INTRODUCTION

A variety of chemicals have been reported to induce lesions in the olfactory epithelium (Appleman et. al., 1982)

Sulfur Dioxide, one of the most common air pollutants may increase the risk of various respiratory diseases in exposed individuals. Sulfur Dioxide has been known to cause chronic obstructive lung disease in animals. (Watson, 1980)

The mammalian nasal epithelium is capable of removing inspired sulfur dioxide with remarkable effeciency. Physiologic evidence in humans and rabbits indicates that almost all inhaled sulfur dioxide is absorbed by the nasal mucosa. However, it is not certain wheather nasal absorption of sulfur dioxide causes actual cell injury. Some investigators have reported that sulfur dioxide may destroy the respiratory epithelium. (Margenroth, 1980).

Changes in the olfactory epithelium can be devided into stages of inflammation, degeneration and proliferation. Inflammation may occur as aresult of exposure to infectious organism or as a response to toxic substances. Inflammatory cells are more commmonly seen in the lamina propria than in epithelium itself, and inflammatory changes are non specific and dependent on the dose and concenteration of the chemical. (Woutersen et al., 1981)

However, only few data on changes on the olfactory epithelium following exposure to sulfur dioxide have been reported. Previous histopathologic studies in animals exposed to sulfur dioxide have been limited to the respiratory epithelium such as trachea or lung. (Clark et al., 1981)