

# MANAGEMENT AND TREATMENT OF ASTHMA

Manisha Vikarm Dhage, Pallavi Tukaram Jadhav

Student, Teacher  
Pratibhatai Pawar College Of Pharmacy

## Abstract:

A nationwide survey was conducted in Finnish community pharmacies (n = 785) in September 2016 targeting patients buying prescription medicines for asthma or allergies. Results: Responses were received from 46% of targeted pharmacies. Around 73% of the asthma patients, 61% of patients at risk of anaphylaxis, and less than 50% of the other allergy patients had received a written action plan. The most common source of treatment information for both patient groups was pharmacists. Allergy patients sought information more from written sources than asthma patients. Older males and patients with lower education received less treatment information. About 10% of both asthma and allergy patients did not report receiving any treatment information. Deterioration usually progresses over hours, days, or weeks; however, a few patients have sudden (over minutes) and unexpected in – Creases in airway obstruction. Epidemic asthma, the simultaneous occurrence in place and time of an unusually high number of asthma attacks, has been reported in at least 12 Different locations around the world. A number of circumstances may mimic the diagnosis of AA (COPD, congestive Heart failure, upper airway obstruction, hyperventilation – Taxation syndrome, or vocal cord dysfunction) shortly after the Introduction of the New Zealand and Canadian guidelines, the National Heart, Lung, and Blood Institute of the United States National Institutes of Health participated in additional asthma Guideline endeavors, which were published in the early 1990s.

**Keywords:** Anaphylaxis, Epidemic asthma, congestive Heart failure,

**Introduction:** The Japanese Pediatric Guideline for the Treatment and Management of Asthma was revised in November of 2002 by the 2002 Asthma Guideline Committee of the Japanese Society of Pediatric Allergy and Clinical Immunology. (1) Asthma is a substantial fitness trouble that contributes to the global burden of sickness. (2) However, deterioration or failure to Respond to these measures sometimes leads to severe respiratory failure. (3) the most common causes of these exacerbations are exposure to external agents, such as indoor and outdoor allergens air pollutants, and respiratory tract infections. (4) .Deterioration typically progresses Over hours, days, or weeks; however, a few patients Have sudden (over minutes) and unexpected in – Creases in airway obstruction. (5) Management Other Materials in the Pharmacology in the Pharmacology Sections Patient Education Special Circumstances Target Audience Provision for Frequent Updating Summary. (6) The allergen mission fashions in human beings are of questionable fee in knowledge the pathogenesis of continual allergic disorder and those could be noted simplest in passing. (7) Asthmatic patients were in fact regarded as chronically infirm individuals who needed to be sheltered and avoid physical exertion in order to prevent severe asthma attack. (8) Accordingly, the Global Initiative for Asthma (GINA) recommends people with asthma to engage in regular physical activity in order to improve their general health. (9) (LTC) that is Characterized with the aid of breathlessness, tightness in the chest, coughing and wheezing, alongside with episodes Of surprising worsening in signs and symptoms (asthma assaults or exacerbations) that can show frailty. (10) There are many pulmonary And systemic co morbidities in COPD patients, such as bronchiectasis, asthma, heart failu Cardiovascular diseases, sleep apnea, malnutrition, and frailty .(11)Although it is nicely acknowledged that positive topic status, publicity and sensitization to environmental allergens, and/or familial allergic ailments are large chance elements associated with the improvement of asthma, latest proof propose that nutrition deficiency can also predispose to allergic phenotype .(12)Vitamin through its motion on the immune gadget law appears to play a pivotal function in allergan illnesses.(13) . In medical era In the Middle Ages, the information of allergies and its redress began to cross ahead bit with the aid of bit. Europeans started out the usage of tobacco as an expectorant to resource the elimination of mucus round 1500 AD. Bernardino Ramazani was once the first to find out a relationship between allergies and dust, and recognized “exercise induced asthma”. In South America, the Incas (14)

