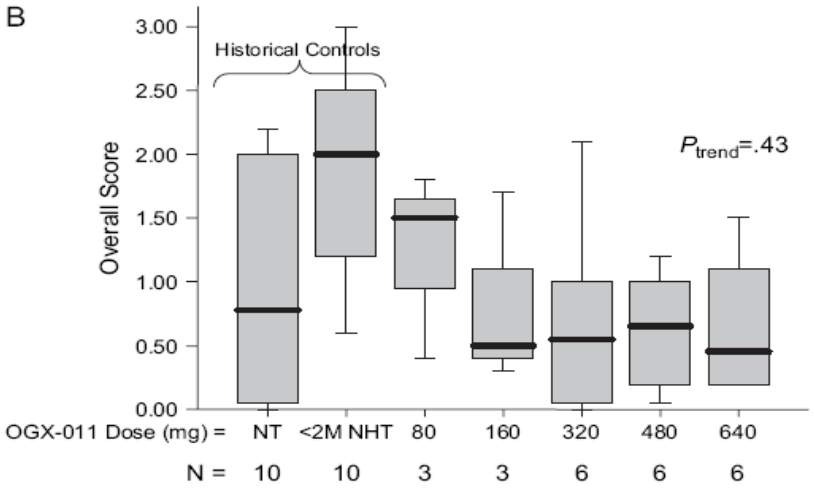
Inhibition of Clusterin mRNA



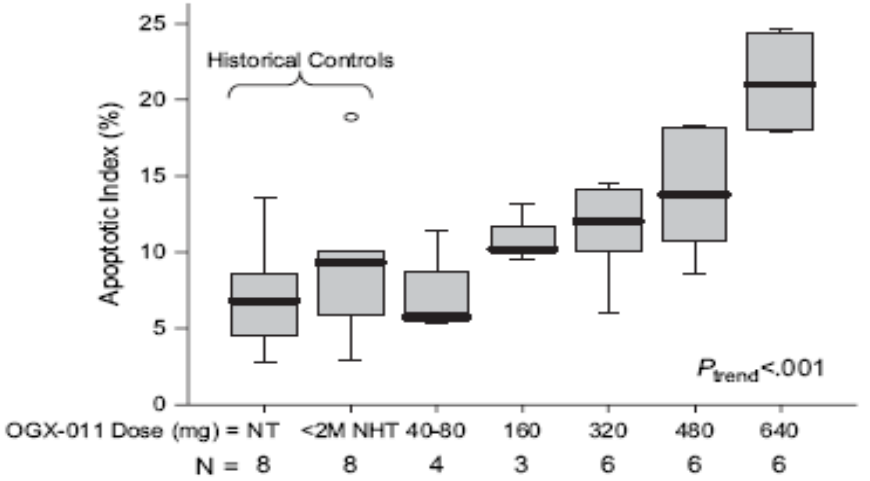
Inhibition of Clusterin Protein: IHC Score=0



Inhibition of Clusterin Protein: IHC Score



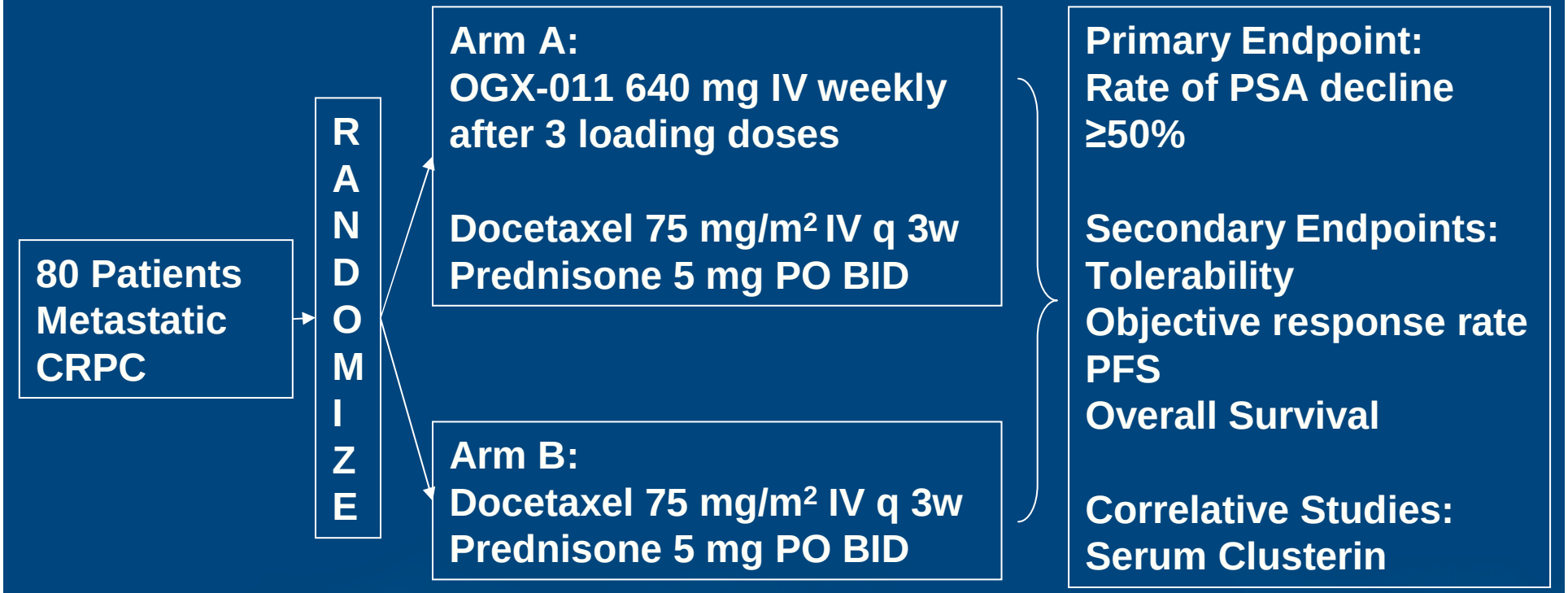
Apoptosis Index



A Randomized Phase II Study of OGX-011 in Combination with Docetaxel and Prednisone or Docetaxel and Prednisone Alone in Patients with Metastatic Castration Resistant Prostate Cancer

Kim N. Chi, Sebastien J. Hotte, Evan Yu,
Dongsheng Tu, Bernard Eigl, Ian Tannock, Fred Saad,
Scott North, Jean Powers, Elizabeth Eisenhauer
NCIC Clinical Trials Group

Study Design



Endpoint Definitions

- Primary
  - Rate of ≥ 50% PSA decline from baseline (minimum 5 ng/ml) confirmed ≥ 3 weeks later
- Secondary
  - Objective response rate by RECIST
  - Progression
    - Objective progression by RECIST
    - PSA progression
      - Non-responders: ≥25% increase from nadir (confirmed)
      - Responder: ≥50% increase from nadir (confirmed)
  - Survival
    - From date of randomization to progression or death

Study Design

- Non-comparative, single stage randomized phase II with internal control
  - H0 < 40%, H1 > 60%, $\alpha = 0.1$, $\beta = 0.1$
  - Further evaluation warranted if >20/40 patients had a PSA ≥50% decline in arm A

Key Eligibility Criteria

- Pathologic diagnosis of prostate adenocarcinoma
- Castrate resistance:
  - Rising PSA
  - New metastatic lesions
- PSA ≥ 5
- ECOG PS = 0-2
- No prior chemotherapy
- Adequate hematologic, renal and hepatic function
  - ANC ≥ 1.5 x 10^9/L, Platelets ≥ 100 x 10^9/L
  - Bilirubin ≤ ULN, AST/ALT ≤ 1.5 x ULN
  - Creatinine ≤ 1.5 x ULN

