A Randomized Phase II Trial of Sipuleucel-T with Concurrent versus Sequential Abiraterone Acetate plus Prednisone in Metastatic Castration-Resistant Prostate Cancer

Eric J. Small1, Raymond S. Lance2, Thomas A. Gardner3, Lawrence I. Karsh4, Lawrence Fong1, Candice McCoy5, Todd DeVries5, Nadeem A. Sheikh5, Debraj GuhaThakurta5, Nancy Chang5, Charles H. Redfern6, and Neal D. Shore7

Abstract

Purpose: This phase II open-label study evaluated the effect of concurrent or sequential administration of abiraterone acetate plus prednisone (AA + P) on sipuleucel-T manufacture and immune responses in metastatic castration-resistant prostate cancer (mCRPC) patients.

Experimental Design: mCRPC patients received sipuleucel-T followed by AA + P 1 day (concurrent) or 10 weeks (sequential) after the first sipuleucel-T infusion. AA + P treatment continued for 26 weeks. The primary endpoint was cumulative antigen presenting cell (APC) activation, and secondary endpoints included cumulative APC number and total nucleated cell counts. Additional endpoints included in vivo peripheral immune responses to sipuleucel-T (T-cell responses, T-cell proliferation, humoral responses, and antigen spread) as well as safety.

Results: Sixty-nine mCRPC patients were enrolled, with 35 and 34 patients randomized to the concurrent and sequential arms, respectively. Ex vivo APC activation was significantly greater at the second and third infusions compared with baseline in both arms (P < 0.05), indicative of an immunologic prime-boost effect. In both arms, sipuleucel-T product parameter profiles and peripheral immune responses were consistent with previously conducted sipuleucel-T phase III trials. Antigen spread was similarly observed in both arms and consistent with the other immunologic endpoints.

Conclusions: These data suggest that sipuleucel-T can be successfully manufactured during concurrent administration of AA + P without blunting immunologic effects or altering immune parameters that correlate with sipuleucel-T’s clinical benefit. Combination of these agents was well tolerated, with no new safety signals emerging.

Introduction

An expanding array of new treatments for metastatic castration-resistant prostate cancer (mCRPC), including radiopharmaceuticals, androgen signaling inhibitors, cytotoxic chemotherapy, and immunotherapeutics, has improved patient outcomes. However, determining the most appropriate sequence or combination of such agents for optimal and possibly synergistic clinical outcomes has become a major challenge for physicians. Similarly, it is not known if there are treatment combinations to avoid; limited data are available to inform such decisions.

An important potential therapeutic approach is the combined use of sipuleucel-T and abiraterone acetate (AA), and both these agents are commercially available for use in similar patient populations. These agents have presumed nonoverlapping mechanisms of action and toxicities. Thus, combining these agents might have additive clinical benefits without increasing toxicities. Furthermore, the sipuleucel-T treatment effect appears to develop over time, and although data suggest it does impact the natural progression of the disease (1), it typically does not show immediate effects on traditional measures of disease progression (DP; i.e., prostate-specific antigen [PSA] or radiographic response; refs. 2 and 3). Therefore, because of this nonmeasurable onset of action, patients may experience radiographic or PSA progression following sipuleucel-T treatment. Using abiraterone in combination with sipuleucel-T would yield more immediate antitumor effects and thus allow time for the sipuleucel-T antitumor response to develop. Furthermore, suppression of testosterone has been demonstrated to have immunostimulatory properties (4–9). Thus, reduction of testosterone levels with AA plus prednisone (AA + P) may enhance the immunologic effects of sipuleucel-T. To date, because AA requires the coadministration of...
Translational Relevance

Determining the most appropriate sequence or combination of agents for optimal and possibly synergistic clinical outcomes has become a major challenge for physicians managing prostate cancer. A potential combination approach is abiraterone acetate and sipuleucel-T. Nonoverlapping mechanisms of action and toxicities make them attractive agents for combinatorial use, with abiraterone acetate exerting an immediate antitumor effect and sipuleucel-T promoting a sustained immunologic and antitumor effect. We demonstrate that the combination of these agents did not appear to alter immune parameters known to correlate with the clinical benefit observed with sipuleucel-T. Prednisone, which is coadministered with abiraterone, did not appear to impair the manufacture of sipuleucel-T or blunt the peripheral immune responses generated from treatment. Although long-term follow-up for overall survival needs to be evaluated, the combination of an immunotherapy, such as sipuleucel-T, combined with a novel, oral oncolytic therapy, such as abiraterone acetate, constitutes an important therapeutic approach.