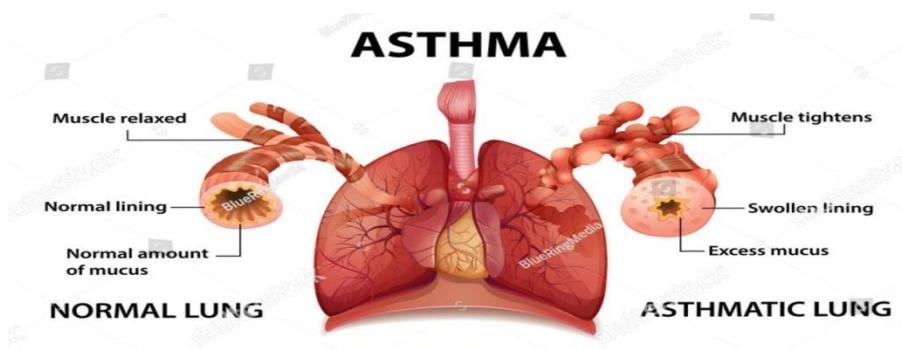


Fig (1) Asthma

## Protective Effects in Asthma:

### 1. Improving Mucociliary Clearance:

**-Mucociliary clearance (MCC)** is a vigorous innate defense to remove mucus and related Toxins, germs, viruses, and inflammatory cells from the respiratory airways. As airway Epithelial cultures become increasingly dehydrated, they lose their ability to transport Mucus, establishing a critical link between mucus dehydration and deficient MCC. (15) There is evidence that the mucus obtained from asthmatic patients is dehydrated more than expected(16) dissociating deoxyribonucleic acid from the mucoprotein (which results in the mucoprotein being digested by the natural proteolytic enzymes) (17) A small group of well-controlled, moderate-severe female asthmatic patients was Treated with a single test dose of albuterol (four puffs by metered-dose inhaler) followed By HS (7% sodium chloride, 4 mL) (18) A small group of well-controlled, moderate-severe female asthmatic patients was Treated with a single test dose of albuterol (four puffs by metered-dose inhaler) followed By HS (7% sodium chloride, 4 mL) no significant lung function decline before or after inhaled challenges with endotoxin or dust mite allergen (19).

**Therapeutic Effect of Halotherapy on Acute and Chronic Asthma**In addition to mucus rehydration and clearance, additional postulated mechanisms May contribute to the therapeutic effect in asthmatic patients. (20) Halotherapy—An Ancient Natural Ally in the Management of Asthma: A Comprehensive Review. (21) Two studies assessed the veracity of the therapeutic benefits of HT in acute asthma. In a double-blind, randomized clinical trial of 340 adult patients with acute asthma attacks, (22) significantly more powerful antimicrobial agent than hydrogen peroxide, especially Against Escherichia coli (23) Chlamydia pneumonia suggest a link to infection and the onset of asthma. (24) Or the extracellular space, acting as an electron transferees and thus generating a superoxide (25) (MPO), is then converted to a very potent antimicrobial agent, the hypochlorous acid. (26)

### Obesity on the Respiratory Apparatus:

**1.Inflammation :** Obesity associates a non-TH2 subclinical inflammatory state induced by the infiltration of M1-type macrophages capable of liberating proinflammatory cytokines such as IFN- $\gamma$ , IL-6, TNF- $\alpha$ , IL-1 $\beta$  and monocyte chemo tactic peptide (MCP)-1 [23]. The number of M1 macrophages in visceral fat is correlated with the body mass index (BM). (27) This difference has also been shown when asthmatic obese people are compared to non-obese asthmatic (28) Furthermore, a strong negative correlation between plasma IL-6concentrations and expression of the CD8+ T-cell genes network. (29)

**2. Changes in Lung Function:**One study of almost 400 adults described a significant drop, in relation with the BMI, Of the total lung capacity (TLC), expiratory reserve volume (ERV), functional residual Capacity (FRC), vital capacity (VC) and residual volume (RV). (30) Childhood obesity being associated with lower FEV1/FVC because of a disproportional increase of FVC with respect to FEV1 with increasing BM (31).

### Pathophysiology:

**Bronchoconstriction** In asthma, the dominant physiological tournament main to scientific airflow. In acute exacerbations of asthma, bronchial easy muscle contraction (bronchoconstriction) takes place quickly to slender the airways in response to publicity to a range of stimuli along with allergens or irritants. Allergen-induced acute Bronchoconstriction outcomes from an Ige dependent launch of mediators from mast cells that consists of histamine, striptase, leukotrienes, and prostaglandins that at once contract airway clean muscle. (32) Aspirin and different no steroidal anti-inflammatory tablets can additionally motive acute airflow obstruction in some patients, and proof cells. (33)