Accrual and Follow-Up

- 82 patients from 12 sites in Canada and USA were accrued from September 2005 to December 2006
- All patients are now off study treatment
- Median follow-up = 32 months
- 58 deaths

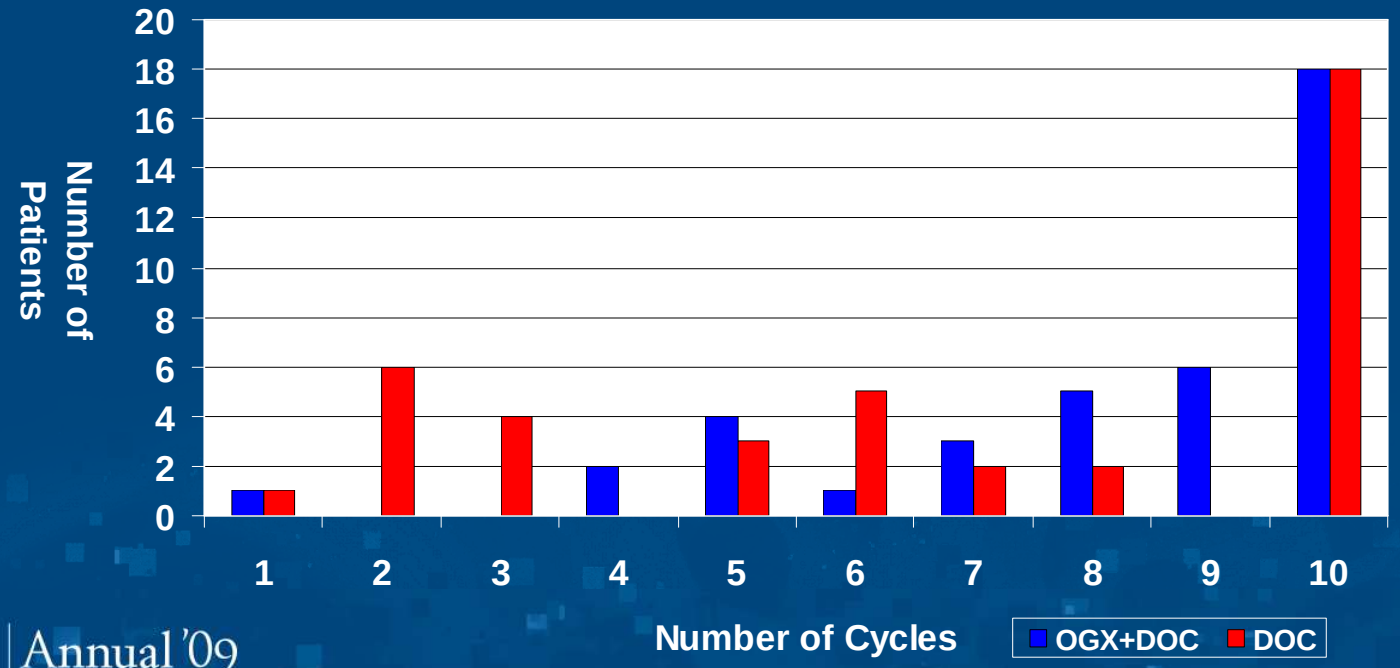
Baseline Characteristics

		*OGX + DOC N=40**	*DOC N=41*
Median age (range)		68 (54-84)	69 (49-87)
ECOG PS	0 : 1	21 : 19	20 : 21
Measurable disease	No : Yes	14 : 26	17 : 24
Bone/nodal metastases only	Yes : No	27 : 13	24 : 17
PSA	≤100 : >100	20 : 20	20 : 21
LDH	≤ULN : >ULN	24 : 16	28 : 13
Alk Phos	≤ULN : >ULN	23 : 17	22 : 19
Hemoglobin	<100 : ≥100	2 : 38	0 : 41
Gleason Score	≤7 : 8-9 : UNK	13 : 26: 1	18 : 22 : 1
Progression at randomization	Objective : PSA	5 : 35	9 : 32
Halabi nomogram predicted median OS		12.7 m (3.6-28.0)	11.1 m (3.5-30.1)

*One patient cancelled for ECOG PS=3

Cycles Administered

	Median Cycles (Range)	*Receiving > 90% Planned DOC Dose Intensity*
OGX + DOC	9 (1-10)	66.7
DOC	7 (1-10)	70.7



Reasons for Protocol Therapy Discontinuation

	OGX + DOC *N (%)*	*DOC* *N (%)*
Treatment complete (10 cycles)	18	16
Adverse event	9	5
Progression		
Total	7	16
Objective	3	7
PSA	2	6
Objective and PSA	2	3
Symptomatic progression	1	0
Death	0	1
Intercurrent illness	0	1
Refused treatment	3	0
Other	2	2

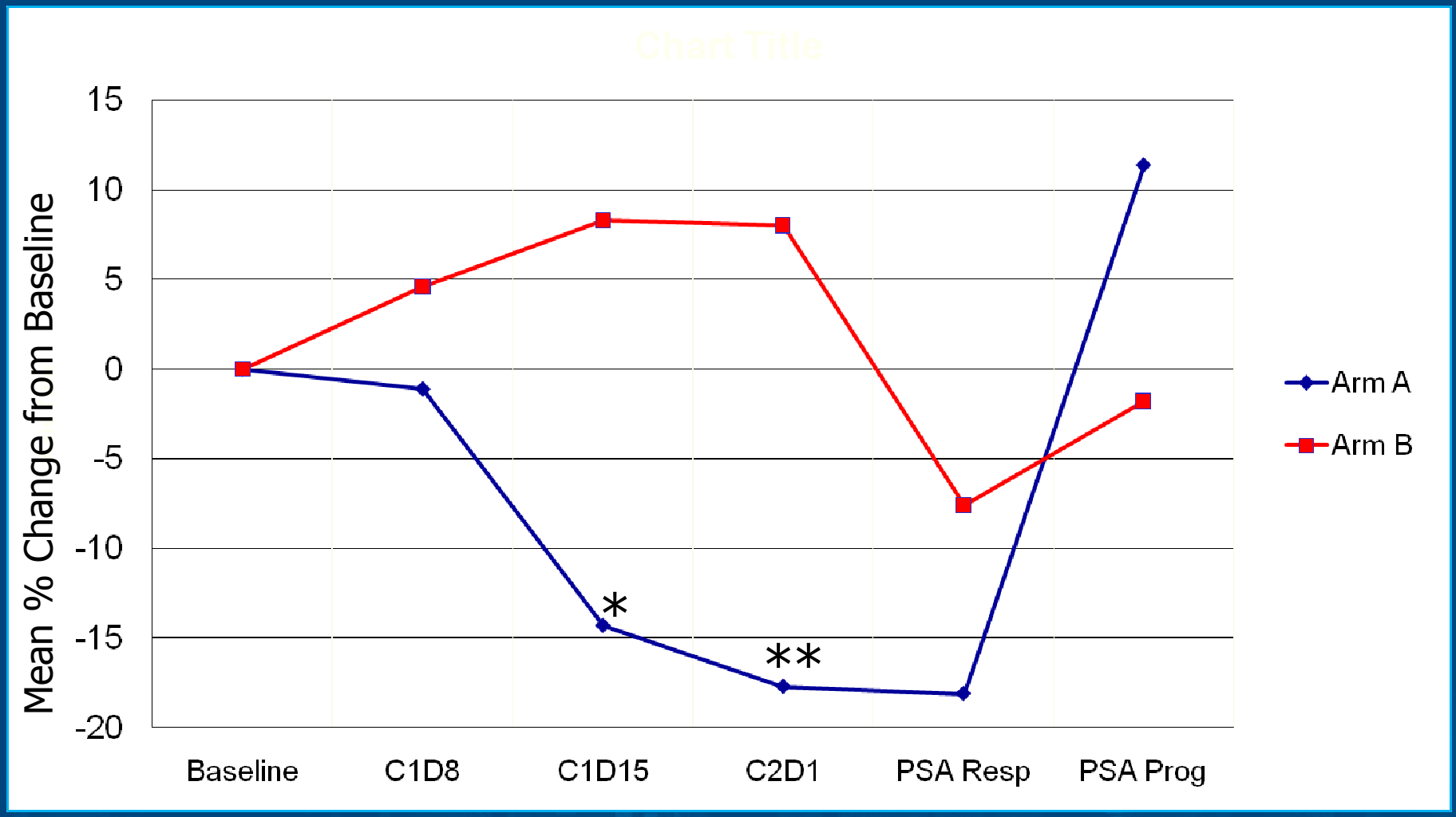
Grade 3-4 Hematologic Adverse Events

	OGX + DOC (N=40)	*DOC (N=41)*
Granulocytes	29	26
Leukocytes	18	22
Lymphocytes	**21**	**9**
Hemoglobin	0	3
Platelets	1	0

