# Public Disclosure Summary
# Study SP002

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**NAME OF SPONSOR**

SOTIO a.s., Jankovcova 1518/2, 170 00 Prague, Czech Republic

**NAME OF FINISHED TEST PRODUCT**

DCVAC/PCa

**NAME OF ACTIVE INGREDIENT(S)**

Autologous dendritic cells pulsed with killed prostate cancer cells and matured by a Toll-like receptor 3 ligand

**TITLE OF STUDY**

Randomized, open-label, parallel-group, multi-centre phase II clinical trial with active cellular immunotherapy DCVAC/PCa in combination with hormone therapy in patients with metastatic prostate cancer (protocol number SP002, EudraCT number 2011-004986-34)

**STUDY CENTERS**

A total of 18 clinical study centers in the Czech Republic participated in the study, and 14 recruited at least 1 patient onto the study.

**STUDY PERIOD**

**Study initiation date (first patient signed the Informed Consent Form):** 05-Mar-2012

**Completion of study main part:** 02-Nov-2015

**Study completion date:** 27-Jun-2016

**REPORTING PERIOD**

**From:** 05-Mar-2012 (first patient signed the Informed Consent Form)

**To:** 02-Nov-2015 (completion of study main part)

(the survival follow-up period after 02-Nov-2015 will be reported in an addendum to this Public Disclosure Summary)

**PHASE OF DEVELOPMENT**

II

**BACKGROUND AND RATIONALE**

Prostate cancer is primarily treated by surgery (radical prostatectomy) or radiotherapy. Unfortunately, in 27% to 53% of patients the disease relapses after primary curative treatment. Patients with relapsed or metastatic disease are treated with androgen deprivation therapy (ADT), which induces apoptosis of tumor cells. Median survival of patients with metastatic androgen-sensitive prostate cancer has been indicated to vary by number and location of bone metastases, presence/absence of visceral metastases, Gleason score, performance status, and initial prostate-specific antigen (PSA) and alkaline phosphatase values, but can be assumed to be 3.5 years in a broader group of patients as included in clinical studies.

A way to potentially enhance treatment outcomes in patients with metastatic castration-sensitive prostate cancer might be to add immunotherapy to the standard ADT. Cancer immunotherapy can employ various immune system components to combat cancer. In this study, autologous dendritic cells (DCs) were used. The test product DCVAC/PCa is an active cellular immunotherapy which consists of autologous DCs activated by *ex vivo* exposure to killed cells of the prostate cancer cell line LNCaP. The potency of DCVAC/PCa and its biological activity were characterized by extensive *in vitro* studies. These studies demonstrated that DCVAC/PCa possesses the capacity to present exogenous antigens of apoptotic tumor cells on major histocompatibility complex class I and II molecules and can activate both $CD4^+$ (helper) and $CD8^+$ (cytotoxic) tumor-specific T lymphocytes. We hypothesized that this immune response might impact the viability of the tumor and thereby prolong the time until further progression of the disease and improve overall survival. To test this hypothesis, several clinical trials with DCVAC/PCa in different settings were set up, including the current trial.

To enhance the effect of DCVAC/PCa, the patients received the immunomodulators cyclophosphamide and imiquimod. Cyclophosphamide inhibits regulatory T lymphocytes, which are involved in the induction of the immune system's tolerance of tumors; and imiquimod, when applied to the skin, augments local maturation of DCs.

**OBJECTIVES**

The objectives of this clinical trial were to estimate the proportion of patients with PSA and disease progression within 2 years of randomization; to evaluate quality of life and pain scale scoring using the standardized European Organization for Research and Treatment of Cancer Quality of Life Questionnaire (EORTC QLQ)-C30 questionnaire, version 3; and to evaluate the incidence of adverse events (AEs; with the exception of disease progression-related AEs) and overall survival.

An exploratory objective was to search for potential biomarkers that could play a role as prognostic factors, indicate the biological effect of active cellular immunotherapy on immune response, or identify a subgroup of patients benefiting from cancer immunotherapy based on gene expression profiling.

