**BIOLOGICAL ACTIVITY:**
Erdafitinib (JNJ-42756493) is a potent and orally available FGFR family inhibitor; inhibits FGFR1-4 with IC$_{50}$ values of 1.2, 2.5, 3.0 and 5.7 nM, respectively. IC50 & Target: IC50: 1.2 nM (FGFR1), 2.5 nM (FGFR2), 3.0 nM (FGFR3) and 5.7 nM (FGFR4)\[1\]

In Vitro: Erdafitinib inhibits the tyrosine kinase activities of FGFR1-4 in time-resolved fluorescence assays with IC$_{50}$ values of 1.2, 2.5, 3.0 and 5.7 nM, respectively. The closely related VEGFR2 kinase is less potently inhibited (30-fold less potent compared to FGFR1) by erdafitinib, with an IC$_{50}$ value of 36.8 nM. JNJ-42756493 binds FGFR1, 3, 4, and 2 with K$_d$ values of 0.24, 1.1, 1.4 and 2.2 nM, respectively. The K$_d$ value for VEGFR2 is higher at 6.6 nM. JNJ-42756493 inhibits proliferation of FGFR1, 3, and 4 expressing cells with IC$_{50}$ values of 22.1, 13.2, and 25 nM, respectively\[1\].

In Vivo: In xenografts from human tumor cell lines or patient-derived tumor tissue with activating FGFR alterations, Erdafitinib administration results in potent and dose-dependent antitumor activity accompanied by pharmacodynamic modulation of phospho-FGFR and phospho-ERK in tumors\[1\].

**PROTOCOL (Extracted from published papers and Only for reference)**

Cell Assay: Erdafitinib is dissolved in DMSO. KATO III, RT-112, A-204, RT-4, DMS-114, A-427 and MDA-MB-453 cells are treated with erdafitinib (from 10 μM to 0.01 nM in 2% DMSO, final concentration). Following 4-day incubation, cell viability is determined using MTT reagent. The optical density is determined at 540 nm\[1\].

Animal Administration: Mouse: Mice bearing SNU-16 human gastric carcinoma (FGFR2 amplified) xenograft tumors are dosed orally with 0, 3, 10 or 30 mg/kg JNJ-42756493. Tumor tissue and mouse plasma (3 mice per time point) are harvested at 0.5, 1, 3, 7, 16 and 24 h post-dosing\[1\].

**References:**