Related Grade 3-4 and Non-Hematologic Adverse Events

	Arm A (OGX + DOC)			*Arm B (DOC)*		
AE	***Grade 1-2***	***Grade 3-4***	***Total %***	***Grade 1-2***	***Grade 3-4***	***Total %***
Fatigue	28	4	80	25	8	80
Neuropathy (sensory or motor)	22	2	60	18	0	44
Diarrhea	20	1	53	18	2	49
Nausea	14	1	38	18	3	51
Pain	12	2	36	12	1	33
Vomiting	6	0	15	10	1	27
Febrile neutropenia	0	4	10	0	5	12
Dehydration	4	0	10	2	3	12
Rigors/chills	**23**	**0**	**58**	**2**	**0**	**5**
Fever	**18**	**0**	**45**	**5**	**0**	**12**
Elevated creatinine (normal baseline)	**8**	**0**	**20**	**2**	**0**	**5**

Results: Serum Clusterin (ELISA)



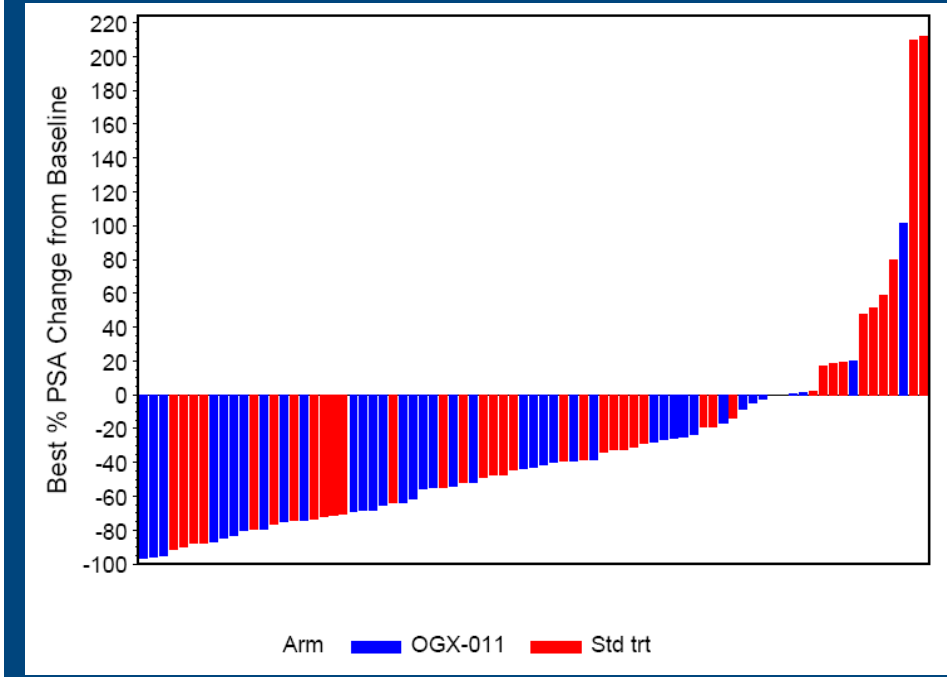
*P=0.05 **P=0.0005

Post-Treatment PSA Changes

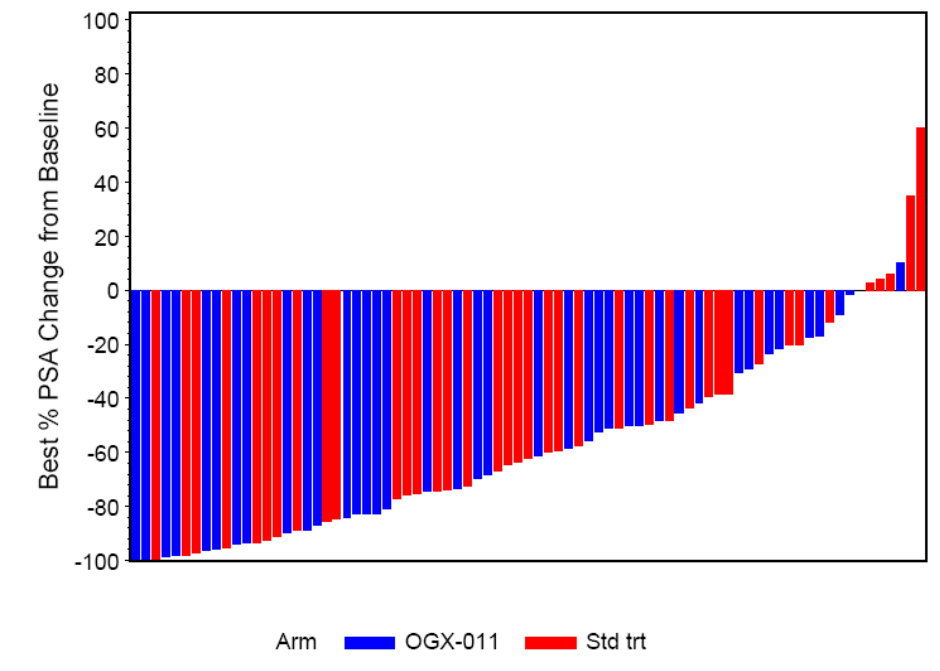
PSA Decline Criteria	*OGX + DOC N=40*	*DOC N=41*
≥ 50% decline (confirmed)	23 (58%)	22 (54%)
≥ 50% decline	26 (65%)	25 (61%)
≥ 30% decline at 12 weeks	26 (65%)	24 (59%)
≥ 30% decline	32 (80%)	31 (76%)
PSA progression	0 (0%)	3 (7%)
Inevaluable	1	1

