ONCOGENEX PHARMACEUTICALS, INC.

FORM 425

(Filing of certain prospectuses and communications in connection with business combination transactions)

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Commission File No.: 033-80623

On February 21, 2017, OncoGenex Pharmaceuticals, Inc. provided copies of the following posters to investors:

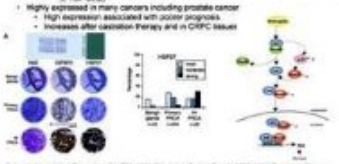
PACIFIC Trial: A Randomized Phase II Study of Apatorsen and Abiraterone in Patients with Metastatic CRPC Who Have Had PSA Progression While Receiving Abiraterone

Kim N. Chi[1,2], Mark Fleming[3], Katherine Sunderland[1], Costantine Albany[4], Joel Gingerich[5], Fred Saad[6], Scott North[7], Alexander Starodub[8], Richard Lauer[9], Dean Ruether[10], Madelaine Sgroi[11], Mark Scholz[12], Christopher Sweeney[13]

[1]BC Cancer – Vancouver Centre, [2]Vancouver Prostate Centre, [3]Virginia Oncology Associates, [4]Indiana University Melvin and Bren Simon Cancer Center, [5]CancerCare Manitoba, [6]Centre Hospitalier de l Université de Montréal (CHUM), [7]Cross Cancer Institute, [8]Goshen Center for Cancer Care, [9]University of New Mexico Cancer Center, [10]Alberta Health Services Tom Baker Cancer Centre, [11]IU Health Central Indiana Cancer Centers, [12]Prostate Oncology Specialists Inc, [13]Dana-Farber Cancer Institute

BACKGROUND

- Heat Shock Protein 27 (Hsp27)
  - Stress activated, ATP-independent member of the small heat shock protein group
  - Phospho-activated to form chaperoning oligomer which regulates multiple cell signaling and survival pathways
    - Inhibits apoptosis along intrinsic and extrinsic pathways
    - Involved in proteasome mediated degradation
    - Facilitates normal protein folding and function
      - Steroid receptors (AR, ER); growth factors (IGF-1, VEGF-1, FGF); cytokines (IL-6, TGF-beta)
  - Highly expressed in many cancers including prostate cancer
    - High expression associated with poorer prognosis
    - Increases after castration therapy and in CRPC tissues



Rutkowski et al., J Natl Cancer Inst, 97:1708, 1999; Rocci et al, Cancer Res, 64:6595, 2004; Rocci et al, Cancer Res, 65:11302, 2005; Zoubeidi et al, Cancer Res, 67:10455, 2007

- Apatorsen (OGX-427, OncoGenex Technologies Inc.)
  - Second generation phosphorothioate antisense oligonucleotide with 2'-MOE modifications
    - Prolonged tissue half life >10 days
    - Inhibits Hsp27 expression *in vitro* and *in vivo* with single agent anti-tumour activity
    - Enhances efficacy of hormone and chemotherapy
    - Disrupts AR function and enhances AR degradation
  - Phase I Studies (Chi *et al*, Annals Oncol 2016; 1116-1122, 2016)
    - Dose-dependent grade 1-2 infusion reactions
    - Reductions in tumor markers in patients with prostate and ovarian cancer
    - Declines in circulating tumour cells (CTC) observed.
  - Randomized Phase II study in Treatment Naïve CRPC (Chi *et al, J Clin Oncol 30, 2012 (suppl; abstr 4534)*
    - PSA >50% decline in 41% (OGX-427) vs 20% (Prednisone)

Figure 1. Apatorsen suppresses Pb-Luc Bioluminescence, AR, Hsp90 and Hsp27 *in vivo* (LNCaP)

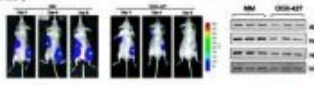
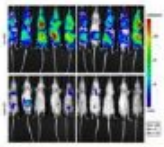


Figure 2. Apatorsen prevents metastases (PC-3M)