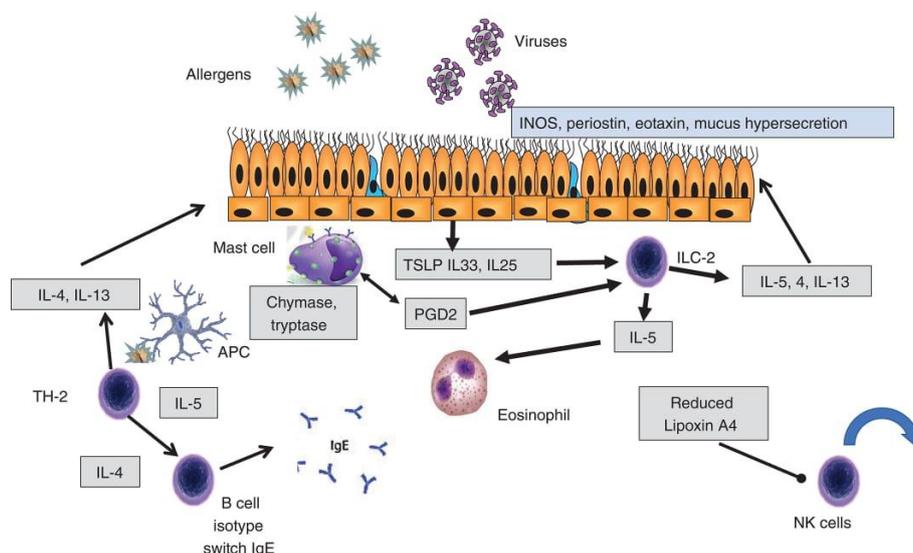


Fig2.Pathophysiology of severe asthma

In addition, different stimuli (including exercise, bloodless air, and irritants) can motive acute airflow obstruction. The mechanisms regulating their way response to these elements are much less properly defined; however the depth of the response seems related to underlying airway inflammation. Stress may additionally play a position in precipitating asthma exacerbations. The mechanisms concerned have but to be mounted and May also include superior technology of pro-inflammatory cytokines. Airway Edam As the sickness turns into greater chronic and irritation greater progressive, different factors in addition restrict airflow (figure 2–2). These consist of edam, as structural adjustments which include hypertrophy and hyperplasia of the airway easy muscle. no longer always unique, characteristic of asthma. (34)

#### Risk factor:

**Outdoor allergens:** Exposure to ambient grass pollen is an important trigger for childhood asthma exacerbations requiring emergency department attendance and this have been recently confirmed by a systemic review. (35) Ig Sensitization to fungal species is associated with increased asthma severity, neutrophilic inflammation, and reduced lung function consistent with ACOS. (36) There is also scant evidence on the role of early life exposure to pollen in the development of childhood asthma. (37) Those asthmatic children are susceptible to exacerbations that lead to hospitalization when exposed to outdoor fungal spores. (38)

**Thunderstorm Asthma:** Thunderstorm asthma is defined as epidemics that occur during or shortly after a thunderstorm, where individuals affected would experience asthma-related symptoms such as breathlessness, wheezing and coughing. “Thunderstorm asthma” (39, 40) this can occur in sensitized individuals who may or may not have a prior history of asthma or asthma symptoms, but who often have a history of allergic rhinitis. Fungal spore allergens may also be involved (41, 42)

**Personal Smoking:** In utero maternal smoking and parental smoking in early life has been shown to be temporally associated with increased asthma in young children (43). Tobacco smoking may cause heritable modifications of the epigenome, which increase the risk of asthma in future generations (44) many studies have found that personal smoking predisposes an individual to increased risk of incident or new-onset asthma, although smoking-onset in adolescence or adulthood typically occurs after early-onset asthma (45).

**Lifestyle Factors:** These dietary patterns feature a high calorie intake which is high in saturated fat and refined sugars and associated with a high glycemic index, as well as low nutritional value in terms of dietary fiber and vitamins. While this “obesogenic diet” may lack antioxidant and anti-inflammatory properties (46), a meta-analysis has found being overweight and obese to be associated with a dose-response increase in incident asthma in adults (47). Non-atopic asthma end type that is symptom-predominant and less steroid-responsive in previous cluster and LCA (48, 49). And this in turn can lead to poorer fitness levels and a propensity to weight gain (50)

**Air Pollutants:** Outdoor air pollution almost certainly has a major global impact on asthma for children and adults, especially in China and India. (51) Exposure to PM<sub>1</sub> has been found to increase the risk of asthma and asthma-related symptoms, especially among boys, and those with allergic predisposition. (52) Oxford Street was associated with reductions in lung function, neutrophilic inflammation and airway acidification (53)

