Koch's postulates	Postulates fulfilled by oleic acid vs. IL-1 β & KC/GRO in SAP
The microorganism must be found in abundance in all organisms suffering from the disease, but should not be found in healthy organisms.	Oleate concentrations (1235±412 μM) in 14 patients with necrotic collections (NC) are >100 fold of those in pancreatic cystic neoplasms (11.7 ± 3.9 μM) from 10 patients. Previous studies have found higher serum oleate in AP patients with complications (Ref 117) than in mild AP. IL-1 β and KC/GRO were similarly increased in NCs.
The microorganism must be isolated from a diseased organism and grown in pure culture	Oleate and IL-1 β/KC-GRO were found using very specific methods in NCs and also used in pure form *in vitro* or *in vivo.*
The cultured microorganism should cause disease when introduced into a healthy organism.	1. *In vivo* GTO hydrolysis to oleate <u>but not IL-1 β and KC/GRO</u> converted caerulein pancreatitis into a lethal disease with MSOF, with necro-apoptotic PBMCs, necrotic cells in the BAL and TUNEL positive cells in the lungs and kidneys and kidney injury molecule-1 (KIM-1) positive tubules. Caerulein pancreatitis itself caused none of these. 2. *In vitro* Glyceryl trilinoleate (GTO) hydrolysis resulted in glycerol formation, thus oleate generation (Figure 3) and caused acinar injury. Oleate at < 50% on NC concentrations <u>but not IL-1 β and KC/GRO</u> at >10x these concentrations caused acinar injury. 3. Others have shown oleic acid to cause lung injury (ref 109-112) and renal failure (Ref 110, 121) in healthy rodents.
The microorganism must be reisolated from the inoculated, diseased experimental host and identified as being identical to the original specific causative agent.	Serum Oleate was significantly higher in the CER+GTO group which developed MSOF, <u>but not the CER+IL-1 β and KC/GRO group which neither had an increase in serum oleate</u> nor MSOF.

Supplementary Figure 6