Modeling Molecular Pathogenesis of Idiopathic Pulmonary Fibrosis-Associated Lung Cancer in Mice

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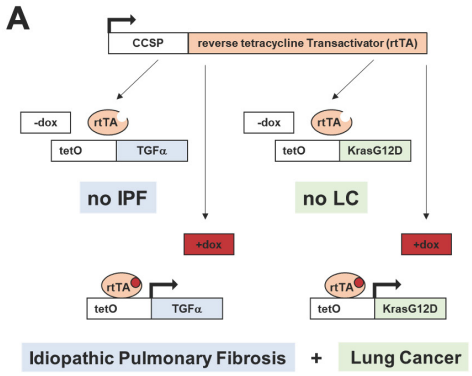
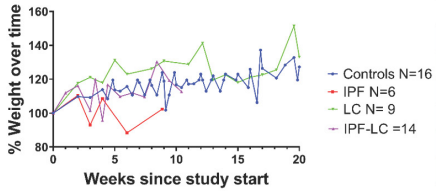
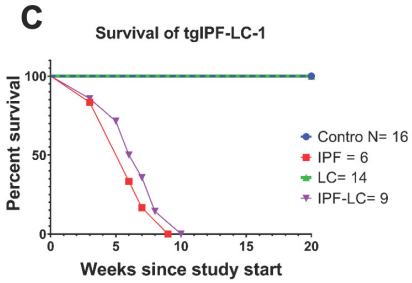
Supplemental Figure S1





Group	P-value	P-Value summary
Control vs. IPF	<0.0001	****
Control vs. LC	0.1532	ns
Control vs. IPF-LC	0.0111	*
LC vs. IPF	0.0372	*
LC vs. IPF-LC	<0.0001	****
IPF vs. IPF-LC	0.0003	***



Group	P-value	P-Value summary
Control vs. IPF	< 0.0001	****
Control vs. LC	> 0.9999	ns
Control vs. IPF-LC	< 0.0001	****
LC vs. IPF	< 0.0001	****
LC vs. IPF-LC	< 0.0001	****
IPF vs. IPF-LC	0.646	ns

Supplemental Figure S1: Transgenic mouse IPF-LC model development. (**A**) Schematic description of the transgenic mouse model used in the study. Expression of reverse tetracycline transactivator (rtTA) is controlled by the Clara cell secretory protein (CCSP) promotor in Club cells (formerly Clara cells). Addition of doxycycline to the drinking water of mice results in activation of TGF alpha and oncogenic Kras (KrasG12D), which are under the control of Tetracycline Operon (TetO). (**B**) Weight change and (**C**) Kaplan-Meier Survival analysis of mice in control, IPF, LC and IPF-LC groups. Weights were analyzed with ordinary one-way ANOVA with Tukey's multiple comparison test and survival curves were analyzed using the Log Rank (Mantel-Cox) test. No statistical difference was determined between IPF and IPF-LC groups.