# Bronchial Asthma Of Childhood In Tripoli Medical Center, Libya.

Fathia M Alfassal

Department of Pediatrics, Faculty of Medicine, Al-mergib University, Al-khoms, Libya

# ABSTRACT:

Asthma varies considerably across the life course. Childhood asthma severity is associated with duration of asthma symptoms, medication use, lung function, low socioeconomic status, racial/ethnic minorities, and a neutrophilic phenotype. Adult onset disease is associated with more respiratory symptoms and asthma medication use despite higher prebronchodilator FEV1/FVC. There is less quiescent disease in adult onset asthma and it appears to be less stable than childhood-onset disease with more relapses and less remissions. Asthma in older children is characterised by a histopathology of a chronic inflammatory process in the conducting airways. Genetic predisposition, in combination with environmental factors, such as allergens and viral infections, may contribute to the development of asthma. In research which including 190 children from age 1 year to 15 years old who are asthmatic at our out patients department showed, there is 69% of asthmatic children with consanguinity compared with 31% with no consanguinity, while allergic rhinitis with 51% in consanguinity and 49% in No consanguinity is 85% and No consanguinity is 15%. Finally pollen consanguinity is 83% and No consanguinity is 13%.

Keywords: Bronchial asthma, children, Tripoli Medical Center, Libya.

Date of Submission: 29-07-2023

Date of Acceptance: 09-08-2023

# I. INTRODUCTION:

Asthma is a chronic disorder of the bronchial tree, characterized by completely or partially reversible airway obstruction, which may improve spontaneously or may subside only after specific therapy <sup>(1)</sup>. Airway hyperresponsiveness is defined as the narrowing of the airways as response to a variety of stimuli, such as allergens and nonspecific triggers and infections. Asthma is a chronic disorder of both children and adults <sup>(2)</sup>, with 300 million individuals afflicted worldwide (Global Initiative for Asthma (GINA) guidelines <sup>(3)</sup>. Asthma is characterized by inflammation leading to bronchoconstriction, edema, and increased mucous production in the airways <sup>(4)</sup>. Interestingly, the disorder is more prevalent in boys in the first decade of life. However <sup>(5)</sup>, after puberty and in the second decade of life, it appears that asthma is more prevalent in young women <sup>(6)</sup>. Asthma disproportionately affects minority and low-income children with African American and Hispanic children having the highest prevalence rates, morbidity and mortality due to asthma <sup>(7)</sup>. Asthma is considered a chronic disease of childhood however there are periods of time during which disease can go into remission or resolve altogether. Important risk factors for the development of childhood asthma have been identified <sup>(8)</sup>. The phenotypes of childhood asthma and varied presentations are best defined through the periods of the pediatric life course and are described herein <sup>(9)</sup>.

Maternal tobacco smoking during pregnancy has been shown to increase the risk of childhood asthma <sup>(10)</sup>. Maternal diet in pregnancy has also been implicated as an asthma risk factor with reports of maternal diets higher in vitamin E, zinc, and polyunsaturated fatty acids as protective against the development of childhood asthma <sup>(11)</sup>. In contrast, high sugar intake in the maternal diet during pregnancy has been associated with increased risk of asthma in offspring <sup>(12)</sup>. Other maternal dietary factors have been studied but with less conclusive results including the intake of vitamin D, vitamin C, and a Mediterranean diet. Other perinatal risk factors for childhood asthma that have been reported are neonatal jaundice, maternal preeclampsia, and cesarean section delivery <sup>(13)</sup>, all which have been associated with higher risk of childhood asthma development. Ultimately gene-environment interactions (the genetic-environmental axis) are critical for the development of asthma in a child <sup>(14)</sup>.

# Study Place:

# II. METHODOLOGY:

This prospective study has done from the enrolled 190 children from age 1 year to 15 year old who diagnosed as bronchial asthma in the respiratory out-patients department at Tripoli Medical Center, Tripoli, Libya.

# Study Period:

The study period was determined from January 2020 – October 2020, for 10 months preceded the data collection.