PSA Waterfall Plots

12 weeks



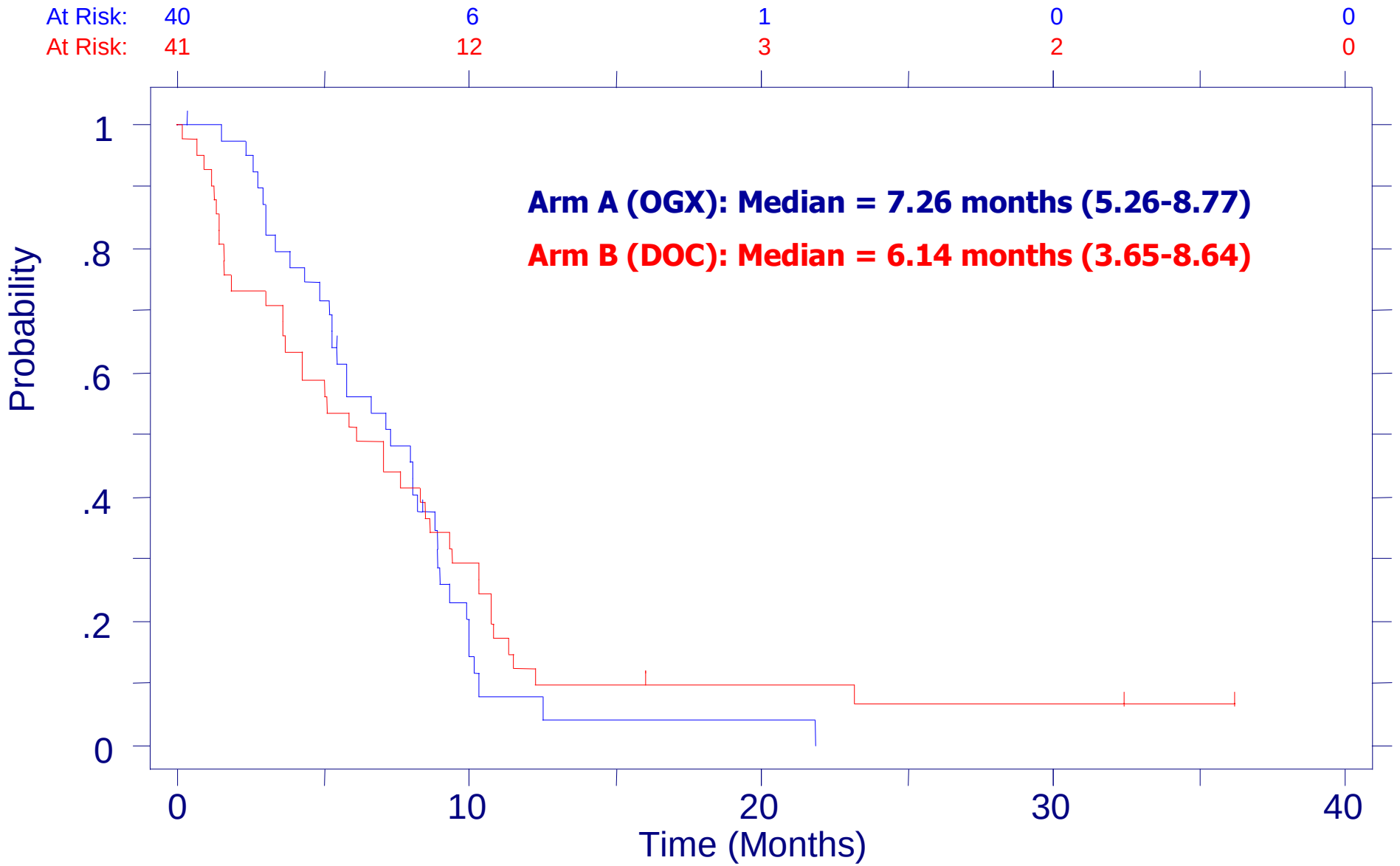
At any time



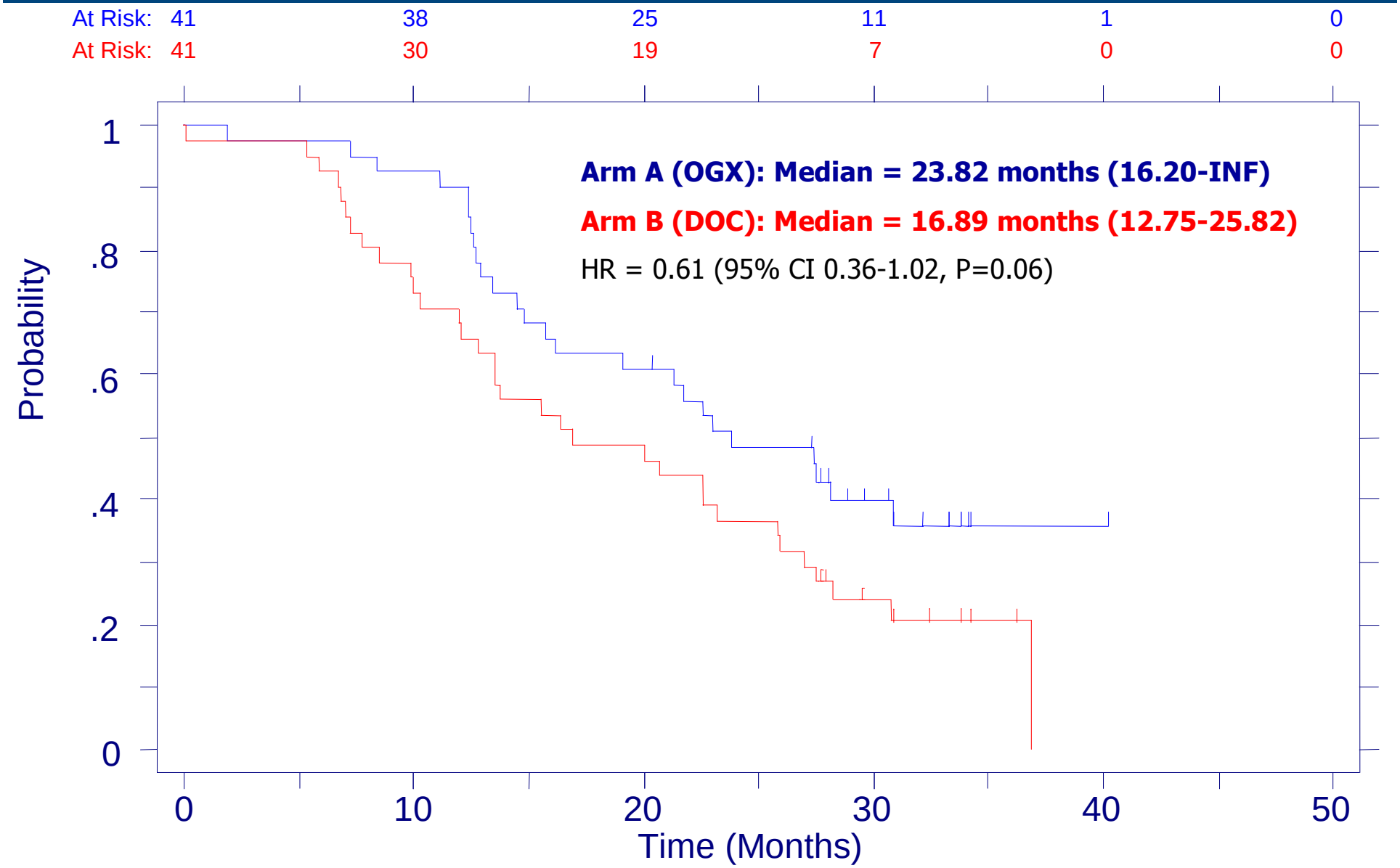
Measurable Disease Response

RECIST	*Arm A (OGX + DOC) N=26*	*Arm B (DOC) N=24*
Complete Response	0	0
Partial Response	5 (19%)	6 (25%)
Stable Disease	20 (77%)	12 (50%)
Progressive Disease	1 (4%)	4 (17%)
Inevaluable	0	2

Progression Free Survival



Overall Survival



Cox Multivariate Analysis

Variable	*N*	*HR (95% CI)*	*P*
OGX-DOC **DOC**	**41** **41**	**0.49 (0.28-0.85)**	**0.012**
PS 0 **PS 1**	**41** **41**	**0.28 (0.15-0.53)**	**<0.0001**
Other metastases **Bone/node only**	**31** **51**	**2.13 (1.20-3.77)**	**0.01**
HGB≥100 HGB<100	29 52	0.52 (0.27-1.02)	0.06
LDH≤ULN LDH>ULN	52 29	0.63 (0.34-1.20)	0.16
ALP≤2.5xULN ALP>2.5xULN	45 36	1.14 (0.58-2.22)	0.70
Pain No Pain Yes	22 60	1.27 (0.63-2.58)	0.51
PSA≤100 PSA>100	40 41	0.77 (0.44-1.33)	0.34

