The rising prevalence of asthma in the developed world has focused the attention of researchers on changes in the host and the host's immediate environment [1]. In the later part of the twentieth century, the emphasis was on the role of infections, and the ‘hygiene hypothesis’ [2] was proffered as an explanation for the underlying immune dysfunction associated with asthma and allergic disorders. This hypothesis, however, cannot explain the disproportionate prevalence of asthma in certain populations or ethnic groups [3], leading to the exploration of genetic factors that could modulate the risk for the development of asthma and allergic disorders [4]. The first decade of 21st century saw a great deal of work on establishing the genetic basis using genome-wide association studies [5] and whole genome sequencing [6]. Genetic studies also helped to identify susceptibility genes as well as the mediators (such as IL-33 and IL-25) that were involved in the pathogenesis of allergic inflammation [7]. The recognition of some of these mediators that influence gut immune responses led to the exploration of the role of various food items and how they change the gut’s immune profile [8]. Several studies have looked at specific nutrients (such as Vitamin D) [9] as well as diet patterns [10] on the risk and severity of asthma, and the results have been mixed. Another offshoot of this emphasis on the impact of certain foods or diets is the study of the changes in the gut's microbial profile (or 'microbiome') [11]. This microbiome varies between individuals and can be influenced by a combination of early life exposures such as infections or infestations, genetic factors and exposure to various macro- and micronutrients in the developing gut (either in utero or in early infancy). Alterations in the gut microbial profile influence the immune response that modulates the inflammatory mediators and skews the pathways of inflammation towards an allergic phenotype [12].

Modulation of diet has been shown to affect a number of chronic illnesses in adults such as diabetes [16], heart disease [17] and cancer [18], but the evidence for asthma is lacking. A recent meta-analysis of all the published studies related to diet patterns from various countries failed to show a relationship between diet patterns and asthma outcomes [19]. However, the authors did note that for studies involving children, the Mediterranean diet did have a protective effect on current wheezing and lifetime diagnosis of asthma. The authors included 31 studies from their systematic review of the literature, but included only adult studies for the meta-analysis. There were significant differences among the included studies regarding the definition of Mediterranean diet and what food groups were included during data collection. In addition, the food intake patterns are also influenced significantly by local food availability and cultural practices. Therefore, a prospective (preferably randomized controlled) study design with larger sample size and an in-depth collection of food consumption data stratified by different categories of asthma severity

**Asthma and Diet: Could Food be thy Medicine?**

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should be done to further explore the impact of various foods on severity, clinical course and outcomes of chronic diseases such as asthma. Only then will there be some definitive evidence to support this controversial [20] quote from Hippocrates: “Let food be thy medicine, and medicine be thy food.”

REFERENCES

Risk Factors Associated With MRSA Infection in Children

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The prevalence rates of methicillin-resistant Staphylococcus aureus (MRSA) infections has been linked to the quality of care, and are considered the benchmark for hospital infection control practices. As the therapeutic management of MRSA infections is very different from those due to methicillin-sensitive Staphylococcus aureus (MSSA), there is a need to study risk factors associated with acquisition of MRSA which will guide the empirical antibiotic choices. A delay in appropriate initial antibiotic choice can increase mortality and morbidity associated with these infections, especially in intensive care settings. An understanding of these factors would help to generate evidence not only to decide the choice of empirical