

## A Simple, Inexpensive and Easily Reproducible Experimental Model of Acute and Chronic Pancreatitis: Biliopancreatic Injection of Ethanol in Rats

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Pancreatitis is a severe disease with high mortality. In recent years, great advances have been achieved in our understanding of the inflammatory diseases of the pancreas and in their clinical management. Clinical studies can bring some data about etiology, pathogenesis and the course of the disease. However, studies concerning early and late events of this disease seem to be insufficient in explaining the disease process, and the new concepts of treatment cannot be performed on humans due to ethical reasons. Therefore, animal models of acute pancreatitis (AP) have been developed to solve these problems. Several different experimental pancreatitis models have been described in the literature with varying success rates. The ideal experimental AP model should be technically simple to create, minimally invasive, reproducible, inexpensive and should resemble the human disease with respect to its triggering event, pathophysiology and disease course. However, none of the existing AP models fulfill all of these criteria. Furthermore, chronic pancreatitis (CP) models are very scant. Therefore, it is not surprising that none of the pancreatitis models is universally used and that the pathophysiology of disease still remains poorly understood.

In the present Editorial Letter, we want to mention about a published article mentioned a new experimental model producing both AP and CP in rats [1,2]. The result for this model in previous experimental pancreatitis models in rats were well [3-8]. In this simple and easily reproducible model, AP is induced by biliopancreatic ductal injection of ethyl alcohol. 70 Wistar albino rats were divided equally into seven groups randomly: the control group (group 1), AP expected groups induced by 20% ethanol (group 2), 48% ethanol (group 3), 80% ethanol (group 4) and CP expected groups induced by 20% ethanol (group 5), 48% ethanol (group 6) and by 80% ethanol (group 7). AP groups were sacrificed on postoperative day 3, while the control group and CP groups were killed on postoperative day 7. Histopathologic evaluation revealed that all rats in group 3 developed AP (100%). Inflammatory infiltration of neutrophils and mononuclear cells, interstitial edema and focal necrotic areas were conclusive with AP. Similarly, all rats in group 6 developed CP (100%). Interstitial fibrosis, lymphocytic infiltration, ductal dilatation, acinar cell atrophy and periductal hyperplasia were seen in the pancreatic tissues. Mortality was seen only in group 7. The biliopancreatic ductal injection of 48% ethanol induced AP and CP had 100% success rate. The most important advantage of this model is its effects on pathophysiology like the one in human. Its direct cytotoxic and oxidative stress producing effects are well-known, similar to those shown in human studies. The reduction of pancreatic blood flow and microcirculation and damaging effects of ethanol metabolites and free oxygen radicals are known as well.

In conclusion, we highly recommend this successful and proven experimental model to all researchers interested in AP and CP disease pathophysiology.

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