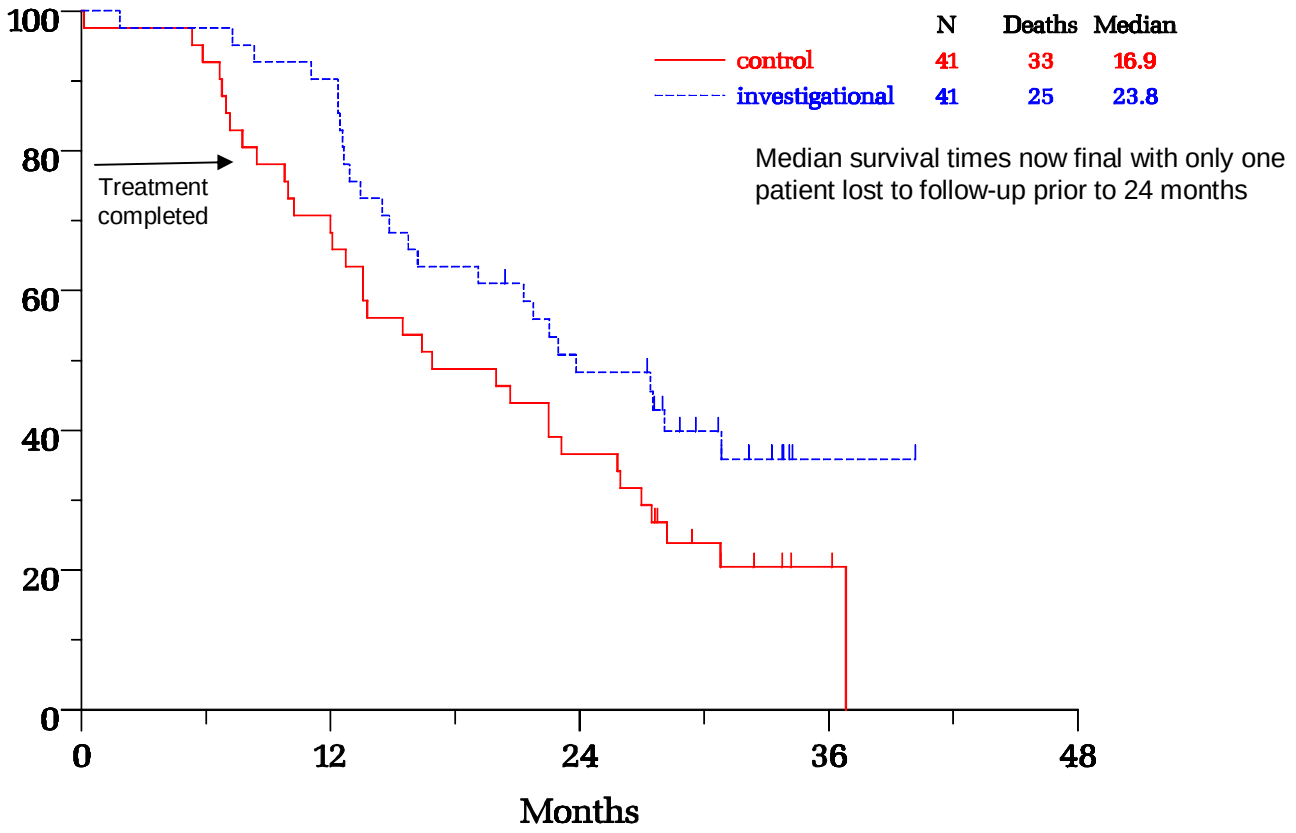
Exploratory Analysis: Number of Treatment Cycles and Overall Survival

Number of Cycles	*OGX + DOC N*	*DOC N*	*HR (95% CI)*
≤ 6 cycles	9	19	0.30 (0.08-1.12)
≤ 9 cycles	23	23	0.35 (0.15-0.83)
10 cycles	18	18	0.20 (0.04-0.93)

Conclusions

- OGX-011 is well tolerated in combination with docetaxel
- Evidence of biologic effect with serum clusterin decrease
- Primary endpoint: PSA decline rate with OGX-011/docetaxel therapy met protocol criteria for further study but control arm was similar
- Treatment with OGX-011/docetaxel combination was independently associated with improved overall survival in a pre-planned multivariate analysis (HR=0.49, P=0.012)
- Further evaluation of this combination in patients with CRPC is warranted

First-Line CRPC Phase 2 Study OGX-011-03:
Kaplan-Meier Survival Curves as of April 2009



Median for OGX-011: **23.82** 95% C.I. [16.2 - .Inf]
Median for Std Trt: **16.89** 95% C.I. [12.75 - 25.82]

Unadjusted **HR=0.61** [0.36-1.02], P=0.06
Multivariate analysis[1] **HR=0.49** [0.28-0.85], P=0.01

[1] Variables predictive of OS on multivariate analysis: PS 0 vs 1 (P < 0.0001), presence of visceral metastasis (P = 0.01) and treatment assignment

Observed Median Difference

- Often used to compare event time distributions.
- Advantage: Clinically intuitive.
- But:
  - Can change dramatically as more events are accrued (i.e. lacks stability).
  - HR is much more robust to accrual of more events.

Hazard Rate

- Probability of event in the next instant of time for patient yet to have event.
- Element of mathematical description of distribution of event times (complicated).
- Hazard rate may be constant or vary with time.
- While not often explicitly estimated, the hazard rate is integral to analyses of event times.

Hazard Ratio (HR)

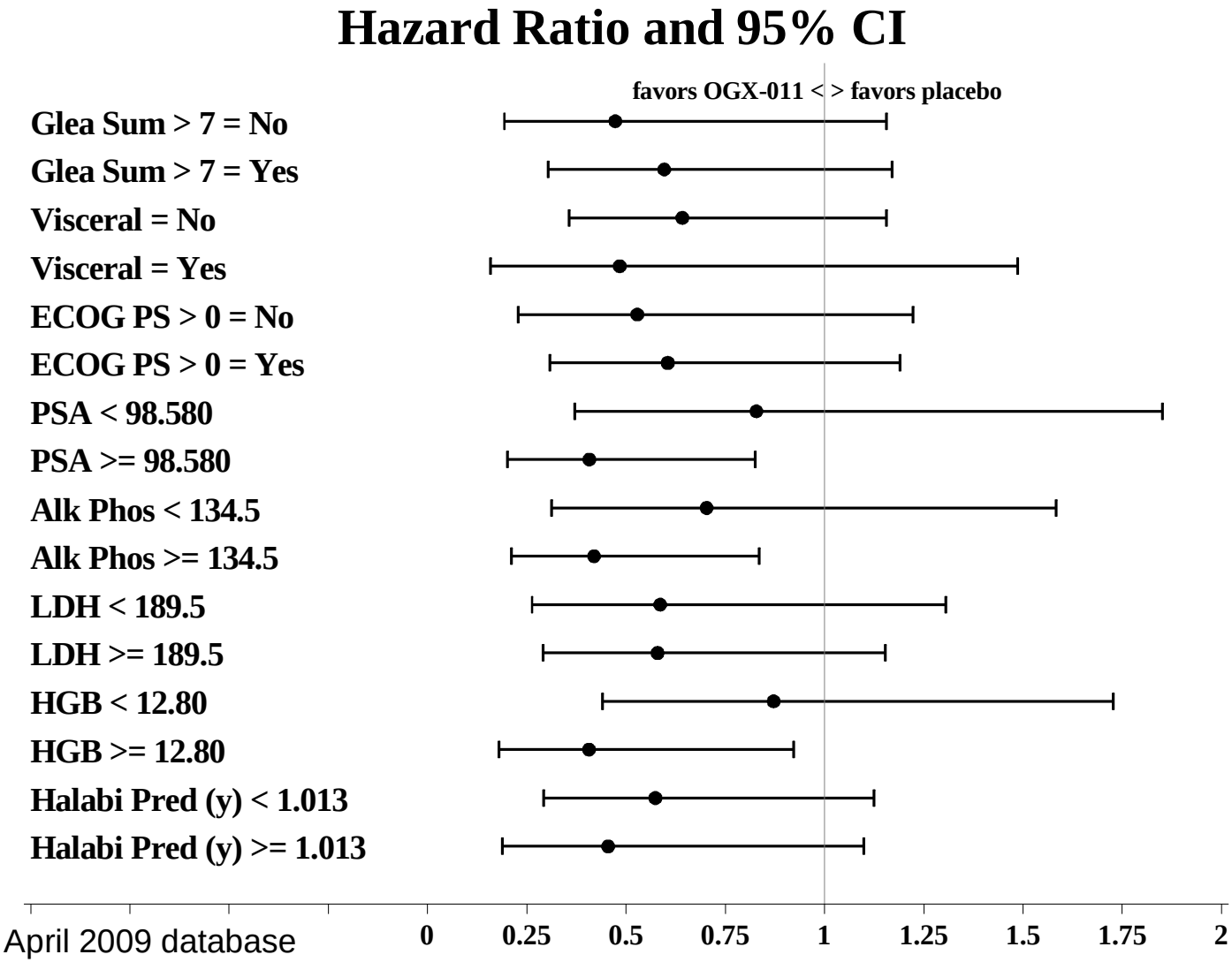
- Measure used to compare event time distributions, e.g., from arms of randomized trial.
- Usual definition: Experimental arm hazard rate divided by control arm hazard rate.
  - HR = 1 → hazard rates are equal.
  - HR < 1 → experimental arm has lower rate.
- Assume N control arm events are observed:
  - If HR < 1 then number of experimental arm events will be < N over same time period.
  - Formula for number of experimental arm events is complicated.