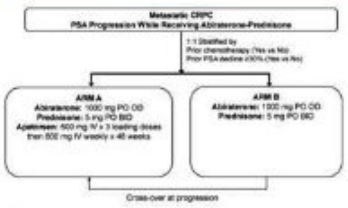


Zoubeidi et al, Cancer Res, 67:10455, 2007

OBJECTIVES

- Primary
  - Progression-Free Survival at the milestone Day 85 assessment: to ascertain whether Apatorsen added to Abiraterone-Prednisone has a greater proportion of patients observed to be alive without progression at Day 85 (±7 days) as compared to continuing Abiraterone-Prednisone in men with mCRPC and PSA progression on Abiraterone-Prednisone.
    - Disease progression is defined by any one of the following: Measurable disease, progression per RECIST 1.1 by CT, Bone scan: ≥ 2 new lesions confirmed; Disease related deterioration of health status. Need for palliative radiation therapy
- Secondary
  - The proportion of patients who have a PSA response (≥ 30% decline) and any PSA decline post-randomization
  - Objective response rate
  - Progression-free survival (PFS)
  - Time to disease progression
  - Circulating tumor cell (CTC) counts at baseline and on study

STUDY DESIGN



- Key Inclusion Criteria
  - Metastatic CRPC
  - ECOG performance status 0-1
  - Evidence of PSA progression only while on Abiraterone and Prednisone
  - 0-1 prior lines of chemotherapy for CRPC
  - Baseline laboratory values:
    - ANC ≥ 1.5 x 10^9 cells /L, platelet count ≥ 10^5 x 10^9/L, and hemoglobin ≥ 9 g/dL
    - Creatinine ≤ 1.3 x ULN
    - Total bilirubin ≤ 1.1 x ULN, ALT and AST ≤ 2.0 x ULN
- Statistical Considerations
  - Planned sample size was 74 patients with patients randomized with equal probability to the two treatment arms.
  - The one-sided type I error probability for the outcome is specified to be 10%, giving an overall type I error probability for the primary endpoint of 20%. Assuming control arm success probability is 5%, then there will be 90% power to detect a hypothesized 25% increment in the success probability.

BASELINE PATIENT CHARACTERISTICS

Parameter	ARM A (ABI + P + Apatorsen) (N=36)	ARM B (ABI + P) (N=36)
Median Age, years (range)	71 (53-85)	72 (57-89)
ECOG PS 2 : 1	16 (44%): 20 (56%)	16 (44%): 20 (56%)
Median PSA, ug/L (range)	34.10 (2.51-387.60)	18.17 (4.24-302.15)
Median Hemoglobin, g/L (range)	12.9 (10-152)	13.2 (10-132)
Disease Sites		
Bone	21 (58%)	24 (69%)
Lymph Node	13 (36%)	6 (17%)
Liver/Lung	2 (5%)	5 (14%)
Laciate Dehydrogenase >ULN	12 (33%)	6 (17%)
Alkaline Phosphatase >ULN	12 (33%)	6 (17%)
Prior Therapy		
Abiraterone	1 (3%)	0
Docetaxel	8 (22%)	7 (19%)
Enzalutamide	0	1 (3%)
PSA Decline ≥ 30% with prior Abiraterone	33 (92%)	33 (92%0
CTC≥5 ml		
Median (Range)	2 (0-600)	2 (0-115)
≥5	15 (46%)	15 (48%)

Treatment Duration

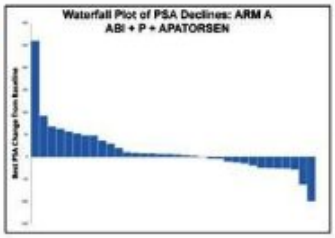
Status	ARM A (ABI + P + Apatorsen) (N=36)	ARM B (ABI + P) (N=36)
Median Treatment Duration, days (range)	196 (7-348)	74.5 (4-341)

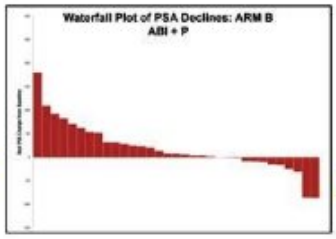
PRIMARY ENDPOINT: DISEASE PROGRESSION

	ARM A (ABI + P + Apatorsen) (N=36)	ARM B (ABI + P) (N=36)	P-value
Progression Free at Day 85 (+/- 7 days)	12 (33%)	6 (17%)	0.17
Progression Free Survival (weeks)	8.6 (95% CI, 4.43-10.14)	7.9 (95% CI, 4.57-9.00)	0.69

PSA DECLINE

Best PSA Decline from Baseline	ARM A (ABI + P + Apatorsen) (N=36)	ARM B (ABI + P) (N=36)	P-value
≥50%	2 (6%)	1 (3%)	1
≥30%	2 (6%)	2 (6%)	1
Any	13 (36%)	11 (31%)	0.62