**Epidemiology:** The most recent Centers for Disease Control (CDC) Asthma epidemiology statistics were published in 2012 for the 2001–2010 decade. A major increase of 2.9% each year, with 20.3 million persons carrying a diagnosis of asthma in 2001 and 25.7 million persons having this diagnosis in 2010. This national surveillance report estimated (54) prevalence in past years, with prior CDC reports documenting the prevalence of asthma in the United States as 3.0% in 1970 and 5.5% in 1996. (55) many asthma patients may be undiagnosed, as a recent study from Russia demonstrated that 6.9% of adults answering a questionnaire reported an asthma diagnosis, but 25.7% of the same population reported asthma symptoms (56)

#### Global impact:

**Age:** In terms of age, 18.7 million of the 25.7 million Persons with asthma in access United States in 2010 Were adults (with 3.1 million being age 65 or older), And 7.0 million were children. (57) United States had asthma in 2007, with a life-time prevalence as high as 13% (56). The global prevalence of pediatric asthma is reported to be approximately 14%, and similarly to the adult group, is rising (58) In fact, from 2008–2010, the average annual asthma prevalence was higher in children aged 0–17 years. (59) A recent study of schoolchildren in Poland (ages 7–10 years) showed an increase in asthma prevalence from 3.4% in 1993 to 12.6% in 2014. (60) In fact, from 2008–2010, the average annual asthma prevalence was higher in children aged 0–17 years (9.5%) than in adults aged 18 and over (7.7%) [1].

**Impact on gender:** In terms of gender, studies have shown a male predominance in the diagnosis of asthma prior to puberty, but a higher prevalence in females in adulthood, as well as more severe cases of asthma in women than in men. (61)

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**Asthma trends:** The epidemiological trends of asthma are as follows: asthma prevalence has increased at a rate of 1.4% per year in the pediatric population (0–17 years) and at a rate of 2.1% per year in the adult population (18 years and older). Current asthma prevalence has increased at a yearly rate of 1.8% for both males and females. Trends in race include a yearly rate increase of 1.4% among the white population and yearly rate increase of 3.2% among the black population; whereas trends in ethnicity include a

yearly rate increase of 3.2% among Hispanic persons (7.2% of the population in 2010) and a yearly rate increase of 1.9% among non-Hispanic persons (8.7% of the population in 2010), (62)

**B2-Adrenergic Agonists:** Inhaled short-acting  $\beta$ 2-adrenergic agonists should be administered immediately on presentation, and administration can be repeated up to three times within the first hour after presentation. The use of a metered-dose inhaler with a valve holding chamber is as effective as the use of a pressurized nebulizer in randomized trials, 4,5but proper technique is often difficult to ensure in ill patients. Most guidelines recommend the use of nebulizers for patients with severe exacerbations; metered-dose inhalers

**Anticholinergic Agents:** Because of its relatively slow onset of action, inhaled ipratropium is not recommended as mono-therapy in the emergency department but can be added to a short-acting  $\beta$ 2-adrenergic agonist for a greater and longer-lasting bronchodilator effect.<sup>10,11</sup> In patients with severe airflow obstruction, the use of ipratropium together with a  $\beta$ 2-adrenergic agonist in the emergency department, as compared with a  $\beta$ 2-adrenergic agonist alone, has been shown to reduce rates of hospitalization by approximately 25%,

**Systemic Corticosteroids:** In most patients with exacerbations that necessitate treatment in the emergency department, systemic corticosteroids are warranted. The exception is the patient who has a rapid response to initial therapy with an inhaled  $\beta$ 2-adrenergic agonist.

**Inhaled Corticosteroids:** Although high-dose inhaled corticosteroids are often used to treat worsening of asthma control and to try to prevent exacerbations, the evidence does not support the use of inhaled corticosteroids as a substitute for systemic corticosteroids in the emergency department. In a randomized, controlled trial of 1006 consecutively enrolled patients with acute asthma treated in a Canadian emergency department, the addition at discharge of inhaled budesonide (for 21 days) to treatment with oral corticosteroids (for 5 to 10 days) was associated with a 48% reduction in the rate of relapse at 21 days and with improvement in the quality of life with respect to asthma (as measured by the Asthma Quality of Life Questionnaire) and symptoms, as compared with treatment with oral corticosteroids alone.<sup>(63)</sup>

**Medication:** When prescribed and used appropriately, the medications presently accessible to deal with allergies are very effective in controlling signs and symptoms in most patients. Asthma medications are divided into two categories: reliever medicines for the speedy comfort of signs and controller medicines for day by day manipulate of bronchial asthma and prevention of symptoms.