Estimating HR from Data

- Usually estimated via proportional hazard (Cox) regression (PHR).
- PHR assumes HR is constant for all time (proportional hazards assumption):
  - Does not assume hazard rates are constant.
  - Has been found to be generally reasonable (though there are exceptions).
- Estimated HR (and its confidence interval) is a reasonable and useful summary comparing event time distributions of two arms.

First-Line CRPC Phase 2 Study OGX-011-03:

OGX-011 Effect Hazard Ratios for Baseline Prognostic Factors



Based on April 2009 database

First-Line CRPC Phase 2 Study OGX-011-03: Site Sensitivity Analyses for OGX-011 Treatment Effect for the 12 Sites

PHR Results for Arm
Site Stratified PHR Model with Covariates PS and Visceral

clin site del	*arm est log(hr)*	*arm hr*	*arm z stat*	*arm p*
none	-0.9126	0.4015	-2.8579	0.0043
CABN	-0.8948	0.4087	-2.7890	0.0053
CAEJ	-0.9877	0.3724	-3.0276	0.0025
CAHN	-0.9453	0.3885	-2.8582	0.0043
CALM	-0.8045	0.4473	-2.3347	0.0196
CAMP	-0.9906	0.3714	-2.9049	0.0037
CANL	-0.8828	0.4136	-2.7470	0.0060
CARM	-0.8700	0.4189	-2.7001	0.0069
CATC	-0.9105	0.4023	-2.7895	0.0053
CATW	-0.9343	0.3928	-2.7834	0.0054
CAVA	-1.0887	0.3367	-2.5433	0.0110
CAVK	-0.8975	0.4076	-2.7957	0.0052
USVY	-0.8422	0.4307	-2.5524	0.0107

The OGX-011 treatment benefit on survival remained significant even when one site at a time was deleted from the analysis.

Thus, the survival benefit was not dependent on results predominantly from one site.

Based on April 2009 database

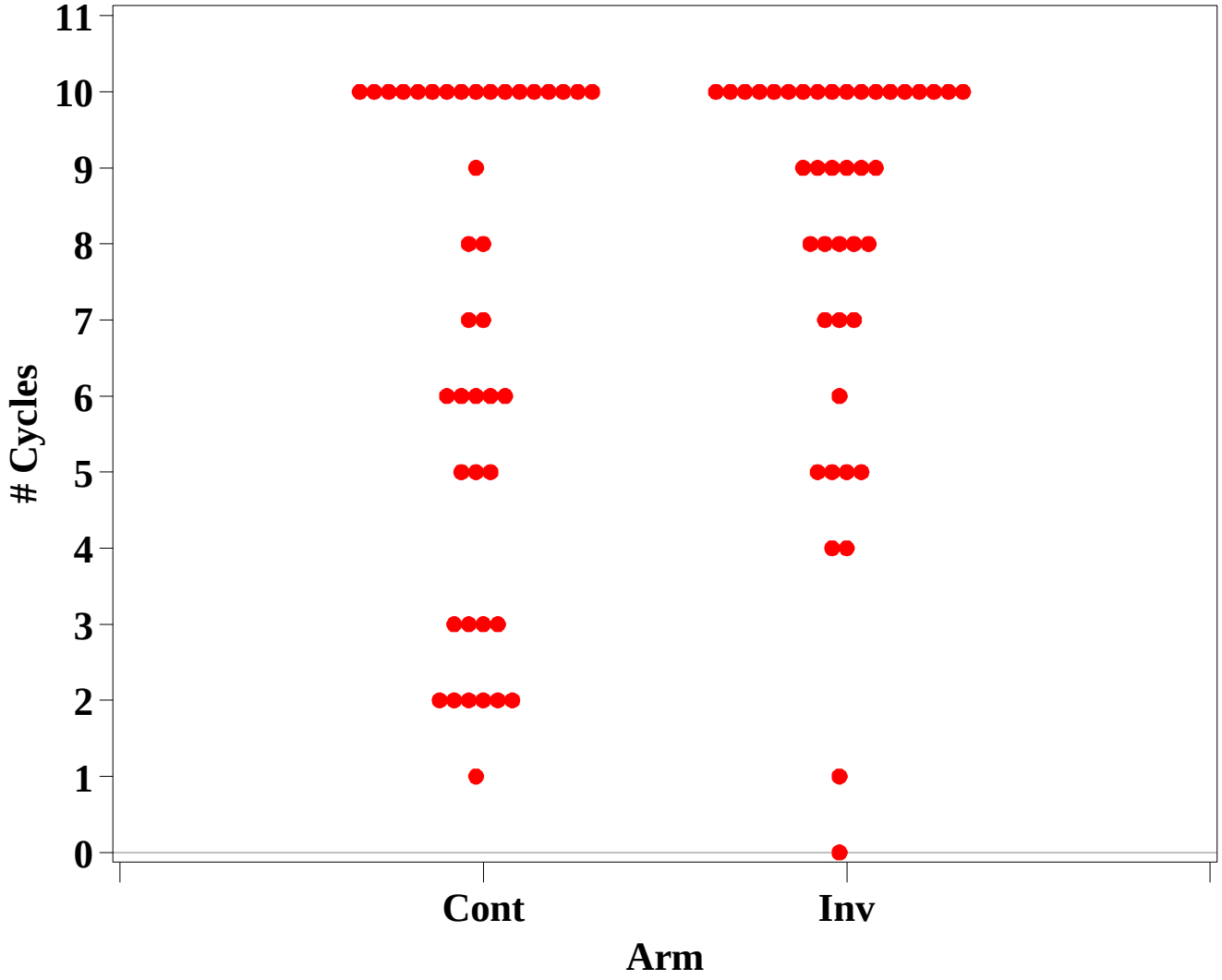
First-Line CRPC Phase 2 Study OGX-011-03:
Early Treatment Discontinuation Prior to Cycle 5, Day 1