#### Sampling Procedure:

We attempted an extensive collection of survey results by different sources in which bronchial asthma among Indian children was reported, including meeting presentations and personal communications. Through an extensive website-scanned search in indexed literatures and study reports, we identified 15 epidemiological studies of the development of asthma in Libyan children from 300 potentially relevant articles. Reviews of citations and reference lists were performed to identify additional eligible studies. The search terms included bronchial asthma, Indian children, prevalence, asthmatic bronchitis, wheeze, wheezy bronchitis, and reactive airway disease. Where possible, sources were contacted for further information on survey data not readily available in the public domain. Manual searches were conducted from review articles and previous meta-analyses.

#### Statistical analysis:

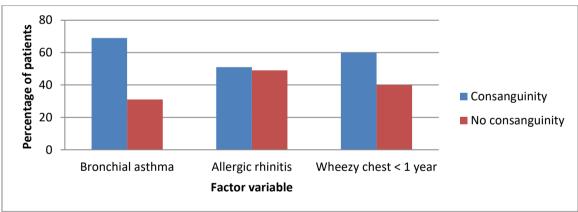
The data were analyzed and processed using the SPSS 22.0 statistical package (IBM Corp.USA). The Independent Samples t-test was used for comparison of means. P<0.05 was considered to indicate a statistically significant difference.

# III. RESULTS AND DISCUSSION:

From the research which including 190 children from age 1 year to 15 years old who are asthmatic at our out patients department showed, there is 69% of asthmatic children with consanguinity compared with 31% with no consanguinity. While allergic rhinitis with 51% in consanguinity and 49% in No consanguinity.

Table A1. Comparison of factor variable between the two groups		
Factor Variable	Consanguinity	No Consanguinity
Bronchial asthma	69%	31%
Allergic rhinitis	51%	49%
Wheezy chest < 1year	60%	40%

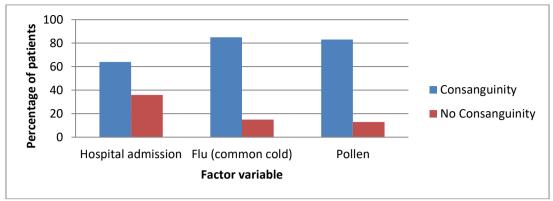
Table A1: Comparison of factor variable between the two groups



Graph A1: Comparison of factor variable between the two groups.

Table 2 is tabulated with factor variable like hospital admission, Flu (common cold) and Pollen. In consanguinity hospital admission include 64% and No consanguinity is 36%. Meanwhile in flu consanguinity is 85% and No consanguinity is 15%. Finally pollen consanguinity is 83% and No consanguinity is 13%.

Factor Variable	Consanguinity	No Consanguinity
Hospital admission	64%	36%
Flu (common cold)	85%	15%
Pollen	83%	13%



Graph A2: Comparison of factor responsible for Bronchial asthma between the two groups

#### IV. CONCLUSION:

Asthma is one of the most chronic disorders in children <sup>(15)</sup>. The prevalence of asthma has increased during the last decades but seems to have reached a plateau. The burden of asthma is considerable <sup>(16)</sup>. It influences quality of life, may prevent children from participating in sports and play, may hamper social contacts, and may cause school absence and hamper career development <sup>(17, 18)</sup>. Asthma begins in early life. Before the age of 6 many children wheeze, but only 40% of these early wheezers develop asthma. Due to lack of national representative data on the prevalence, risk factors, and prognosis of the disease, there is an urgent need for more public health research in this field of priority attention and direction.