**Reliever medications:** Fast-acting bronchodilators, which relax the muscle groups surrounding the airways, are used to relieve allergies attacks. They are normally inhaled and take effect within minutes. The results generally remaining for 4 to six hours, so if the person is no longer uncovered to the attack's set off after that time, a single dose of treatment may be enough. If allergies signs and symptoms are introduced on by continuous publicity to such triggers as viral infections.

**Alternative medicine:** Approximately 1/2 of asthma patients use some structure of unconventional therapy—such as acupuncture, air ionizers, and chiropractic treatments—to manipulate their illness. There is too little proof on the subject matter to both assist or refute choice medicine treatments of asthma, even though sufferers who use herbal treatments have to be counseled about the achievable for allergic reactions and interactions with traditional asthma medications, and have to be warned no longer to pass their prescribed medications (64)

**Prevention:** Lung function abnormalities and airway remodeling can be seen in early life in those who develop persistent asthma. The degree of FEV1/FVC ratio impairment in childhood was found to be the largest predictor of asthma remission by adulthood. Decreased airway responsiveness in childhood was also a predictor of remission of asthma.<sup>30</sup> Interventions to improve the lung function of young children at high risk of developing asthma have been evaluated in asthma prevention. In an extension of the Prevention of Early Asthma in Kids study, high-risk 2- to 3-year-olds were treated with 2 years of inhaled fluticasone and observed for 1 year after.<sup>43</sup> During the treatment period, the intervention increased episode-free days and reduced asthma exacerbations. However, after cessation of therapy, no evidence was found that indicated that treatment improved lung function or modified asthma development.<sup>31</sup> This study, and others evaluating the use of inhaled corticosteroids in at-risk children, did not support the use of inhaled corticosteroids in modifying the course of disease. (65)

**Conclusion:** Asthma is characterized by variable airway obstruction, airway hyper responsiveness, and airway inflammation. Management of persistent asthma requires avoidance of aggravating environmental factors, use of short-acting  $\beta$ 2-agonists for rapid relief of symptoms, and daily use of inhaled corticosteroids. Other controller medications, such as long-acting bronchodilators and biologics, may be required in moderate and severe asthma. Patients with severe asthma generally benefit from consultation with an asthma specialist for consideration of additional treatment, including inject able biologic agents. Despite the availability of approved asthma treatments, this literature analysis confirms that SUA poses a substantial epidemiologic, clinical, humanistic, and economic burden. Published data are limited for certain aspects of SUA, highlighting a need for further research.