Reasons for Off Study Prior to Cycle 5, Day 1	*OGX-011 + Docetaxel N = 40*	*Docetaxel N = 41*
Death	**0**	**1**
PSA Progression	**0**	**2**
PSA and Objective disease progression	**2**	**1**
Objective disease progression	**0**	**4**
Adverse Event	**1**	**3**
Total # of patients (%)	**3 (7.5%)**	**11 (26.8%)**

The percent of patients discontinuing study treatment prior to Cycle 5, Day 1, in the control arm (26.8%) was 3.6 fold greater than in the OGX-011 arm (7.5%), primarily due to disease progression

First-Line CRPC Phase 2 Study OGX-011-03:

Number of Chemotherapy Cycles Administered on Study



First-Line CRPC Phase 2 Study OGX-011-03:

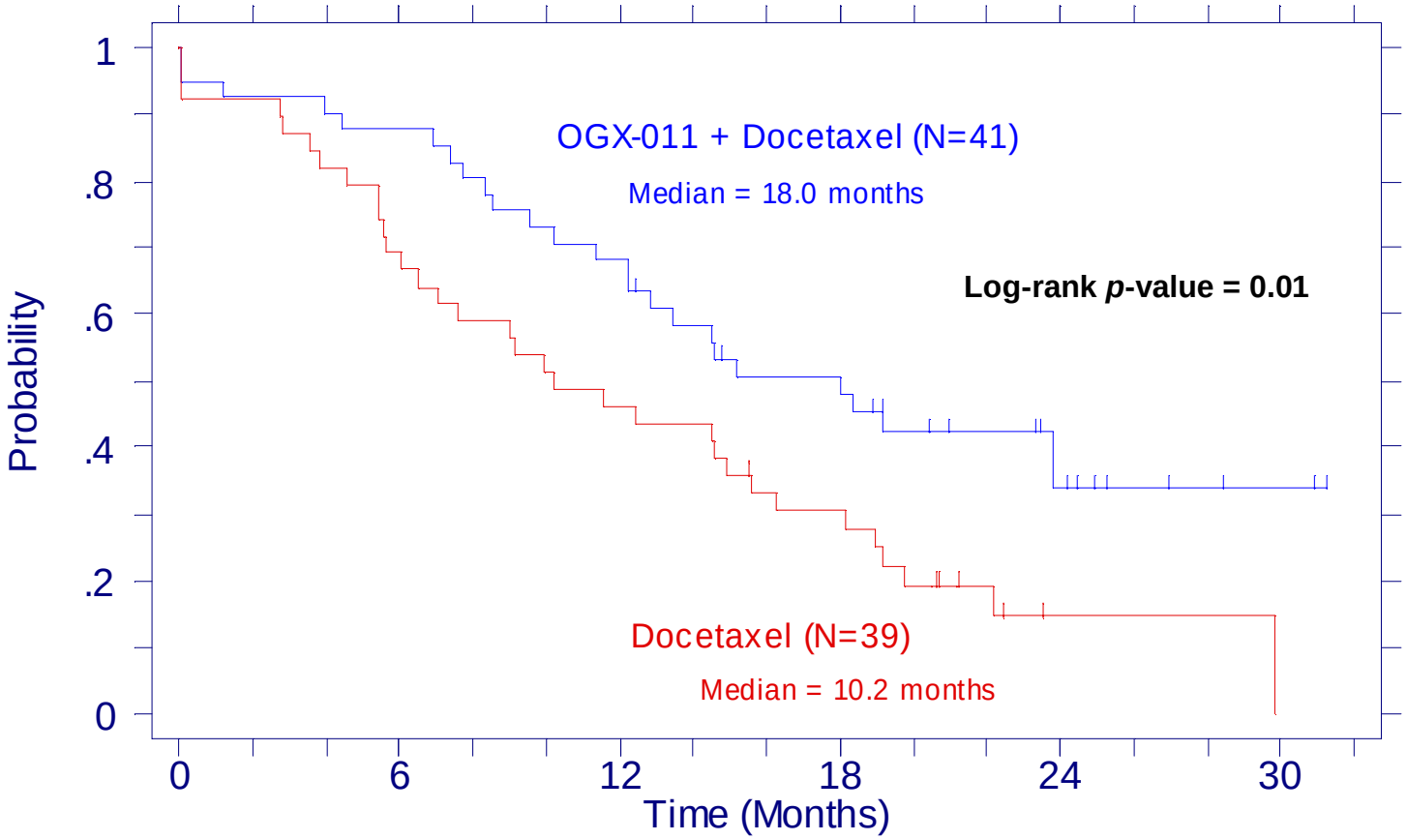
Exploratory Analysis on Chemotherapy Cycles Administered

- Table below breaks down the survival trend by number of chemotherapy cycles received. The number of alive patients (i.e. censored as of April 2009) / total of patients who received the number of cycles are stated below with the median survival time

No. of Chemotherapy cycles	**Docetaxel + OGX-011 (median survival)**	**Docetaxel (median survival)**
1 – 5 cycles	1 pt / 7 pts (14.8 mo)	1 pt / 14 pts (9.1 mo)
6 – 9 cycles	4 pts / 15 pts (22.5 mo)	0 pt / 9 pts (12.0 mo)
10 cycles	11 pts / 18 pts (Estimate not available*)	7 pts/ 18 pts (27.8 mo)
Total patients (median survival)	**16 pts/ 40 pts (23.8 mo)**	**8 pts / 41 pts (16.9 mo)**

* **Median follow-up at 32 months**

First-Line CRPC Phase 2 Study OGX-011-03:
Time from Disease Progression to Death



First-Line CRPC Phase 2 Study OGX-011-03:
Anticancer Therapies Post Study Completion (April 2009)

Therapy Received	**Docetaxel + OGX-011 N=40 Number of patients (%)**	**Docetaxel N=41 Number of patients (%)**
Investigational Agent	10 (25)	11 (27)
Anti-Androgen Therapy	5 (13)	1 (2)
Prednisone Therapy	18 (45)	13 (32)
Chemotherapeutic Agents		
– Docetaxel	13 (33)	12 (29)
– Mitoxantrone	12 (30)	7 (17)
– Epothilone B	8 (20)	4 (10)
– Other chemotherapy agents	3 (8)	1 (2)
Other Therapies*	3 (8)	1 (2)

*Other therapies consisted of ketoconazole, strontium 89 and zoledronate

First-Line CRPC Phase 2 Study OGX-011-03:
Frequency & Timing of Anticancer Therapies

Number of patients or Timing of Subsequent Therapy	**Docetaxel + OGX-011 N=40**	**Docetaxel N=41**
Patients receiving any subsequent therapy	28 (70%)	22 (54%)
Median Time from disease progression (DP) to any subsequent therapy (Range)	2.5 months (0+ to 12.5 months)	2.3 months (0+ to 21.9 months)
Patients receiving subsequent chemotherapy	26 (65%)	18 (44%)
Median Time from DP to subsequent chemotherapy (range)	2.6 months (0+ to 22.7 months)	2.3 months (0+ to 6.7 months)
Median Time from end of study treatment to subsequent chemotherapy (range)	4.2 months (0.1 to 24.6 months)	5.1 months (0.7 to 12.1 months)

Slight trend for more post therapy in the OGX-011 treatment arm; however, patients living longer will have more opportunity for post therapy (Note: no 2nd-line therapy has shown a survival benefit)

FDA Highlights for OGX-011

2008

- Fast Track designation received from FDA for the development of OGX-011 based on survival correlation to serum clusterin levels during treatment
- SPA approved with FDA for Phase 3 trial of OGX-011 evaluating overall survival in 2nd-line docetaxel treatment of CRPC

