INTRODUCTION

Chronic pancreatitis (CP) has been defined as a continuing inflammatory disease of the pancreas characterized by irreversible morphologic changes that typically cause pain and/or permanent loss of function (1–3). Rather than a single disease, CP is a syndrome of destructive inflammatory conditions that encompasses the many sequelae of long-standing pancreatic injury (4). Until recently, most research and treatment energy focused on managing end-stage CP and its complications after most of the functional pancreas was destroyed, and the patient was left with endocrine and exocrine insufficiency and chronic pain. New research is focusing on identifying environmental, genetic, and other risk factors, as well as on the inflammatory and cellular mechanisms driving the process. Furthermore, efforts are underway to improve the
diagnosis of CP at the earliest stages when the organ might be saved and returned toward normal.

In developed countries, CP is usually attributed to excess alcohol consumption (4). However, a single factor seldom causes pancreatitis, and even prolonged excessive alcohol consumption in animal models and in most humans does not cause CP (4). Instead, alcohol appears to be one of several specific environmental, metabolic, and genetic factors that play synergistic roles in increasing susceptibility to acute pancreatitis (AP) and in driving the pancreatic inflammation, fibrosis, and parenchymal destruction that eventually results in clinically recognized CP. In addition, CP may require a triggering event to initiate the inflammatory process that is a key factor in this disease (5). Organizing, classifying, and understanding the major issues involved in the etiology and progression of CP will form the basis for updated methods of early detection, diagnosis, treatment, and prevention.

**DIAGNOSIS**

The gold standard for diagnosing CP is histology (4) with representative specimens demonstrating the presence of chronic inflammatory cells, acinar cell drop-out, and fibrosis (Fig. 1). Other pathological features, including duct proliferation, nerve trunk inflammation and enlargement, and distortion or loss of islets are later findings that confirm

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**Fig. 1.** Histology of chronic pancreatitis (CP). During the progression of CP the normal acini (A) are lost and replaced by progressive fibrosis (F). Note the lymphocytic infiltration associated with fibrosis. The islets (i) are relatively spared until late in the disease process.