Patients and Methods

Patients

Men ≥18 years old with asymptomatic or minimally symptomatic metastatic prostate cancer and castrate level testosterone (≤50 ng/dL) were eligible if they met the following criteria: current or historical evidence of DP as demonstrated by PSA progression (as defined by the Prostate Cancer Clinical Trials Working Group; ref. 18) or the progression of measureable or nonmeasurable disease or bone disease; serum PSA ≥2.0 ng/mL; baseline Eastern Cooperative Oncology Group (ECOG) performance status ≤1; and adequate baseline hematologic, renal, and liver function. Patients with known lung, liver, or brain metastases, malignant pleural effusions, or malignant ascites were inelligible, as were men with known adrenocortical insufficiency, uncontrolled hypertension, New York Heart Association Class III or IV congestive heart failure, myocardial infarction or ventricular or atrial arrhythmia within 6 months prior to registration, or Child-Pugh Class B or C hepatic insufficiency. Patients previously treated with sipuleucel-T, AA, ipilimumab, any investigational vaccine or immunotherapy, systemic corticosteroids within 60 days, or any chemotherapy were ineligible. This phase II, multicenter, open-label study was conducted in accordance with applicable regulations of the Food and Drug Administration and the Good Clinical Practice guidelines of the International Conference on Harmonization. The study was approved by the institutional review board at each study center. Patients provided written informed consent before participation.

Treatment

Potential patients were screened and if eligible were registered and randomized 1:1 into either the concurrent arm or the sequential arm (see study schematic; Supplementary Fig. S1).

Study treatment consisted of sipuleucel-T and AA (1,000 mg once daily) + P (5 mg twice daily). Patients in both arms underwent a standard leukapheresis followed approximately 3 days later by an intravenous sipuleucel-T infusion. A complete treatment course consisted of 3 sipuleucel-T infusions, administered approximately every 2 weeks. In the concurrent arm, AA + P treatment began 1 day after the first sipuleucel-T infusion. The AA + P therapy was initiated after the first sipuleucel-T infusion so that the first sipuleucel-T product could serve as a control, compared with sipuleucel-T manufactured during AA + P administration. In the sequential arm, AA + P treatment began at week 10, 6 weeks after the last planned infusion of sipuleucel-T. In both arms, AA + P treatment continued for 26 weeks or until DP, unacceptable toxicity, or death, whichever occurred first. All patients were followed for changes in serum PSA from screening until 30 days after the last dose of study treatment. After completion of the 26-week treatment period, continued AA + P therapy in either arm was permitted at the treating physician’s discretion.

Immunologic endpoints

Product parameters, defined as antigen-presenting cell (APC) activation, APC number, and total nucleated cell (TNC) count, were determined for every sipuleucel—T product (ex vivo endpoints), as previously described (ref. 11; Supplementary Fig. S2). APCs were defined as large cells expressing CD54. APC activation was measured as the increase in surface CD54 on APCs and expressed as a upregulation ratio of the average number of molecules on post-culture versus pre-culture cells (19). The primary endpoint of this trial was to evaluate cumulative APC activation, an important ex vivo measure of potency, in sipuleucel-T products for each patient. Cumulative APC activation, as well as APC number and TNC count, was defined as the sum of the values across the three treatment doses. Secondary endpoints, also reflecting ex vivo immune activation, were cumulative APC number and cumulative TNC count from manufactured products from each patient. Secondary endpoints also included in vivo measures of PA2024 (a recombinant fusion protein composed of prostatic acid phosphatase [PAP] linked to granulocyte–macrophage colony stimulating factor)-specific and PAP-specific peripheral immune responses, including T-cell

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responses as measured by IFN\textgamma enzyme-linked immunosorbent assay (ELISPOT) assay, T-cell proliferation as measured by \textsuperscript{3}H-thymidine uptake, and humoral responses as measured by enzyme-linked immunosorbent assay (ELISA; serum IgG-IgM levels; ref. 11).

