**Background**

- CDK7 is a key regulator of two biological processes that drive tumor development: transcription and cell cycle control.

**Methods**

- **SY-5609** is an oral, noncovalent, highly selective and potent inhibitor of CDK7.
- **Syros Pharmaceuticals** has developed SY-5609, an oral, noncovalent, highly selective and potent inhibitor of CDK7.
- SY-5609 demonstrates deep and sustained tumor regressions, including activity in models known to be refractory to standard of care therapies.

**SY-5609 Profile**

- **Key Inclusion**
  - Any metastatic or unresectable solid tumor histology
  - Patients are required to have measurable disease.
  - received previous treatment(s) and have disease progression
  - Karnofsky performance status ≥ 70
  - Life expectancy ≥ 3 months

**Key Exclusion**

- Prior treatment with an FDA approved kinase inhibitor, except for inhibitors such as inhibitors of CDK4 and CDK6

**Eligibility Criteria**

- **Dose Level 1**
  - **Starting Dose** 1 mg QD
  - **Dose Level 2**
  - **Starting Dose** 3 mg QD
  - **Dose Level 3**
  - **Starting Dose** 6 mg QD
  - **Dose Level 4**
  - **Starting Dose** 9 mg QD

**Study Design**

- The study is a multi-center, open label Phase 1 study expected to enroll approximately 60 adult patients with ovarian, breast, colorectal, or lung cancer, and those with any solid tumor histology with molecular evidence of deregulated RB cell cycle control.

**Stability of SY-5609**

- SY-5609 is an oral, noncovalent, highly selective and potent inhibitor of CDK7.

**Study Summary**

- This is a multi-center, open-label Phase I study expected to enroll approximately 60 adult patients with ovarian, breast, colorectal, or lung cancer, and those with any solid tumor histology with molecular evidence of deregulated RB cell cycle control, for which standard curative or palliative measures do not exist or are no longer effective, including:
  - Histologically confirmed metastatic or unresectable solid tumor histology
  - Any metastatic or unresectable solid tumor histology with evidence of deregulated RB cell cycle control
  - RB1, CDKN1A, CDKN2A, TP53, RB1, CDKN1A, CCNE1, CCND1, CCND2, CDK4, CDK6, and/or alterations in CCNE1, CCND1, CCND2, CDK4, CDK6.

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