## ENDPOINTS

**Primary endpoint:**

- Proportion of patients with PSA progression within 2 years after randomization

**Secondary endpoints:**

- Proportion of patients with disease progression within 2 years after randomization
- Incidence of AEs
- Overall survival
- Quality of life as per the standardized EORTC QLQ-C30 v3 questionnaire
- Pain scale influence scoring as per the standardized EORTC QLQ-C30 v3 questionnaire

**Exploratory endpoints:**

- Immune response
- Gene expression profiling and/or expression levels of a defined set of immune- or cancer-related genes

## METHODOLOGY

This was a randomized, open-label, parallel-group, multicenter phase II clinical trial to evaluate the efficacy and safety of DCVAC/PCa in patients with histologically confirmed metastatic prostate cancer who started ADT with luteinizing hormone-releasing hormone (LHRH) analogs or underwent orchiectomy 1 to 3 months before screening.

Patients were evaluated for eligibility within 2 weeks (±2 weeks) and centrally randomized to 2 groups at a ratio of 1:1 to receive DCVAC/PCa in combination with ADT (immunotherapy group) or ADT alone (control group). Patients in both groups who had not undergone orchiectomy continued with LHRH analogs in accordance with the Summary of Product Characteristics. The target was to randomize a total of 60 patients.

Patients randomized to the immunotherapy group underwent leukapheresis within 3 weeks of randomization. To modulate their immune system, the patients in the immunotherapy group were treated with 50 mg/day of oral cyclophosphamide for 7 days before the first dose of DCVAC/PCa and applied imiquimod cream to the planned DCVAC/PCa injection sites 24 hours before each DCVAC/PCa administration. DCVAC/PCa (approximately $1\times10^7$ autologous DCs) was administered at each dosing time to the patients from the immunotherapy group subcutaneously in up to 10 doses. The first dose of DCVAC/PCa was administered 4 weeks (±1 week) after leukapheresis, the second dose was administered 2 weeks (±1 week) after the first dose, and subsequent doses were administered 4 weeks (±1 week) apart.

Patients randomized to the control group received only ADT; placebo was not used. These patients did not undergo leukapheresis, and they were not treated with cyclophosphamide or imiquimod. Study visits of the patients in the control group were identical to the visits of the patients in the immunotherapy group. The treatment phase ended with Visit (V) 10/End of treatment (EOT). For patients in the immunotherapy group, the EOT was the last dose of DCVAC/PCa; for the patients in the control group, the EOT was the last visit in the treatment phase.

After the end of the treatment phase, the patients were followed up at 3-month intervals (±2 weeks) at Follow-up visits until 2 years after randomization. Patients randomized to the immunotherapy group who terminated their participation before receiving the first dose of DCVAC/PCa underwent the End of study (EOS) visit and were not monitored in the study any further. All other patients underwent the EOS visit 2 years after randomization or within 4 weeks after the decision about premature termination of the participation in the trial was made, if permitted by the condition of the patient and if the patient agreed to come for the visit.

If approved in writing, data about PSA progression and survival were to be collected after the EOS visit every 3 months until the patient's death or study termination by contacting the patient or the patient's family members or attending physician (survival follow-up).

The main part of the study was terminated at the time of the EOS visit of the last patient on 02-Nov-2015. All data available at this cut-off date were analyzed and are presented in this Public Disclosure Summary. The study as such continued to collect data about PSA progression and survival of study patients, and it was terminated by the sponsor on 27-Jun-2016.

This study was not blinded. However, following the evaluation of disease progression at the investigational sites, all computed tomography (CT) and bone scintigraphy scans were to be submitted for blinded central reading by independent radiologists.

## NUMBER OF PATIENTS

**Planned:** 60

**Screened:** 74

**Randomized:** 63

**Analyzed for efficacy:** 63

**Analyzed for safety:** 61

## DIAGNOSIS AND CRITERIA FOR INCLUSION AND EXCLUSION

Patients with metastatic prostate cancer who met all inclusion criteria and none of the exclusion criteria were eligible for this clinical trial.

**Inclusion criteria:**

Inc-1) Men aged ≥18 years

Inc-2) Histologically confirmed prostate carcinoma

Inc-3) Bone or soft-tissue (other than brain or leptomeningeal) metastases confirmed by CT of the abdomen and lesser pelvis and/or bone scintigraphy

Inc-4) ADT with LHRH analogs started not less than 1 month and not more than 3 months prior to screening, or orchiectomy performed not less than 1 month and not more than 3 months prior to screening