**Reference:**

- (1) Mitsufumi Mayumi The 2002 Japanese Pediatric Guideline For the Treatment and Management of Asthma Allergology International. 2005;54:217-221
- (2) Masahiko Shigemura, Tetsuya Homma and Jacob I Sznajder 1, Review Hypercapnia: An Aggravating Factor in Asthma. J Clin. Med. 2020, 9, 3207;
- (3) Eirini Kostakou, Evangelos Kaniaris, Effrosyni Filiou et al, Acute Severe Asthma in Adolescent and Adult Patients: Current Perspectives on Assessment and Management. Clin. Med. 2019, 8, 1283;
- (4) Gustavo J Rodrigo, Carlos Rodrigo, Jesse B Hall Acute asthma in adults: a review Chest 125 (3), 1081-1102, 2004
- (5) Timothy R Myers RRT-NPS. Guidelines for Asthma Management: A Review and Comparison of 5 Current Guidelines Respiratory Care June 2008 Vol 53 No 6.
- (6) A J Wardlaw, C Brightling, R Green et al, Eosinophils in asthma and other allergic diseases. British Medical Bulletin 2000; 56 (No. 4): 985-1003.
- (7) Worsnop, C.J. Asthma and physical activity. Chest 2003, 124, 421–422.
- (8) Marios Panagiotou, Nikolaos G. Koulouris and Nikoletta Rovina. Physical Activity: A Missing Link in Asthma Care. J. Clin. Med. 2020, 9, 706
- (9) Sarah de Oliveira Rodrigues, Carolina Medina Coeli da Cunha, Giovanna Martins Valladão Soares et al, Mechanisms, Pathophysiology and Currently Proposed Treatments of Chronic Obstructive Pulmonary Disease. Pharmaceuticals 2021, 14, 979.
- Decisionmaking cognition in mania and depression. Psychol Med 2001; 31:679–92001;31:679–93.
- (10) Royal College of Physicians. Why asthma still kills: the national review of asthma deaths (NRAD) confidential Enquiry report RCP(RevCubaPediater);2015. <https://www.rcplondon.ac.uk/projects/outputs/whyasthma-still-kills>. Accessed October 2, 2018
- (11) Katayoun Bahadori, Mary M Doyle-Waters, Carlo Marra et al, Economic burden of asthma: a systematic review. BMC Plum Med. 2009; 9:24
- (12) Nunes C, Pereira AM, Morais-Almeida M. Asthma costs and social impact. Asthma Res Pract. 2017; 3(1):1.
- (13) Thomas M. Why aren't we doing better in asthma: time for personalised medicine? NPJ Primary Care Respirat Med. 2015;25:15004.
- (14) H. Huang, K. Porpodis, P. Zarogoulidis, K. Domvri, P. Giouleka, A. Papaiwannou, et al., Vitamin D in asthma and future perspective Drug Design, Development and Therapy 2013;7 1003–1013
- (15) N. Eid, R. Morton, B. Olds, P. Clark, S. Sheikh, S. Looney, Decreased morning serum cortisol levels in children asthma treated with inhaled fluticasone propionate, Pediatrics 109 (2) (2002) 217–221.
- (16) M.J. Visser, E. van der Veer, D.S. Postma et al, Side-effects of fluticasone in asthmatic children: no effects after dose reduction Eur Respir J 2004; 24: 420–425
- (17) Wen, C.-H. Wei, Z.-Q. Hu, K. Srivastava, J. Ko, S.-T. Xi, D.-Z. Mu, J.B. Du, G.- H. Li, S. Wallenstein, H. Sampson, M. Kattan, X.-M. Li, Efficacy and tolerability of antiasthma herbal medicine intervention in adult patients with moderate-severe allergic asthma, J. Allergy Clin. Immunol. 116 (3) (2005) 517.
- (18) Anderson, W.H. Coakley, R.D.; Button, B.; Henderson, A.G.; Zeman, K.L.; Alexis, N.E.; Peden, D.B.; Lazarowski, E.R.; Davis, C.W.; Bailey, S.; et al. The Relationship of Mucus Concentration (Hydration) to Mucus Osmotic Pressure and Transport in Chronic Bronchitis. Am. J. Respir. Crit. Care Med. 2015, 192, 182–190.
- (19) Loughlin, C.E.; Esther, C.R., Jr.; Lazarowski, E.R.; Alexis, N.E.; Peden, D.B. Neutrophilic inflammation is associated with altered airway hydration in stable asthmatics. Respir. Med. 2010, 104, 29–33.
- (20) Lieberman, J.; Kurnick, N.B. Influence of deoxyribonucleic acid content on the proteolysis of sputum and pus. Nature 1962, 196, 988–990.
- (21) Bennett, W.D.; Burbank, A.; Almond, M.; Wu, J.; Ceppe, A.; Hernandez, M.; Boucher, R.C.; Peden, D.B. Acute and durable effect of inhaled hypertonic saline on mucociliary clearance in adult asthma. ERJ Open Res. 2021, 7, 12. Anderson, S.D. Exercise-induced asthma and the use of hypertonic saline aerosol as a bronchial challenge. Respirology 1996, 1, 175–181.
- (22) Alexis, N.E.; Bennett, W.; Peden, D.B. Safety and benefits of inhaled hypertonic saline following airway challenges with endotoxin and allergen in asthmatics. J. Asthma 2017, 54, 957–960.
- (23) Radu Crisan-Dabija 1,2, Ioan Gabriel Sandu 3, Iolanda Valentina Popa 1,\* , Dragos-Viorel Scripcariu 1,4, Adrian Covic 1,5 and Alexandru Burlacu 1,6,7 Healthcare 2021, 9, 1604.
- (24) Forouzan, A.; Masoumi, K.; Delirrooyfard, A et al. Effect of Nebulized 3% Hypertonic Saline with Salbutamol on Management of Acute Asthma in Outpatient Adults: A Double-blind, Randomized Clinical Trial in Emergency Department. Iran J. Allergy Asthma Immunol. 2017, 16, 370–377.
- (25) Lyman, S.V.; Hurst, J.K. Role of compartmentation in promoting toxicity of leukocyte-generated strong oxidants. Chem. Res. Toxicol. 1995, 8, 833–840.)
- (26) Hahn, D.L.; Dodge, R.W.; Golubjatnikov, R. Association of Chlamydia pneumoniae (strain TWAR) infection with wheezing, asthmatic bronchitis, and adult-onset asthma. JAMA 1991, 266, 225–230
- (27) Babior, B.M. NADPH oxidase. Curr. Opin. Immunol. 2004, 16, 42–47.
- (28) Klebanoff, S.J.; Kettle, A.J.; Rosen, H.; Winterbourn, C.C.; Nauseef, W.M. Myeloperoxidase: A front-line defender against phagocytosed microorganisms. J. Leukoc. Biol. 2013, 93, 185–198
- (29) Periyalil, H.A.; Wood, L.G.; Wright, T.A.; Karihaloo, C.; Starkey, M.R.; Miu, A.S. Obese asthmatics are characterized by altered Adipose tissue macrophage activation. Clin. Exp. Allergy 2018, 48, 641–649.
- (30) Hay, C.; Henrickson, S.E. The impact of obesity on immune function in pediatric asthma. Curr. Opin. Allergy Clin. Immunol. 2021 21, 202–215.