2009

- SPA approved with FDA for Phase 3 trial of OGX-011 evaluating durable pain palliation in 2nd-line docetaxel treatment of CRPC
- FDA has agreed to our plan for amending the Phase 3 trial of OGX-011 evaluating overall survival in 2nd-line docetaxel treatment to 1st-line docetaxel treatment based on Study OGX-011-03 results

Clinical Development Strategy for Approval

- Clinical Development Strategy is to obtain the following initial indication:

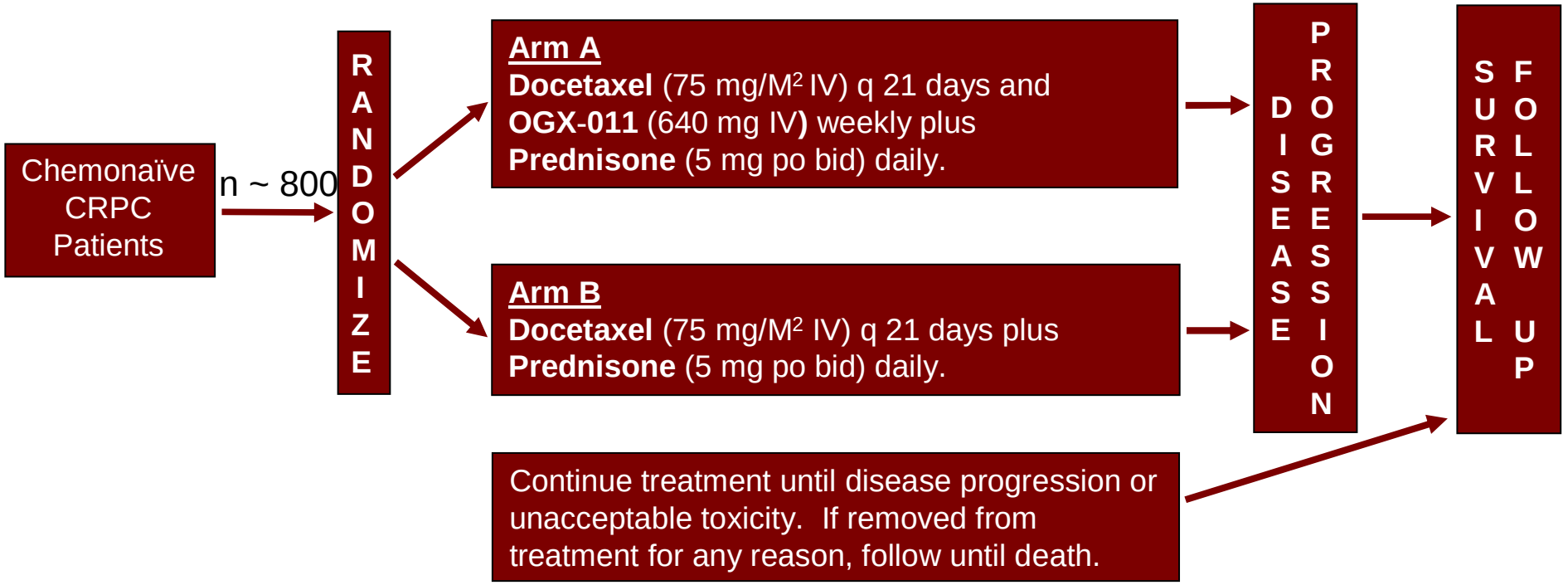
  OGX-011 in combination with docetaxel chemotherapy is indicated for the treatment of metastatic CRPC

- Two Phase 3 trials will provide evidence of safety and efficacy for OGX-011 in patients with metastatic CRPC.
  - OGX-011-11 (OGX-011-08 amended): Primary endpoint is overall survival in 1st-line docetaxel chemotherapy
  - OGX-011-10: Primary endpoint is durable pain palliation in 2nd-line docetaxel chemotherapy

Phase 3 Trial in CR Prostate Cancer:

Clinical Benefit of OGX-011 with 1st Line Docetaxel Chemotherapy

Study Design



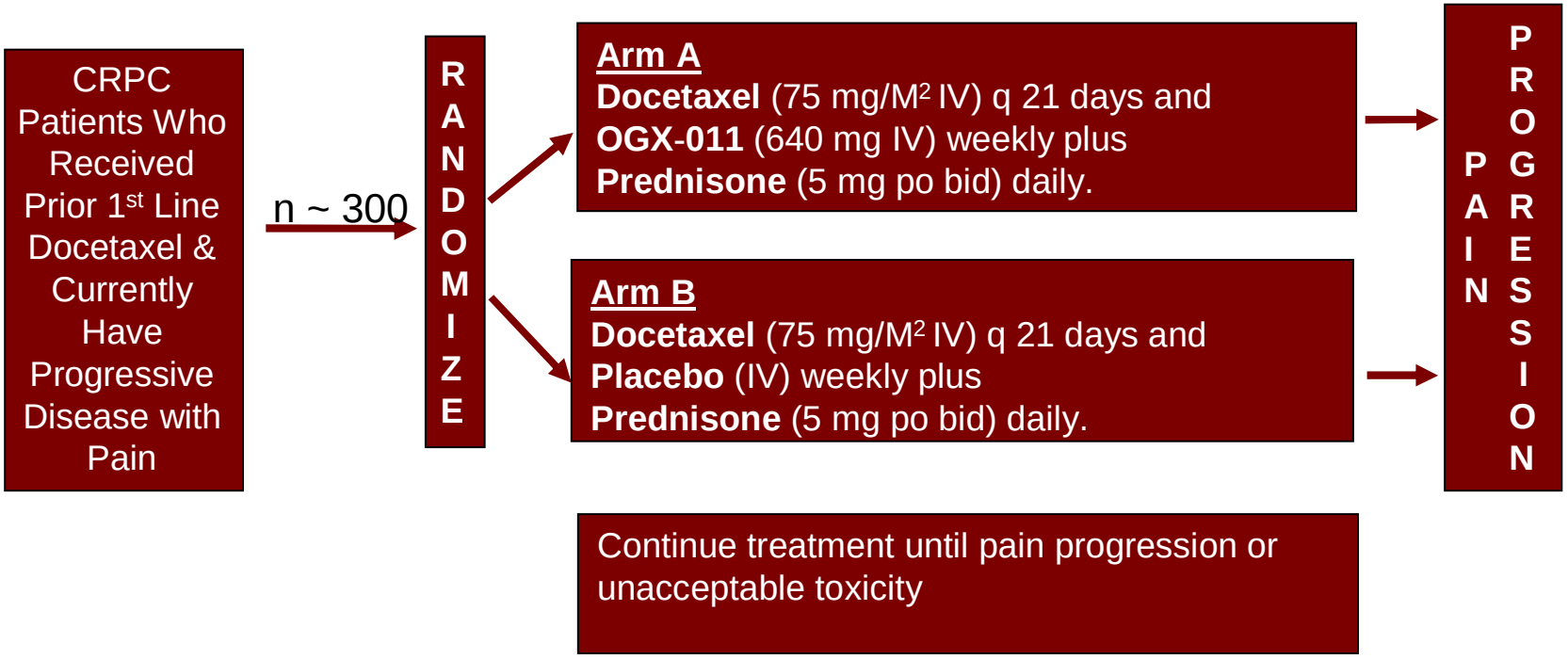
Primary Endpoint = Overall Survival

Amendment to SPA to be completed with FDA

Phase 3 Study in CR Prostate Cancer:

Clinical Benefit of Adding OGX-011 to 2nd Line Docetaxel Chemotherapy

Study Design



Primary Endpoint = Pain Palliation for 12 week duration

Special Protocol Assessment (SPA) with FDA Completed