Background

PRL-02 is a long-acting intramuscular (IM) depot injection of abiraterone decanoate, a novel lipidic prodrug of abiraterone that is delivered through the lymphatic system.

In a castrate monkey model, single-dose PRL-02 suppressed testosterone (T) through 14 weeks to levels comparable to clinical results with oral abiraterone acetate (AA) but with lower and less variable plasma exposure.

PRL-02 normally inhibits CYP17 hydroxylase and preferentially inhibits CYP17 lyase, and therefore blocks androgens without harmful increases in mineralocorticoids or depletion of steroids in the glucocorticoid pathway (Fig 1).

Phannacokinetics (PK), a known intrinsic effect of abiraterone prostate cancer through the activation of androgen receptor (AR)- and non-AR-driven pathways, is elevated in patients (pts) treated with oral AA.

Nonclinical study results show higher abiraterone exposures from PRL-02 compared to oral AA in arterial, lymph, and bone lower exposures in tissue, brain, and plasma, suggesting that PRL-02 has the potential for a superior therapeutic index and safety profile compared to oral AA.

Methods

Patients with metastatic castration-resistant or sensitive prostate cancer with prior docetaxel (T) or prior enzalutamide, apalutamide, and/or darolutamide (Cohort E) were included. Documentation of evidence of progression within 64 days (1 cycle) along with daily co.

Results

As of 18 Jan 2023, 41 pts enrolled in this trial.

Phase 1 dose escalation was a standard 3+3 design that identified a recommended Phase 2 dose. The median baseline T level in dose escalation pts was 7.45 ng/dL.

In a castrate monkey model, single-dose PRL-02 suppressed testosterone (T) through 14 weeks to levels comparable to clinical results with oral abiraterone acetate (AA) but with lower and less variable plasma exposure.

PRL-02 normally inhibits CYP17 hydroxylase and preferentially inhibits CYP17 lyase, and therefore blocks androgens without harmful increases in mineralocorticoids or depletion of steroids in the glucocorticoid pathway (Fig 1).

Phannacokinetics (PK), a known intrinsic effect of abiraterone prostate cancer through the activation of androgen receptor (AR)- and non-AR-driven pathways, is elevated in patients (pts) treated with oral AA.

Nonclinical study results show higher abiraterone exposures from PRL-02 compared to oral AA in arterial, lymph, and bone lower exposures in tissue, brain, and plasma, suggesting that PRL-02 has the potential for a superior therapeutic index and safety profile compared to oral AA.

Background

PRL-02 is a long-acting intramuscular (IM) depot injection of abiraterone decanoate, a novel lipidic prodrug of abiraterone that is delivered through the lymphatic system.

Methods

Patients with metastatic castration-resistant or sensitive prostate cancer (mCRPC) with prior docetaxel (T) or prior enzalutamide, apalutamide, and/or darolutamide (Cohort E) were included. Documentation of evidence of progression within 64 days (1 cycle) along with daily co.

Results

As of 18 Jan 2023, 41 pts enrolled in this trial.

Phase 1 dose escalation was a standard 3+3 design that identified a recommended Phase 2 dose. The median baseline T level in dose escalation pts was 7.45 ng/dL.

In a castrate monkey model, single-dose PRL-02 suppressed testosterone (T) through 14 weeks to levels comparable to clinical results with oral abiraterone acetate (AA) but with lower and less variable plasma exposure.

PRL-02 normally inhibits CYP17 hydroxylase and preferentially inhibits CYP17 lyase, and therefore blocks androgens without harmful increases in mineralocorticoids or depletion of steroids in the glucocorticoid pathway (Fig 1).

Phannacokinetics (PK), a known intrinsic effect of abiraterone prostate cancer through the activation of androgen receptor (AR)- and non-AR-driven pathways, is elevated in patients (pts) treated with oral AA.

Nonclinical study results show higher abiraterone exposures from PRL-02 compared to oral AA in arterial, lymph, and bone lower exposures in tissue, brain, and plasma, suggesting that PRL-02 has the potential for a superior therapeutic index and safety profile compared to oral AA.

Background

PRL-02 is a long-acting intramuscular (IM) depot injection of abiraterone decanoate, a novel lipidic prodrug of abiraterone that is delivered through the lymphatic system.

Methods

Patients with metastatic castration-resistant or sensitive prostate cancer (mCRPC) with prior docetaxel (T) or prior enzalutamide, apalutamide, and/or darolutamide (Cohort E) were included. Documentation of evidence of progression within 64 days (1 cycle) along with daily co.