The assessment of antigen spread, as measured by the development of IgG responses against nontargeted secondary antigens (12–14), was not a prespecified endpoint of this trial and represents an ad hoc analysis. Induction of IgG responses to secondary antigens (PSA [KLK3, KLK2, K-RAS, E-RAS, LGALS3, and LGALS8]) has been previously demonstrated after treatment with sipuleucel-T (15–17). Serum IgG levels pre- and posttreatment against these secondary antigens were evaluated by Life Technologies Corporation using Luminex xMAP from all available pre- and posttreatment serum sample pairs from STAMP patients as previously described (15–17). The change in the IgG levels to the above antigens, from baseline to posttreatment, was compared between the two arms.

Blood samples for measuring peripheral immune responses (including antigen spread) were collected at baseline (screening), prior to each leukapheresis, after each sipuleucel-T infusion, and at weeks 6, 10, 14, and 26 after treatment initiation (see Supplementary Fig. S1).

PSA endpoints

PSA endpoints included the maximal decrease of serum PSA levels and the percentage of patients with a ≥50% decrease of serum PSA levels, relative to the last PSA level obtained prior to the administration of AA + P, which served as a baseline PSA measure because sipuleucel-T was not expected to appreciably impact PSA.

Safety

The safety population included all randomized patients who underwent at least 1 leukapheresis. Safety assessments were performed in both arms every 2 weeks throughout the treatment period, including adverse event (AE) monitoring, laboratory tests (complete blood count, serum chemistry, and liver function), and physical examinations. AEs were summarized and listed by treatment arm per the Medical Dictionary for Regulatory Activities (MedDRA) preferred terms within each system organ class.

Statistical considerations

The primary endpoint of the study was cumulative APC activation. The study had approximately 85% power to detect a fold change of 1.3 for the ratio of cumulative APC activation means between the arms assuming a coefficient of variation (CV) of 0.325. A 1.3-fold increase in cumulative APC activation was deemed to be clinically relevant as it would translate into a median fold change of 1.3 for the ratio of cumulative APC activation means between the arms assuming a CV of 1.25. Secondary endpoints included cumulative APC number, cumulative TNC, PA2024- and PAP-specific peripheral immune responses over time, and frequency of immune responders (yes or no). Immune response variables (including change from baseline of IgG levels against the secondary antigens related to antigen spread) over time were analyzed using a repeated measures model with terms for treatment group, visit, and treatment by time interaction. Immune response variables were log transformed prior to analysis except for IFN\textgamma ELISPOT responses, which were analyzed on a rank scale. Positive thresholds for treatment-related immune responses were selected to ensure that ≤5% would exceed the value at baseline. Differences in T cell and antibody responder frequencies were compared between the arms at each visit using the Fisher exact test. The exploratory endpoint of maximal PSA decrease from baseline (prior to initiation of AA + P) was compared between arms using a Wilcoxon test. The ad hoc endpoint of the percentage of patients with a ≥50% decrease in PSA was compared using the Fisher exact test. All P values reported are two-tailed. No adjustment for multiplicity of endpoints or time points was made.

Results

Patients and treatment

Sixty-nine mCRPC patients were enrolled between December 2011 and February of 2013, with 35 patients randomized to the concurrent arm and 34 patients to the sequential arm. Baseline demographics and disease characteristics are shown in Table 1. All 69 patients underwent at least 1 leukapheresis (safety population; Supplementary Fig. S3).

Of the 35 patients randomized to the concurrent arm, 33 (94.3%) received all 3 infusions of sipuleucel-T, and 2 (5.7%) patients received only 1 infusion. One patient received only 1 infusion and was subsequently removed from the study because of DP. A second patient received only 1 infusion, but after 2 failed attempts to manufacture product (due to insufficient TNC), the decision was made to discontinue. Of the 34 patients randomized to the sequential arm, 33 (97.1%) received all 3 infusions of sipuleucel-T, and 1 (2.9%) patient refused the third infusion because of a catheter-related infection.

Twenty-eight patients (80%) in the concurrent arm and 22 (65%) in the sequential arm completed 26 weeks of AA + P (P = 0.19). Nineteen patients did not receive the full 26 weeks of AA + P. Of these patients, 14 did not complete AA + P because of DP (concurrent arm n = 5; sequential arm n = 9). The remaining 5 patients did not complete AA + P because of AEs (n = 2), ALT and AST > 5 times upper limit of normal (n = 1), investigator decision (n = 1), and study withdrawal (n = 1).