Inc-5) Eastern Cooperative Oncology Group (ECOG) 0-2 Performance Status

Inc-6) The following laboratory values: white blood cell count $>4\times10^9$/L; absolute neutrophil count $\geq1.5\times10^9$/L; platelet count $>100\times10^9$/L; hemoglobin ≥90 g/L; hematocrit >30%; creatinine under 1.5 times the upper limit of normal; bilirubin, aspartate transaminase, and alanine transaminase under 1.5 times the upper limit of normal

Inc-7) Castrate level of serum testosterone at screening (≤1.7 nmol/L or ≤50 ng/dL)

Inc-8) Signed informed consent to participate in the study

**Exclusion criteria:**

E-1) Sexually active fertile men not using effective birth control, if their partners are of fertile age and of child-bearing potential

E-2) Comorbidities of the patient:

- E-2a) HIV-positive
- E-2b) Active hepatitis B or C
- E-2c) Active bacterial, viral, or mycotic infection requiring systemic treatment
- E-2d) Clinically significant cardiovascular disease, including myocardial infarction or ventricular tachyarrhythmia in previous 6 months, percutaneous coronary intervention or surgical revascularization in the past 6 months, heart failure New York Heart Association Functional classification (NYHA) II-IV, known left ventricular dysfunction with ejection fraction <40%, or hemodynamically significant arrhythmias or conduction problems (unless treated with permanent cardiac pacing)
- E-2e) Pleural or pericardial effusion of any Common Terminology Criteria (CTC) grade
- E-2f) Peripheral neuropathy CTC Grade ≥2
- E-2g) Other uncontrolled coexisting condition, uncompensated psychiatric illness or social situation that would limit the patient's compliance
- E-2h) Patients with a history of second malignancy other than non-melanoma skin cancer
- E-2i) Unresolved ongoing clinically significant urinary tract obstruction
- E-2j) Active autoimmune disease requiring therapy
- E-2k) History of primary immunodeficiency

E-3) Allergy and adverse drug reactions

- E-3a) History of allergic reaction to a compound of the same or similar structure as medication used in this study
- E-3b) History of anaphylaxis or other severe reaction following vaccination

E-4) Any other serious reason the investigator considers the patient should not participate in the study

E-5) History of or ongoing chemotherapy for prostate cancer

E-6) Patients with brain metastases or leptomeningeal metastases

E-7) Ongoing unresolved post-operative complication of orchiectomy, which either requires treatment or restricts the patient's everyday activities

E-8) Participation in another clinical trial or administration of another investigational medicinal product (IMP) within 30 days preceding screening

E-9) Immunotherapy other than specified in this Protocol

E-10) Prohibited concomitant medication

**TEST PRODUCT, DOSE AND MODE OF ADMINISTRATION, BATCH NUMBER**

**Test product:**

DCVAC/PCa, an active cellular immunotherapy product containing DCs activated by exposure to killed tumor cells of the prostate cancer cell line LNCaP and matured by the Toll-like receptor 3 ligand poly(I:C).

**Dose and mode of administration:**

An aliquot of 5 mL of cell suspension containing approximately $1\times10^7$ autologous DCs was divided into 2 injections (2.5 mL each) that were applied subcutaneously to the inguinal and axillary regions.

**Batch number:**

DCVAC/PCa was prepared on an individual basis, each product with a unique lot number.

**DURATION OF TREATMENT WITH TEST PRODUCT**

Up to 10 doses of DCVAC/PCa were administered during 34 weeks to patients randomized to the immunotherapy group.

**REFERENCE THERAPY, DOSE AND MODE OF ADMINISTRATION, BATCH NUMBER**

Not applicable.

**DURATION OF REFERENCE THERAPY**

Not applicable.

**STATISTICAL METHODS**

Randomized patients were allocated to trial populations for analysis purposes. Intention-to-treat population (ITT) consisted of all randomized patients. The Per protocol set (PPS) was a subset of the ITT population without patients with a significant protocol deviation and patients who did not undergo any treatment visit. The Safety population consisted of all randomized patients who underwent at least V1 (control group) or received at least 1 dose of DCVAC/PCa (immunotherapy group).

No interim analysis was planned or performed for this trial.

Demographic and other baseline characteristics were summarized and presented by treatment group.

