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***Correlation Between Gastric Epithelial Cells
Apoptosis and Helicobacter
Pylori Infection***

YV110P

A THESIS

*Submitted for partial fulfillment of the MASTER DEGREE
in TROPICAL MEDICINE*

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TO MY FAMILY
THE EVERLASTING
SOURCE OF LOVE
AND SACRIFICE

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INTRODUCTION

INTRODUCTION

Since its discovery 15 years ago, *Helicobacter pylori* (*H. pylori*) infection has generated a vast amount of research activity. *H. pylori* was considered as the principal cause of type B gastritis and peptic ulcer disease and has been classified as type I carcinogen for gastric ulcer on the basis of seroepidemiological evidence; in addition to its association with low grade gastric mucosa associated lymphoid tissue lymphoma (*Blaser et al., 1994 and Cover & Blaser, 1996*). The mechanism by which this chronic infection of the gastric antrum predisposes to ulceration of the duodenum is unclear (*Buck, 1990*). In recent years, interest in apoptosis has been intensified. Apoptosis is a highly regulated form of programmed cell death defined by distinct morphological and biochemical features. It is a type of cell death in which the cell actively uses a genetically controlled program to cause its own demise such as what occurs during tissue remodeling of embryogenesis (*Schulte et al., 1995*), maintain tissue homeostasis; remove senescent cells and delete cells with genetic damage beyond repair (*Que & Gores, 1996*).

Dysregulation of apoptosis appears to play a key role in the pathogenesis of many diseases including the gastrointestinal diseases (*Patel et al., 1998*).

Evidence for the induction of apoptosis by *H. pylori* and its possible implication in the pathogenesis of ulceration and gastric malignancy has been recently obtained from many studies (*Clarke & Clarke, 1996; Potten, 1996 & Jones et al., 1997*).

One of the most exciting discoveries regarding apoptosis is that it can be triggered by specific cell surface receptors e.g. Fas/APO-1/CD 95 engaging specific ligands leading to cell death. This concept is clearly important because for the first time in human disease, it becomes possible to identify target molecules for pharmacological modulation of the disease process (*Patel et al., 1998*).