Ex vivo analyses

Ex vivo APC activation was significantly greater at the second and third infusions compared with baseline in both the concurrent and sequential arms (P < 0.05), indicative of the previously described immunologic prime-boost effect (ref. 11; Fig. 1). No statistically significant differences between the 2 arms with respect to the median cumulative product parameters were observed. The median cumulative APC activation with sipuleucel-T across the 3 infusions was 33.65 for the concurrent arm and 38.24 for the sequential arm. Both APC numbers and TNC counts were relatively stable across infusions and comparable between arms. Median cumulative number of APCs across the 3 infusions was 1.83 × 10^7 in the concurrent arm and 1.46 × 10^7 in the sequential arm. Median TNC across the 3 infusions was 9.17 × 10^3 in the concurrent arm and 10.80 × 10^3 in the sequential arm (Supplemental Table S1).

In vivo analyses

In vivo cellular and humoral responses through week 26 were comparable between the concurrent and sequential arms (Fig. 2). T-cell proliferation and memory T-cell responses (IFN\textgamma ELISPOT)
were observed in both arms beginning at week 6 and persisting through week 26. Cellular responses to PA2024 were significantly higher than baseline at all timepoints from pre-leukapheresis 3 through week 26 ($P < 0.05$) in both arms. PA2024 and PAP antibody titers were present in both arms beginning at week 6 and were significantly higher at all post-baseline time points through week 26 ($P < 0.001$). No statistically significant differences ($P > 0.10$) between the concurrent and sequential arms in immune responder frequency were observed (Fig. 3). Thirty-two patients (94.1%) in the concurrent arm and all 34 patients (100%) in the sequential arm demonstrated a positive humoral or cellular immune response to PA2024. A response to PAP was observed in 70.6% (24/34) of patients in the concurrent arm versus 76.5% (26/34) in the sequential arm.

**Table 1. Demographics, disease characteristics, and study treatment administration**

<table>
<thead>
<tr>
<th></th>
<th>Concurrent arm ($N = 35$)</th>
<th>Sequential arm ($N = 34$)</th>
<th>Total ($N = 69$)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Median age, years (range)</strong></td>
<td>69 (55–91)</td>
<td>69 (49–90)</td>
<td>69 (49–91)</td>
</tr>
<tr>
<td><strong>Race, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Caucasian</td>
<td>33 (94.3%)</td>
<td>29 (85.3%)</td>
<td>62 (89.9%)</td>
</tr>
<tr>
<td>Asian</td>
<td>0 (0%)</td>
<td>2 (5.9%)</td>
<td>2 (2.9%)</td>
</tr>
<tr>
<td>Black or African American</td>
<td>2 (5.7%)</td>
<td>3 (8.8%)</td>
<td>5 (7.2%)</td>
</tr>
<tr>
<td><strong>ECOG performance status, n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>28 (80.0%)</td>
<td>26 (76.5%)</td>
<td>54 (78.3%)</td>
</tr>
<tr>
<td>1</td>
<td>7 (20.0%)</td>
<td>8 (23.5%)</td>
<td>15 (21.7%)</td>
</tr>
<tr>
<td><strong>Gleason score n (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;6</td>
<td>5 (14.3%)</td>
<td>5 (15.2%)</td>
<td>10 (14.7%)</td>
</tr>
<tr>
<td>7</td>
<td>12 (34.3%)</td>
<td>8 (24.2%)</td>
<td>20 (29.4%)</td>
</tr>
<tr>
<td>≥8</td>
<td>18 (51.4%)</td>
<td>20 (60.6%)</td>
<td>38 (55.9%)</td>
</tr>
<tr>
<td><strong>Median time from diagnosis to randomization, years (range)</strong></td>
<td>7.0 (1.0–22.2)</td>
<td>4.1 (0.8–19.8)</td>
<td>4.8 (0.8–22.2)</td>
</tr>
<tr>
<td><strong>Prior radical prostatectomy</strong></td>
<td>29 (82.9%)</td>
<td>27 (79.4%)</td>
<td>56 (81.2%)</td>
</tr>
<tr>
<td><strong>Antigen spread analyses</strong></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Increased IgG levels to all secondary antigens ($P < 0.01$) were observed in both arms at week 6, 10, and 14 (Fig. 4). There were no significant differences in the magnitude of IgG increase between the 2 arms ($P > 0.05$) posttreatment.