- (31) Peters, M.C.; Ringel, L.; Dyjack, N.; Herrin, R.; Woodruff, P.G.; Rios, C.; O'Connor, B.; Fahy, J.V.; Seibold, M.A. A Transcriptional Method to Determine Airway Immune Dysfunction in T2-High and T2-Low Asthma. *Am. J. Respir. Crit. Care Med.* 2019, 199,465–477.
- (32) Jones, R.L.; Nzekwu, M.-M.U. The Effects of Body Mass Index on Lung Volumes. *Chest* 2006, 130, 827–833
- (33) Köchli, S.; Endes, K.; Bartenstein, T.; Usemann, J.; Schmidt-Trucksäss, A.; Frey, U.; Zahner, L.; Hanssen, H. Lung function, obesity and physical fitness in young children: The EXAMIN YOUTH study. *Respir. Med.* 2019, 159, 105813
- (34) Busse W, Corren J, Lanier BQ, McAlary M, Fowler-Taylor A, Cioppa GD, van As A, Gupta N. Omalizumab, IgE recombinant humanized monoclonal antibody, for the treatment of severe allergic asthma. *J Allergy Clin Immunol* 2001;108(2):184–90
- (35) Stevenson DD, Szczecin A. Clinical and pathologic perspectives on aspirin sensitivity and asthma. *J Allergy Clin Immunol* 2006;118(4):773–86; quiz 787–8.
- (36) Miss Lipte Gayatri Namdeo, Ms. Pallavi Jadhav. Asthma-A review. *International Journal of Research Publication and Reviews*, Vol 3, no 6, pp 2609-2615, June 2022.
- (37) Erbas B, Jazayeri M, Lambert KA, Katelaris CH, Prendergast LA, Tham R, et al. Outdoor pollen is a trigger of child and adolescent asthma emergency department presentations: a systematic review and meta-analysis. *Allergy*(2018) 73:1632–41.
- (38) Fairs A, Agbetile J, Hargadon B, Bourne M, Monteiro WR, Brightling CE, et al. IgE sensitization to *Aspergillus fumigatus* is associated with reduced lung function in asthma. *Am J Respir Crit Care Med.* (2010) 182:1362–8.
- (39) Erbas B, Lowe AJ, Lodge CJ, Matheson MC, Hosking CS, Hill DJ, et al. Persistent pollen exposure during infancy is associated with increased risk of subsequent childhood asthma and hayfever. *Clin Exp Allergy.* (2013)43:337–43.
- (40) Tham R, Vicendese D, Dharmage SC, Hyndman RJ, Newbiggin E, Lewis E, et al. Associations between outdoor fungal spores and childhood and adolescent asthma hospitalizations. *J Allergy Clin Immunol.* (2017)139:1140–7.
- (41) Thien F, Beggs PJ, Csutoros D, Darvall J, Hew M, Davies JM, et al. The Melbourne epidemic thunderstorm asthma event 2016: an investigation of environmental triggers, effect on health services, and patient risk factors. *Lancet Planet Health.* 2018 Jun;2(6):e255-e263
- (42) Packe GE, Ayres JG. Asthma outbreak during a thunderstorm. *Lancet.* (1985)2:199–204
- (43) Alderman PM, Sloan JP, Basran GS. Asthma and thunderstorms. *Arch Emerg Med.* (1986) 3:260–2
- (44) Burke H, Leonardi-Bee J, Hashim A, Pine-Abata H, Chen Y, Cook DG, et al. Prenatal and passive smoke exposure and incidence of asthma and wheeze: systematic review and meta-analysis. *Pediatrics.* (2012) 129:735–44.
- (45) Accordini S, Calciano L, Johannessen A, Portas L, Benediktsdottir B, Bertelsen RJ, et al. A three-generation study on the association of tobacco smoking with asthma. *Int J Epidemiol.* (2018) 47:1106–17
- (46) McLeish AC, Zvolensky MJ. Asthma and cigarette smoking: a review of the empirical literature. *J Asthma* (2010) 47:16.
- (47) Wood LG. Diet, Obesity, and Asthma. *Anna Am Thor Soc.* (2017)14(Supplement\_5):S332–S8