Results

As of 18 Jan 2023, 41 pts enrolled in this trial.

Phase 1 dose escalation was a standard 3+3 design that identified a recommended Phase 2 dose. The median baseline T level in dose escalation pts was 7.45 ng/dL.

In a castrate monkey model, single-dose PRL-02 suppressed testosterone (T) through 14 weeks to levels comparable to clinical results with oral abiraterone acetate (AA) but with lower and less variable plasma exposure.

PRL-02 normally inhibits CYP17 hydroxylase and preferentially inhibits CYP17 lyase, and therefore blocks androgens without harmful increases in mineralocorticoids or depletion of steroids in the glucocorticoid pathway (Fig 1).

Phannacokinetics (PK), a known intrinsic effect of abiraterone prostate cancer through the activation of androgen receptor (AR)- and non-AR-driven pathways, is elevated in patients (pts) treated with oral AA.

Nonclinical study results show higher abiraterone exposures from PRL-02 compared to oral AA in arterial, lymph, and bone lower exposures in tissue, brain, and plasma, suggesting that PRL-02 has the potential for a superior therapeutic index and safety profile compared to oral AA.

Background

PRL-02 is a long-acting intramuscular (IM) depot injection of abiraterone decanoate, a novel lipidic prodrug of abiraterone that is delivered through the lymphatic system.

Methods

Patients with metastatic castration-resistant or sensitive prostate cancer (mCRPC) with prior docetaxel (T) or prior enzalutamide, apalutamide, and/or darolutamide (Cohort E) were included. Documentation of evidence of progression within 64 days (1 cycle) along with daily co.

Results

As of 18 Jan 2023, 41 pts enrolled in this trial.

Phase 1 dose escalation was a standard 3+3 design that identified a recommended Phase 2 dose. The median baseline T level in dose escalation pts was 7.45 ng/dL.

In a castrate monkey model, single-dose PRL-02 suppressed testosterone (T) through 14 weeks to levels comparable to clinical results with oral abiraterone acetate (AA) but with lower and less variable plasma exposure.

PRL-02 normally inhibits CYP17 hydroxylase and preferentially inhibits CYP17 lyase, and therefore blocks androgens without harmful increases in mineralocorticoids or depletion of steroids in the glucocorticoid pathway (Fig 1).

Phannacokinetics (PK), a known intrinsic effect of abiraterone prostate cancer through the activation of androgen receptor (AR)- and non-AR-driven pathways, is elevated in patients (pts) treated with oral AA.

Nonclinical study results show higher abiraterone exposures from PRL-02 compared to oral AA in arterial, lymph, and bone lower exposures in tissue, brain, and plasma, suggesting that PRL-02 has the potential for a superior therapeutic index and safety profile compared to oral AA.

Background

PRL-02 is a long-acting intramuscular (IM) depot injection of abiraterone decanoate, a novel lipidic prodrug of abiraterone that is delivered through the lymphatic system.

Methods

Patients with metastatic castration-resistant or sensitive prostate cancer (mCRPC) with prior docetaxel (T) or prior enzalutamide, apalutamide, and/or darolutamide (Cohort E) were included. Documentation of evidence of progression within 64 days (1 cycle) along with daily co.

Results

As of 18 Jan 2023, 41 pts enrolled in this trial.

Phase 1 dose escalation was a standard 3+3 design that identified a recommended Phase 2 dose. The median baseline T level in dose escalation pts was 7.45 ng/dL.

In a castrate monkey model, single-dose PRL-02 suppressed testosterone (T) through 14 weeks to levels comparable to clinical results with oral abiraterone acetate (AA) but with lower and less variable plasma exposure.

PRL-02 normally inhibits CYP17 hydroxylase and preferentially inhibits CYP17 lyase, and therefore blocks androgens without harmful increases in mineralocorticoids or depletion of steroids in the glucocorticoid pathway (Fig 1).

Phannacokinetics (PK), a known intrinsic effect of abiraterone prostate cancer through the activation of androgen receptor (AR)- and non-AR-driven pathways, is elevated in patients (pts) treated with oral AA.

Nonclinical study results show higher abiraterone exposures from PRL-02 compared to oral AA in arterial, lymph, and bone lower exposures in tissue, brain, and plasma, suggesting that PRL-02 has the potential for a superior therapeutic index and safety profile compared to oral AA.