The analysis of the primary endpoint, i.e., the proportion of patients with PSA progression within 2 years after randomization, was based on time to PSA progression. PSA progression was defined as at least a 50% increase in PSA above nadir and above 0.2 ng/mL at the same time. This increase had to be confirmed by 2 consecutive tests, each at an interval of at least 2 weeks and no more than 3 months from the previous test, and none of the confirming tests had to show more than a 10% drop in PSA versus the baseline, all with castrate testosterone levels ≤1.7 nmol/L (or 50 ng/dL). If for a patient no PSA assessment was identified as confirmed PSA progression, the earliest non-confirmed PSA progression was selected. Data from such a patient were reviewed by a medical expert, who decided whether this non-confirmed PSA progression was considered as PSA progression in the analysis. The primary analysis was performed using the ITT population and data after medical review. For the time-to-event analysis, a survival function in each treatment group was estimated by the Kaplan-Meier method. A 2-sided log-rank test at a 5% significance level was used to test the null hypothesis regarding the equivalence of the survival functions. Summary statistics based on the Kaplan-Meier analysis were determined, including median time to PSA progression and the proportions of patients without progression at 2 years after randomization, along with 95% confidence intervals. An estimate of the hazard ratio with a 95% confidence interval was derived from a Cox proportional hazard regression model. Sensitivity analyses were performed on the ITT population using PSA data which were not reviewed by a medical expert and on the PPS using medically reviewed PSA data. The effect of baseline factors was explored by a Cox regression model.

For each tumor assessment, a medical expert determined whether disease progression occurred or not based primarily on reports prepared by independent radiologists. The proportion of patients with disease progression within 2 years after randomization was defined as the number of patients who progressed divided by the number of patients who progressed plus the number of patients who were progression free at 2 years. The proportions were presented with an exact 95% confidence interval together with their difference. The proportions were compared by Fisher's exact test at a 5% significance level.

Overall survival was analyzed in the same way as time to PSA progression.

For all scales and items of the EORTC QLQ-C30 v3 questionnaire, mean profiles for each treatment group were graphically presented.

The relationship between DCVAC/PCa treatment and the immune response was assessed by 3 exploratory immunomonitoring assays. Immunomonitoring assay 1A evaluated the induction of helper ($CD4^+$) and cytotoxic ($CD8^+$) T lymphocytes specific to the tumor antigens PSA, prostatic acid phosphatase, prostate-specific membrane antigen, melanoma-associated antigen (MAGE)-A1, MAGE-A3, and human epidermal growth factor receptor 2. Immunomonitoring assay 1B evaluated the induction of specific antibodies against the tumor antigens PSA and MAGE-A3. Immunomonitoring assay 1D evaluated the percentage of myeloid-derived suppressor cells and regulatory T lymphocytes and the expression of immune check point inhibitors on helper and cytotoxic T lymphocytes.

AEs were coded using the Medical Dictionary for Regulatory Activities version 18.1. Severity or intensity of an AE was assessed according to the National Cancer Institute's Common Terminology Criteria for Adverse Events version 4.03. Treatment-emergent AEs (TEAEs) were AEs that started or worsened during the period starting at V1 and ending 30 days after the EOT. Data with respect to the number of patients who underwent leukapheresis and had complications during leukapheresis were summarized and listed. The following types of laboratory data were analyzed: hematology, biochemistry, urine, PSA, humoral and cellular immunity, thyroid gland function, 25-OH vitamin D3, testosterone, serology, and coagulation. Measurements of vital signs were summarized. Data regarding ECOG, physical examination, electrocardiogram (ECG), and chest X-ray were listed.

**SUMMARY OF RESULTS AND CONCLUSIONS**

**Patient disposition:**

A total of 74 patients were screened in this study. Of the screened patients, 63 (85.1%) were randomized and 11 (14.9%) were screening failures. Of the 63 randomized patients, 32 (50.8%) were randomized to the immunotherapy group (DCVAC/PCa + ADT) and 31 (49.2%) were randomized to the control group (ADT).

A total of 15 patients (46.9%) randomized to the immunotherapy group received all 10 doses of DCVAC/PCa. One patient in the immunotherapy group did not undergo leukapheresis and did not receive DCVAC/PCa, cyclophosphamide, or imiquimod. All DCVAC/PCa administrations were done within the protocol-specified time window.

Of the 31 patients exposed to DCVAC/PCa, 29 (93.5%) received all 7 doses of cyclophosphamide while 2 patients (6.5%) received 6 doses of cyclophosphamide, and 27 patients (87.1%) applied imiquimod cream as defined by the Protocol while 4 patients (12.9%) did not apply imiquimod cream before DCVAC/PCa administration according to the Protocol.