MEASURABLE DISEASE RESPONSE

Best Response Category	ARM A (ABI + P + Apatorsen) (N=30)	ARM B (ABI + P) (N=30)
Evaluable for RECIST	33 (92%)	28 (82%)
Partial Response	1 (3%)	0
Stable Disease	5 (17%)	5 (17%)
Progressive Disease	26 (72%)	28 (82%)

CTC CHANGES

Best CTC Change from Baseline	ARM A (ABI + P + Apatorsen) (N=32)	ARM B (ABI + P) (N=33)
≥ 5 to < 5	8 (25%)	4 (12%)
< 5 to < 5	16 (50%)	15 (50%)
≥ 5 to ≥ 5	7 (22%)	10 (33%)
< 5 to ≥ 5	1 (3%)	1 (3%)

TREATMENT EMERGENT ADVERSE EVENTS

All grade 3-4 adverse events and grade 1-2 occurring in > 5 patients in either arm

Incidence of Adverse Events	ABI + P + Apatorsen (N=36) Grade		ABI + P (N=36) Grade	
Adverse Event	1-2	3-4	1-2	3-4
ANEMIA	7 (19%)	1 (3%)	10 (28%)	3 (8%)
URINARY RETENTION		1 (3%)	4 (11%)	
NEUTROPHIL COUNT DECREASED		1 (3%)		3 (8%)
ALT INCREASED		1 (3%)		1 (3%)
ACUTE KIDNEY INJURY		1 (3%)		
FATIGUE	18 (50%)	1 (3%)	11 (31%)	2 (6%)
BACK PAIN	11 (31%)	1 (3%)	5 (14%)	2 (6%)
VOMITING	8 (22%)	1 (3%)	3 (8%)	
HYPERTENSION	6 (17%)	1 (3%)	2 (<50%)	4 (11%)
FACIAL PAIN	7 (19%)	1 (3%)	3 (8%)	
DIARRHEA	7 (19%)	1 (3%)	2 (6%)	
HYPOXIA	5 (14%)	1 (3%)	1 (3%)	
PAIN IN EXTREMITY	5 (14%)	1 (3%)	0 (<3%)	2 (6%)
HYPONATREMIA		1 (3%)		2 (6%)
URINARY TRACT INFECTION		1 (3%)		2 (6%)
ASTHENIA		1 (3%)		1 (3%)
HYPERGLYCEMIA		1 (3%)		1 (3%)
PLATELET COUNT DECREASED		1 (3%)		1 (3%)
INFECTION - NOS		1 (3%)		1 (3%)
LUNG INFECTION		1 (3%)		
MUSCULOSKELETAL PAIN LOWER LIMB		1 (3%)		
SYNCOPE		1 (3%)		
ALT INCREASED		1 (3%)		
BLADDER SPASM		1 (3%)		
CREATININE INCREASED		1 (3%)		
CYSTITIS (CYSTITIS NOS)		1 (3%)		
URINARY TRACT OBSTRUCTION		1 (3%)		
VASOVAGAL REACTION		1 (3%)		
SWEATING		1 (3%)		
NAUSEA	18 (50%)		5 (14%)	
DYSPNEA	14 (39%)		5 (14%)	
FEVER	9 (25%)		1 (3%)	2 (6%)
PAIN	7 (19%)		5 (14%)	2 (6%)
HEADACHE	7 (19%)		5 (14%)	
INJECTION SITE	5 (14%)		2 (6%)	2 (6%)
PERIPHERAL SENSORY NEUROPATHY	5 (14%)		1 (3%)	
FALL	5 (14%)		1 (3%)	
COUGH	5 (14%)		0 (<3%)	
ANOREXIA	5 (14%)		1 (3%)	2 (6%)
CONSTIPATION	5 (14%)		0 (<3%)	
CHOLECYSTITIS, NOS	5 (14%)		1 (3%)	
URINARY FREQUENCY	5 (6%)		5 (14%)	
BRUISING	1 (6%)		5 (14%)	
DEHYDRATION				2 (6%)
HYPOTENSION				2 (6%)
ABDOMINAL PAIN				2 (6%)
ORAL INFLAMMATION				2 (6%)
BILIRUBIN INCREASED				2 (6%)
FEBRILE NEUTROPENIA				2 (6%)
HYPOALBUMINEMIA				2 (6%)
HYPOCALCEMIA				2 (6%)

CONCLUSIONS

- These data provide clinical evidence for the role of Hsp27 as a therapeutic target for prostate cancer
- Apatorsen may have activity when added to abiraterone for mCRPC patients progressing on abiraterone. Establishing predictors of response should be a priority for future studies.