- (48) Beuther DA, Sutherland ER. Overweight, obesity, and incident asthma: a meta-analysis of prospective epidemiologic studies. *Am J Respir Crit Care Med.* (2007) 175:661–6.
- (49) Haldar P, Pavord ID, Shaw DE, Berry MA, Thomas M, Brightling CE, et al. Cluster analysis and clinical asthma phenotypes. *Am J Respir Crit Care Med.*(2008) 178:218–24
- (50) Jeong A, Imboden M, Hansen S, Zemp E, Bridevaux PO, Lovison G, et al. Heterogeneity of obesity-asthma association disentangled by latent class analysis, the SAPALDIA cohort. *Respir Med.* (2017) 125:25–32.
- (51) Global Initiative for Asthma. Global Strategy for Asthma Management and Prevention 2018. Available online at: [www.ginasthma.org](http://www.ginasthma.org) AAnenberg SC, Henze DK, Tinney V, Kinney PL, Raich W, Fann N, et al.
- (52) Yang M, Chu C, Bloom MS, Li S, Chen G, Heinrich J, et al. Is smaller worse? New insights about associations of PM1 and respiratory health in children and adolescents. *Environ Int.* (2018) 120:516–24.
- (53) McCreanor J, Cullinan P, Nieuwenhuijsen MJ, Stewart-Evans J, Malliarou E, Jarup L, et al. Respiratory effects of exposure to diesel traffic in persons with asthma. *N Engl J Med.* (2007) 357:2348–58.
- (54) Moorman JE, Akinbami LJ, Bailey CM, et al. National surveillance of asthma: United States, 2001–2010. *Vital Health Stat* 2012; 3:1–58.
- (55) Yang M, Chu C, Bloom MS, Li S, Chen G, Heinrich J, et al. Is smaller worse? New insights about associations of PM1 and respiratory health in children and adolescents. *Environ Int.* (2018) 120:516–24.
- (56) Chuchalin AG, Khaltaev N, Antonov NS, et al. Chronic respiratory diseases and risk factors in 12 regions of the Russian Federation. *Int J Chron Obstruct Pulmon Dis* 2014; 9:963–974.
- (57) Chuchalin AG, Khaltaev N, Antonov NS, et al. Chronic respiratory diseases and risk factors in 12 regions of the Russian Federation. *Int J Chron Obstruct Pulmon Dis* 2014; 9:963–974.
- (58) Maio S, Baldacci S, Carrozzi L, et al. Respiratory symptoms/diseases prevalence is still increasing: a Moorman JE, Akinbami LJ, Bailey CM, et al. National surveillance of asthma: States, 2001–2010. *Vital Health Stat* 2012; 3:1–United States, 2001–2010. *Vital Health Stat* 2012; 3:1–58
- (59) Brozek G, Lawson J, Szumilas D, Zejda J. Increasing prevalence of asthma, respiratory symptoms, and allergic diseases: four repeated surveys from 1993–2014. *Respir Med* 2015; 109:982–990.
- (60) Hill VL, Wood PR. Asthma epidemiology, pathophysiology, and initial evaluation. *Pediatr Rev* 2009; 30:331–335.
- (61) Asher MI, Montefort S, Björkstén B, et al. Worldwide time trends in the prevalence of symptoms of asthma, allergic rhinoconjunctivitis, and eczema in childhood: ISAAC Phases One and Three repeat multicountry cross-sectional surveys. *Lancet* 2006; 368:733–743.
- (62) Brozek G, Lawson J, Szumilas D, Zejda J. Increasing prevalence of asthma, respiratory symptoms, and allergic diseases: four repeated surveys from 1993–2014. *Respir Med* 2015; 109:982–990.

- (63) Postma DS. Gender differences in asthma development and progression. *Gend Med* 2007; 4 (Suppl B):S133–S146
- (64) Stephen C. Lazarus, M.D. Emergency Treatment of Asthma *N Engl J Med* 2010;363:755-64.
- (65) Michelle C. Maciag, MD; and Wanda Phipatanakul, MD. Prevention of Asthma. *CHEST* 2020; 158(3):913-922.