

KRICT | IRAK4 inhibitor (KIC0090s)

Target	
Mechanism of Action	<ul style="list-style-type: none"> NF-κB and IRF signaling suppression through IRAK4 inhibition
Indication - Primary	<ul style="list-style-type: none"> Autoimmune diseases (Systemic Lupus Erythematosus, Rheumatoid Arthritis) Hematological cancers (ABC-DLBCL, MyD88-pathway activated Lymphoma)
Indication - Expansion	<ul style="list-style-type: none"> Autoimmune disease, Malignant tumor
Route of Administration	<ul style="list-style-type: none"> PO
Competitive Advantage	<ul style="list-style-type: none"> Competitive in efficacy & PK profiles compared with PF06650833 (Pfizer, Phase I) IRF signaling suppression : >60% inhibition at 1 μM, THP-1 Kinase selectivity : 2/56 profiling (>90% inhibition at 1 μM, IRAK4, PIM1) Novel chemical structure
Data Files	<ul style="list-style-type: none"> In vitro IRAK4 enzyme inhibition : IC₅₀ 1~10 nM In vitro cell-based inhibition : <ul style="list-style-type: none"> Inflammatory signaling , NF-κB (>30% at 0.1 μM), IRF (>20% at 0.1 μM) Cytokine secretion, TNF-α, IFN-α (IC₅₀ 0.1 ~ 1 μM, PBMC) In vivo cytokine reduction : TNF-α 35% (25 mpk, po, q-d, rodent model) DMPK (mouse) : AUC 0.239 μM·h ; T_{1/2} 8.91 h; F = 11% (10 mpk, po) Tox : CYP450s (IC₅₀ > 10 μM) hERG binding (IC₅₀ > 10 μM), Cytotoxicity (GI₅₀ >10 μM)
IP Status	<ul style="list-style-type: none"> Patent filing underway
Collaboration Model	<ul style="list-style-type: none"> Licensing out, Collaborative research
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