**PSA outcomes**

The percentage of patients with a ≥50% PSA decrease from the last PSA prior to AA + P administration was 23 of 35 (65.7%) for patients on the concurrent arm and 20 of 34 (58.8%) for patients on the sequential arm ($P = 0.624$). The maximal percentage decrease in PSA was calculated for each patient along with the median values for the 2 treatment groups. No statistically significant difference was observed between the concurrent and sequential arms in median maximal PSA decrease from baseline ($−78.6\%$ vs. $−63.5\%; P = 0.277$; Supplementary Table S2).

**Safety**

The most common AEs occurring in ≥15% of patients are summarized in Table 2. AEs were similar across both treatment arms. All 35 concurrent arm patients and 32 sequential arm patients (94.1%) experienced at least one AE. Muscle spasms were grade 1 to 2, and approximately 40% occurred within 1 day of a leukapheresis. AEs within 1 day after sipuleucel-T infusion in >5% of all randomized patients were similar across treatment arms and included chills, back pain, fatigue, pyrexia, influenza-like illness pain, muscle spasms, and nausea (Supplementary Table S3). AEs of grade 3 or more were reported in 10 patients (28.6%) in the concurrent arm and 13 patients (38.2%) in the sequential arm. Grade 4 to 5 AEs occurred in 3 patients in each arm and included DP ($n = 3$), asthenia ($n = 1$), cerebrovascular accident ($n = 1$), and subdural hematoma ($n = 1$). There was no difference in clinically significant (grade ≥3) laboratory toxicities between treatment arms. In general, AEs were similar to those reported previously in IMPACT (20).

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Figure 1:
Box-and-whisker plots of APC activation in patients from the concurrent (blue boxes) and sequential (orange boxes) arms at infusions 1, 2, and 3. *Ex vivo* APC activation was significantly greater at the second and third infusions compared with infusion 1 in both arms ($P < 0.05$), indicative of an immunologic prime-boost effect.
The use of intermediate biologic endpoints was an important design feature of this trial. With the large number of potential combinations of therapies that can be used in mCRPC, conducting large, costly phase III trials for every possible combination would be impractical. A successful combinatorial approach will require the development of intermediate endpoints and biomarkers. Use of such endpoints in the phase II STAMP trial allowed for reasonable powering with a relatively small number of patients and for earlier readout of results as compared with studies evaluating OS as the primary endpoint. Importantly, data interpretation should reflect the fact that at each timepoint, the full sample number for each blood draw was not obtained. The endpoints were carefully chosen biomarkers to reflect both properties of sipuleucel-T during ex vivo manufacturing (i.e., the degree of product potency as measured by APC activation) and in vivo immune responses. The degree of product potency (cumulative APC activation) achieved during the manufacture of sipuleucel-T and the generation of in vivo cellular or humoral immune responses to PA2024 and to PAP have been shown to positively correlate with OS in patients with mCRPC (11, 20).

**Figure 2.** Box-and-whisker plots showing in vivo cellular and humoral responses through 26 weeks were comparable between the concurrent (blue) and sequential (orange) arms. A, T-cell proliferation (³H-thymidine incorporation) and (B) memory T-cell counts (IFNγ ELISPOT) to PA2024 were observed in both arms beginning at week 6 and persisted through week 26. C, PA2024 and PAP titers (combined serum IgG-IgM; ELISA) were present in both arms beginning at week 6 and were significantly higher at all post-baseline time points through week 26 (P < 0.001).
Data from this trial suggest that coadministration of sipuleucel-T and AA + P does not impair the manufacture of sipuleucel-T or blunt the peripheral immune responses generated from treatment. Moreover, these data indicate that prednisone at this dose and frequency does not need to be avoided with sipuleucel-T. Neither sipuleucel-T ex vivo product parameters (APC activation, APC number, and TNC count) nor in vivo peripheral immune responses (T-cell responses, T-cell proliferation, humoral responses, and antigen spread) were affected by the concurrent use of AA + P. Importantly, in the sequential arm, AA + P was not initiated until study week 10. Therefore, product parameter values and the peripheral immune responses through week 10 in the sequential arm reflect sipuleucel-T manufacture in the absence of AA + P. The product parameter profiles and peripheral immune responses in both arms are consistent with experience in previously conducted phase III trials of sipuleucel-T (20).