Phase 2 Trial
Borealis-2
Investigating Apatorsen in Advanced Bladder Cancer in Combination With Second-Line Chemotherapy

ABSTRACT #289

Borealis-2: A randomized phase II study of OGX-427 (apatorsen) plus docetaxel versus docetaxel alone in platinum-resistant metastatic urothelial cancer (mUC)

TK Choueiri[1], N. Hahn[2], M. Regan[3], L. Weiner[4], A. Alva[5], S. George[6], J. Picus[7], R. Alter[8], A. Balar[9], J. Hoffman-Censits[9], P. Grivas[9], R. Lauer[10], E. Guancial[11], C. Homes[12], G. Sonpavde[13], C. Albany[14], M. Stein[15], C. Jacobs[16], P. Stewart[16], A. Lalani[1], S. Pal[17], JE Rosenberg[18]

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hoosier CANCER RESEARCH NETWORK

NCT 01780545

INTRODUCTION

- Heat shock protein 27 (Hsp27) is over-expressed in many malignancies including bladder cancer. Increased Hsp27 has been associated with inhibition of chemotherapy-induced apoptosis, increased tumor cytoprotection, and development of treatment resistance.
- Apatorsen (OGX-427), a 2nd generation antisense oligonucleotide that inhibits expression of Hsp27, has been shown to inhibit tumor growth and sensitize tumor cells to chemotherapy in a variety of malignancies, including urothelial cancer.
- Results of preclinical and phase 1 studies suggest that addition of apatorsen to chemotherapy is well tolerated and may improve treatment efficacy.
- **Borealis-2** is a randomized, multicenter, phase 2 study of apatorsen in combination with docetaxel (DOC) vs. DOC alone in locally advanced/metastatic bladder cancer patients who received at least one line of prior platinum-based therapy.

OBJECTIVES

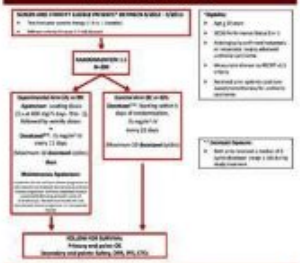
PRIMARY:

- Determine whether ODC administered in combination with apatorsen improves overall survival (OS) compared to DOC alone

SECONDARY:

- Safety and tolerability
- Compare overall response rate (ORR) and progression-free survival (PFS) rates between the arms
- Evaluate the effect of each arm on serum Hsp27 levels
- Evaluate the association of urothelial carcinoma expression of Hsp27 measured by IHC in archival tissue
- Evaluate the effect of therapy on peripheral blood circulating tumor cells (CTCs)

PATIENTS and METHODS



STATISTICAL DESIGN

- Assuming 298 participants enrolled, the trial design had 90% power to detect a 33% reduction in the OS hazard (hazard ratio (HR)=0.6427) corresponding to an approximately 50% improvement in median OS from 5 to 8 months, with one-sided α=0.28 significance level
- p-value <0.1 (one-sided) would result in a positive study for the primary endpoint of OS

RESULTS

Table 1. Baseline characteristics

Characteristics	Arm A: DOC + Apatorsen (n = 91)	Arm B: DOC (n = 101)
Age, median (range)	66 (45-82)	67 (35-82)
Sex		
Male	74.7%	74.3%
Female	25.3%	25.7%
ECOG		
0	43.4%	40.6%
1	56.6%	58.4%
Race		
White	89.9%	91.1%
Black/African American	3.0%	4.0%
Asian	5.1%	3.0%
Unknown	2.0%	2.0%
Urothelial carcinoma at study entry		
Metastatic	85.9%	88.1%
Locally-advanced	6.1%	9.9%
Unknown	8.1%	4.0%
Primary Surgery		
Yes	40.4%	35.6%
Prior Cisplatin use		
Yes	70.7%	71.3%
Prior Carboplatin use		
Yes	38.4%	40.6%
Primary disease site		
Bladder	64.5%	71.3%
Renal pelvis	27.3%	12.9%
Ureter	10.1%	13.9%
Urethra	7.1%	8.9%
Metastatic sites		
Liver	26.3%	24.8%
Lung	34.3%	34.7%
Bone	19.2%	20.8%
Lymph nodes	66.6%	61.5%
Bellmunt criteria		
0	27.3%	31.7%
1	42.4%	34.7%
2	23.2%	25.7%
3	7.1%	8.9%
Unknown	---	1.0%