#### **REFERENCES:**

- [1]. Soriano JB, Abajobir AA, Abate KH, Abera SF, Agrawal A And Ahmed MB (2017) Global, Regional, And National Deaths, Prevalence, Disability-Adjusted Life Years, And Years Lived With Disability For Chronic Obstructive Pulmonary Disease And Asthma, 1990–2015: A Systematic Analysis For The Global Burden Of Disease Study 2015. Lancet Respiratory Medicine, 5(9):691– 706.
- [2]. Chung KF And Adcock IM (2019) Precision Medicine For The Discovery Of Treatable Mechanisms In Severe Asthma. Allergy, 74(9): 1649–1659.
- [3]. Agache I And Akdis CA (2019) Precision Medicine And Phenotypes, Endotypes, Genotypes, Regiotypes, And Theratypes Of Allergic Diseases. The Journal Of Clinical Investigation, 129(4): 1493–1503.
- [4]. Guo Y, Moon J-Y, Laurie CC And North KE (2018) Genetic Predisposition To Obesity Is Associated With Asthma In US Hispanics/Latinos: Results From The Hispanic Community Health Study/Study Of Latinos. Allergy, 73(7): 1547–1550.
- [5]. Celebi Sozener Z, Cevhertas L, Nadeau K, Akdis M And Akdis CA (2020) Environmental Factors In Epithelial Barrier Dysfunction. Journal Of Allergy And Clinical Immunology, 145(6): 1517–1528.
- [6]. Papi A, Brightling C, Pedersen SE And Reddel HK (2018) Asthma. The Lancet, 391(10122): 783-800.
- [7]. Sveiven SN And Nordgren TM (2020) Lung-Resident Mesenchymal Stromal Cells Are Tissue-Specific Regulators Of Lung Homeostasis. American Journal Of Physiology. Lung Cellular And Molecular Physiology, 319(2): L197–L402.
- [8]. Kruk DMLW, Heijink IH, Slebos DJ, Timens W And Ten Hacken NH (2018) Mesenchymal Stromal Cells To Regenerate Emphysema: On The Horizon? Respiration, 96(2):148-158.
- [9]. Eddy RL, Serajeddini H And Knipping D (2020) Pulmonary Functional MRI And CT In A Survivor Of Bronchiolitis And Respiratory Failure Caused By E-Cigarette Use. Chest, 158(4): E147–E151.
- [10]. Adkins SH, Anderson KN And Goodman AB (2020) Demographics, Substance Use Behaviors, And Clinical Characteristics Of Adolescents With E-Cigarette, Or Vaping, Product Use-Associated Lung Injury (EVALI) In The United States In 2019. JAMA Pediatrics, 174(7):E200756.
- [11]. Litonjua AA, Carey VJ And Laranjo N (2020) Six-Year Follow-Up Of A Trial Of Antenatal Vitamin D For Asthma Reduction. New England Journal Of Medicine, 382(6): 525–533.
- [12]. Barcik W, Boutin RCT, Sokolowska M And Finlay BB (2020) The Role Of Lung And Gut Microbiota In The Pathology Of Asthma. Immunity, 52(2): 241–255.
- [13]. Sbihi H, Boutin RC, Cutler C, Suen M, Finlay BB And Turvey SE (2019) Thinking Bigger: How Early-Life Environmental Exposures Shape The Gut Microbiome And Influence The Development Of Asthma And Allergic Disease. Allergy, 74(11): 2103–2115.
- [14]. Stokholm J, Blaser MJ And Thorsen J (2018) Maturation Of The Gut Microbiome And Risk Of Asthma In Childhood. Nature Communications, 9(1): 141.
- [15]. Michalovich D, Rodriguez-Perez N And Smolinska S (2019) Obesity And Disease Severity Magnify Disturbed Microbiome-Immune Interactions In Asthma Patients. Nature Communications, 10(1): 5711.
- [16]. Vieira Braga FA, Kar G And Berg M (2019) A Cellular Census Of Human Lungs Identifies Novel Cell States In Health And In Asthma. Nature Medicine, 25(7): 1153–1163.
- [17]. Lu Y, Kared H And Tan SW (2018) Dynamics Of Helper CD4 T Cells During Acute And Stable Allergic Asthma. Mucosal Immunology, 11(6): 1640–1652.
- [18]. Asayama K, Kobayashi T And D'Alessandro-Gabazza CN (2020) Protein S Protects Against Allergic Bronchial Asthma By Modulating Th1/Th2 Balance. Allergy, 75(9): 2267–2278.