Antigen spread was evaluated as a post hoc endpoint based on recent data showing that this phenomenon was induced by sipuleucel-T and was associated with improved OS in the IMPACT study (15–17). The emergence of in vivo immune responses to tumor antigens not directly targeted by an immunotherapy has been proposed as an indicator of an effective antitumor immune response that could provide pharmacodynamic biomarkers of therapeutic efficacy (12–14). Antigen spread to multiple nontargeted secondary antigens was observed in patients treated with sipuleucel-T in 2 clinical studies (IMPACT and ProACT) but did not occur in patients who received the control intervention in the...
IMPACT trial. Moreover, responses to the nontargeted antigens PSA and LGALS3 following sipuleucel-T treatment were associated with improved OS (15–17). Antigen spread was observed in both arms of the STAMP trial, and consistent with the other immunologic endpoints in the trial, no significant differences in antigen spread were observed between the sequential or concurrent arms. These results further support that coadministration of AA + P does not blunt the immunologic effects of sipuleucel-T. The similar magnitude of antigen spread between the arms prior to the initiation of AA + P in the sequential arm (i.e., on or before study week 10) indicates that AA + P alone does not induce antigen spread. Thus, this phenomenon indeed appears to be specific to sipuleucel-T.

This study was not powered to evaluate the clinical or PSA-modulating effects of sequential or concurrent sipuleucel-T and AA + P. However, the rate of ≥50% decline in PSA, a rough measure of anticanccer activity, was similar in the 2 treatment arms. The overall rate of PSA decline of ≥50% was 62.3% and was consistent with the PSA decline rate observed in the phase III trial of AA + P in the prechemotherapy setting (COU-AA-302 trial), where 62% of patients in the AA + P arm demonstrated a ≥50% decline in PSA levels (21).

Finally, sipuleucel-T appeared reasonably well tolerated in combination with AA + P, with a similar side-effect profile between concurrent and sequential administration. No new safety signals were observed with the combination of sipuleucel-T and AA + P.

In summary, this study suggests that the simultaneous administration of sipuleucel-T and AA + P does not alter immune parameters known to correlate with the clinical benefit observed with sipuleucel-T. Based on the data reported here, the combination of these agents appeared to be well tolerated with no new safety signals emerging. Although this study was not powered to evaluate the clinical utility of combining sipuleucel-T and AA + P, nonoverlapping mechanisms of action and toxicity profiles make these agents an attractive treatment combination. Unlike AA + P and other cytotoxic therapies that have acute antitumor effects on a tumor during the period of administration, the effects of sipuleucel-T appear to develop over a longer period of time and persist, leading to a slowing of the tumor growth rate over time without immediate impacts on disease burden (3). It has been suggested that a combination approach of novel, oral oncolytic therapy, such as AA + P, and immunotherapy would lead to both tumor regression and prolonged reduction in tumor growth rates, potentially resulting in synergistic improvements in OS (3). Currently, long-term follow-up for OS is ongoing in the STAMP study.

Disclosure of Potential Conflicts of Interest

R.S. Lance reports receiving speakers bureau honoraria from and is a consultant/advisory board member for Astellas Pharmaceutical. L.I. Karsh is a consultant/advisory board member for and reports receiving speakers bureau honoraria from and is a consultant/advisory board member for Astellas Pharmaceutical and Janssen. No potential conflicts of interest were disclosed by the other authors.

Authors’ Contributions

Conception and design: E.J. Small, T.A. Gardner, C. McCoy, T. DeVries, N.A. Sheikh, N.D. Shore

Development of methodology: E.J. Small, T.A. Gardner, C. McCoy, N.D. Shore

Acquisition of data (provided animals, acquired and managed patients, provided facilities, etc.): E.J. Small, R.S. Lance, T.A. Gardner, L.I. Kanth, L. Fong, T. DeVries, N.A. Sheikh, C.H. Redfern

Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): L.I. Karsh, C. McCoy, T. DeVries, N.A. Sheikh, D. GuhaThakurta, N. Chang, C.H. Redfern, N.D. Shore

Writing, review, and/or revision of the manuscript: E.J. Small, R.S. Lance, T.A. Gardner, L.I. Kanth, C. McCoy, T. DeVries, D. GuhaThakurta, N. Chang, C.H. Redfern, N.D. Shore

Study supervision: E.J. Small, R.S. Lance, C. McCoy, N. Chang

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References

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