Adherence to ADT was determined indirectly by review of on-study testosterone levels. A total of 3 of 32 patients (9.4%) in the immunotherapy group and 1 of 31 patients (3.2%) in the control group had non-castrate testosterone levels while on study.

Two protocol violations were reported in 2 of the 63 randomized patients (3.2%; violations of the inclusion criterion Inc-

6). These reported protocol violations occurred in the immunotherapy group and did not lead to treatment discontinuation or premature termination of the patients' participation in the trial.

**Patient demographics and baseline characteristics:**

Despite randomization, the resulting patient groups were slightly imbalanced with regard to disease history. Clinical staging and Gleason scores at diagnosis indicated that the immunotherapy group included patients diagnosed with more advanced disease as compared to the control group (Table 1).

**Table 1: Disease characteristics at diagnosis, ITT population**

| | DCVAC/PCa + ADT N = 32 | ADT N = 31 |
|---|---|---|
| Clinical stage, n (%) | | |
| Stage I | 0 (0) | 4 (12.9) |
| Stage II | 5 (15.63) | 2 (6.45) |
| Stage III | 5 (15.63) | 2 (6.45) |
| Stage IV | 22 (68.75) | 23 (74.19) |
| Gleason score, n (%) | | |
| 3 | 0 (0) | 1 (3.23) |
| 6 | 0 (0) | 2 (6.45) |
| 7 | 11 (34.38) | 10 (32.26) |
| 8 | 6 (18.75) | 6 (19.35) |
| 9 | 11 (34.38) | 10 (32.26) |
| 10 | 4 (12.5) | 1 (3.23) |
| Unknown | 0 (0) | 1 (3.23) |

Demographic and baseline characteristics were similar between the treatment groups (Table 2).

**Table 2: Summary of patient demographics and baseline characteristics, ITT population**

| | DCVAC/PCa + ADT N = 32 | ADT N = 31 |
|---|---|---|
| Age, median years (min, max) | 66.09 (40.64, 77.65) | 66.89 (57.28, 80.38) |
| Weight, median kg (min, max) | 82.5 (52.0, 104) | 92 (56.1, 130) |
| Height, median cm (min, max) | 174.5 (160, 185) | 178 (164, 192) |
| ECOG score, n (%) | | |
| 0 | 22 (68.75) | 22 (70.97) |
| 1 | 10 (31.25) | 7 (22.58) |
| 2 | 0 (0) | 2 (6.45) |
| First-line therapy, n (%) | | |
| ADT | 26 (81.25) | 24 (77.42) |
| Radical prostatectomy | 5 (15.63) | 2 (6.45) |
| Radiotherapy | 1 (3.13) | 5 (16.13) |
| ADT before screening, n (%) | | |
| LHRH analog | 21 (65.63) | 18 (58.06) |
| Orchiectomy | 14 (43.75) | 14 (45.16) |

**Efficacy results:**

None of the main efficacy analyses showed any statistically significant differences between the treatment groups. The primary analysis of time to PSA progression estimated that the proportion of patients with PSA progression within 2 years after randomization was numerically higher in the immunotherapy group than in the control group (93.9% vs 77.9%). However, the difference was not statistically significant (Table 3).

**Table 3: Primary analysis of time to PSA progression and proportion of patients with PSA progression within 2 years after randomization, ITT population, data after medical review of PSA progression**

| | DCVAC/PCa + ADT N = 32 | ADT N = 31 |
|---|---|---|
| Median time to PSA progression (months; 95% CI) | 6.39 (3.70, 12.23) | 9.34 (6.43, 13.41) |
| Censored, n | 10 | 11 |
| Events, n | 22 | 20 |
| Log-rank test (p-value) | p = 0.2018 | |
| Cox regression, hazard ratio (95% CI; p-value) | 1.488 (0.806, 2.745; p = 0.2035) | |
| Proportion of patients with PSA progression within 2 years after randomization (95% CI) | 0.939 (0.765, 0.996) | 0.779 (0.599, 0.917) |

The results of the sensitivity analyses were directionally consistent with the results observed in the primary analysis; they showed a longer median time to PSA progression in the control group. The analysis based on the ITT population including

data without medical review of PSA progression did not show any statistically significant difference between the treatment groups; however, the analysis based on the PPS indicated a trend towards a difference (median time to PSA progression in the immunotherapy group 3.8 months and in the control group 9.3 months; p = 0.05, log-rank test).