Table 2. Response rates

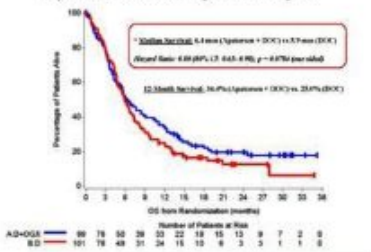
Response	Arm A: DOC + Apatorsen (n = 89)	Arm B: DOC (n = 101)
Complete response (CR)	1 (1%)	1 (1%)
Partial response (PR)	16 (18.1%)	13 (12.9%)
Stable Disease (SD)	21 (23.3%)	23 (24.8%)
Progressive Disease (PD)/NE	52 (57.7%)	65 (64.3%)

CR/PR response duration:

Apatorsen + DOC: median 4.2 months (95% CI 2.8 mo - NE)

DOC: median 4.6 months (95% CI 3.7 mo - 6.8 mo)

Figure 1. Primary Endpoint:
Kaplan-Meier curve of OS according to treatment assignment



Progression-free survival (PFS)
1.8 months for apatorsen + DOC vs 1.6 months for DOC
[HR 0.60 (0.44-1.01); one-sided p = 0.1069]

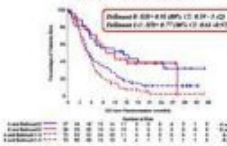
Table 3. Selected grade 3-5 treatment emergent adverse events

Selected ≥ Grade 3 AEs (assuming no SOC's w/ patients)	Arm A: Docetaxel + Apatorsen		Arm B: Docetaxel	
	N	%	N	%
Neutropenia	33	36.1	29	30.3
Anemia	16	17.2	12	12.5
Sepsis	14	15.1	5	5.2
Urinary tract infections (UTI)	13	13.8	7	7.3
Febrile neutropenia	10	10.8	8	8.3
Lymphopenia	11	11.8	7	7.3
Diarrhea	7	7.5	5	5.2
Hypertension	4	4.3	5	5.2
Dyspnea	5	5.4	4	4.1
General muscle weakness	4	4.3	4	4.2
Vomiting	4	4.3	3	3.1
Thromboembolic events	2	2.1	5	5.2
Safety population	93	100	96	100

Additional Analyses

Figure 2. Kaplan-Meier estimate of OS according to Stratification Factors and treatment assignment

Bellmunt Criteria risk factors



Time from prior systemic chemotherapy

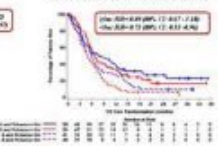
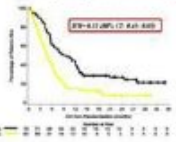


Figure 3. Kaplan-Meier estimate of OS according to baseline serum HSP27 levels (all subjects with baseline Hsp27 levels*)



*101 (80.5%) subjects had baseline Hsp27 levels available

Median baseline serum Hsp27 level 5.7ng/mL (Range: 0.5 – 46.5 ng/mL)

Table 4: Landmark Analysis for Changes in serum Hsp27 levels During Treatment: OS from end of Cycle 2 for treatment effect

% change in Hsp27* from baseline to Cycle 2	Treatment Assignment	N	Deaths	Median survival (mos)	HR	Lower 95%CL	Upper 95%CL	Interaction P
Decreased or ≤20.6% increase from baseline	DOC+apatorsen	22	14	12.2	0.29	0.13	0.48	0.013
	DOC	10	10	5.1				
>20.6% increased from baseline	DOC+apatorsen	10	11	7.9	0.77	0.48	1.30	
	DOC	22	18	6.8				

* Median % change in serum Hsp27 levels from baseline to the end of Cycle 2 was a 20.5% increase in Hsp27 levels

CONCLUSIONS

- **In this randomized phase 2 trial, docetaxel administered with apatorsen (OGX-427) was well tolerated and provided a survival benefit compared to docetaxel alone for patients with metastatic UC that are relapsed/refractory after receiving a platinum-containing regimen**
- **Survival benefit was numerically more apparent in patients treated with apatorsen who have 1 or more Bellmunt risk factors**
- **Higher baseline serum Hsp27 levels appear to be an independent prognostic indicator for shorter survival outcomes**
- **Further studies are warranted evaluating the potential of apatorsen to improve survival in patients being treated with metastatic UC**

REFERENCES: 1. Bellmunt et al. J Clin Oncol. 2009 Apr 1;8;15(10):1909-15.