INTRODUCTION AND AIM OF WORK

Asthma is a leading cause of chronic illness in childhood. It is responsible for a significant proportion of school days lost because of chronic illness *(Sly 2000)*. Asthma is the most chronic lung disorder in children; it affects approximately 2 to 5 million children in the United State *(Chidekel 2000)*.

Asthma is a complex disorder involving autonomic, immunologic, infectious, endocrine and psychologic factors in varying degrees in different individuals *(Sly 2000)*.

The effect of passive smoke exposure on lung function and its possible association with subsequent development of lung disease have become major health issues *(Ashcroft 1998)*.

Continine one of the major metabolites of nicotine, provides more advantages, because it is specific for exposure to nicotine from tobacco smoke, it is chemically stable and urinary pH influences the excretion of cotinine less than it influences nicotin excretion, in addition, its longer half life (19 - 40 h compared with 2 h for nicotine), it means that it reflects long term exposure, whereas nicotine reflects recent exposure *(Oddoze et al., 1999)*.

Variable airflow obstruction is the primary finding in asthma. Objective assessment of degree of obstruction, reversibility, and hyperresponsiveness is essential in the dignosis of asthma. The measurment of changes in airway caliber evaluates the effectivenes of the theraputic intervention. A primary goal of asthma control is normal lung function *(Susan et al., 2000)*.

Aim of work

The aim of this study is to evaluate the effect of passive smoking on pulmonary functions in asthmatic and healthy children.