The proportion of patients with disease progression within 2 years after randomization was comparable in both treatment groups, as were overall survival and scores of all items of the EORTC QLQ-C30 v3 questionnaire.

**Exploratory results:**

Exploratory analyses of parameters of cellular and humoral immunity did not detect any positive statistically significant effect of DCVAC/PCa treatment on the immune response on a group level. The analyses related to gene expression profiling and/or expression levels were not performed because no immune response to DCVAC/PCa was detected, and it was not possible to identify patients that would benefit from DCVAC/PCa treatment.

**Safety results:**

The safety of DCVAC/PCa was benign. Table 4 shows the incidence of TEAEs by PT and decreasing frequency in the immunotherapy group for events occurring in ≥5% of patients in either treatment group.

**Table 4: Incidence of TEAEs occurring in ≥5% of patients in either treatment group, safety population**

| Preferred term | DCVAC/PCa + ADT N = 31 n (%) | ADT N = 30 n (%) | Fisher's exact test (p-value) |
|---|---|---|---|
| Back pain | 4 (12.9) | 3 (10) | 1.0000 |
| Urinary tract infection | 3 (9.68) | 3 (10) | 1.0000 |
| Constipation | 3 (9.68) | 0 (0) | 0.2377 |
| Urinary retention | 3 (9.68) | 1 (3.33) | 0.6124 |
| Arthralgia | 2 (6.45) | 2 (6.67) | 1.0000 |
| Hypertension | 2 (6.45) | 2 (6.67) | 1.0000 |
| Pyrexia | 2 (6.45) | 0 (0) | 0.4918 |
| Bone pain | 1 (3.23) | 2 (6.67) | 0.6124 |
| Oedema peripheral | 1 (3.23) | 2 (6.67) | 0.6124 |
| Paraesthesia | 0 (0) | 2 (6.67) | 0.2377 |

The difference in the overall incidence of TEAEs was not statistically significant when comparing the treatment groups (p = 0.8385, Fisher's exact test).

The treatment groups were comparable with regard to the severity of TEAEs (35.5% of patients in the immunotherapy group and 30% of patients in the control group experienced a severe, life-threatening, or fatal TEAE). The majority of deaths in both treatment groups were attributed to disease progression as the underlying cause (81.3% in the immunotherapy group, 80% in the control group).

Table 5 shows the incidence of serious TEAEs by PT and decreasing frequency in the immunotherapy group.

**Table 5: Incidence of serious TEAEs, safety population**

| Preferred term | DCVAC/PCa + ADT N = 31 n (%) | ADT N = 30 n (%) |
|---|---|---|
| Urinary tract infection | 2 (6.45) | 0 (0) |
| Acute respiratory failure | 1 (3.23) | 0 (0) |
| Cerebral ischaemia | 1 (3.23) | 0 (0) |
| Chronic obstructive pulmonary disease | 1 (3.23) | 0 (0) |
| Lower limb fracture | 1 (3.23) | 0 (0) |
| Pneumonia | 1 (3.23) | 0 (0) |
| Urinary tract obstruction | 1 (3.23) | 0 (0) |
| Vocal cord thickening | 1 (3.23) | 0 (0) |
| Peripheral ischaemia | 0 (0) | 1 (3.33) |
| Syncope | 0 (0) | 1 (3.33) |
| Urinary retention | 0 (0) | 1 (3.33) |

The number of patients with serious TEAEs was comparable between the treatment groups (p = 0.3006; Fisher's exact test).

No AE or serious AE was reported to be related to DCVAC/PCa or led to DCVAC/PCa discontinuation. No patient had any complications during leukapheresis. No notable differences were observed between the treatment groups with regard to the occurrence of clinically significant laboratory or physical examination findings; the measurements of vital signs, ECOG performance status, and ECG; or the use of concomitant medications.

**Conclusions:**

In this study of 63 patients with androgen-sensitive metastatic prostate cancer, the efficacy of DCVAC/PCa added to ADT was not statistically significantly different from the efficacy of ADT alone. Treatment with DCVAC/PCa did not result in meaningful changes in the tested cellular or humoral immunity parameters. Based on the data obtained in this study, DCVAC/PCa has a favorable safety profile.

**DATE AND VERSION OF THIS PUBLIC DISCLOSURE SUMMARY**

Version 1.0, 26-Jan-2017