Asthma is a chronic inflammatory disease and needs to be examined in detail considering its multiple facets and aspects. Since it presents a wide range of nuances and shades in the clinical presentation of its phenotypes, asthma can be seen as a paradigm to studies on genetics, especially on questioning the genetic origin of such plurality of phenotypes. Its origin is genetic and begins immediately at fertilization and subsequent fetal development with environmental pressure that modulates the disease expression after birth and during the individual growth [1].

There is no asthma gene, but, when one analyzes thoroughly, one can find in asthma a picture portraying a genetic landscape with multiple loci acting in its occurrence. Therefore, it is the interaction between them in the forming of a complex network that can - and should - be studied [2].

Asthma genetics studies have advanced in recent years, mainly due to new methods available for screening for genetic variants in a large scale to multiple polymorphisms. Those screening methods help reducing the cost of large population analyzing and stimulate the cooperation among various reference centers, which, for its turn, includes the creation of databases with greater sample sizes, thus allowing both a more accurate analysis of the results and a better patient follow up.

There has been several contributions showing the importance of environmental aggressions related to asthma origin, development and severity, but they are not part of this editorial. Even though it is both an environmental and genetic disease, the genetic features of asthma should not be underestimated, especially if we consider that its genetics involves multiple loci [2].

Linkage studies in asthmatic families show that a specific locus is associated with the disease. However, such studies are not yet completed. Since it is present only in a minor portion of the studied population, the gene locus cannot be specified as the causing gene for asthma. Therefore, animal models have been extensively used to understand the occurrence of asthma. Initially, specific genes and their variants were analyzed on restrict populations of patients with asthma, giving evidence of genes that might be associated with the disease or not, i.e., with asthma prevalence and severity [3]. In doing so, one can only see isolated and fragmented parts of the same figure, but one find it impossible to form a single and comprehensive image.

On the one hand, with the improvement of molecular technology and with better disease understanding (multiples genotypes and phenotypes), new tools are being used to study the whole genome, that is, to understand the multiple loci of hundreds of genes all at the same time forming a complex picture [4,5]. On the other hand, the asthma genetic studies rise facing questions still unanswered. Therefore, a comprehensive understanding of the when’s, how’s, and why’s of the plurality of clinical phenotypes presentations is yet to be revealed. (Figure 1).
The question of how to perform the genetic studies on asthma has been characterized as a boundary to be overcome by researchers in the whole world. Each and every study should consider: (i) the sample size; (ii) the ethnicity of the population studied; (iii) the environment related with the population enrolled in the study; (iv) the treatment used to control the disease; (v) the clinical and laboratory characteristics of patients enrolled in the study; (vi) the type of asthma considered in the study; (vii) the genes that will be screened; (viii) the technique that will be used for the screening of genetic variants. The asthma genetics studies tackle many important issues such as: What is the best way to show the data? What is valid and what really counts for asthma knowledge and management?

Nowadays we have plenty of genetic information from entire populations, but we still have problems with the validity of such information. Unknown variants have been identified as indicators of susceptibility to asthma in restricted populations, but are the results applicable to other populations?

In asthma genetic studies, there is still some more questions about what is individual and what is asthma risk for populations? Which is unique and exclusive for asthma? Which genetic trait is specific or not in all individuals with asthma?

Researchers have tried to understand the genetic variants that are associated with asthma. However, the link between genotypes knowledge and clinical practice (asthma phenotypes) remains distant. The physician has a fundamental role on diagnosis, monitoring and asthma management: So, what would change in knowing which genetic factors are associated with asthma occurrence and severity? What should be considered from the standpoint of genetics before the structuring of a study? Multiple elements can be designed to elucidate the problem: (i) determining genetic risk factors associated with the population - and in some cases, specific family - enables the reduction in the prevalence of asthma; (ii) better environmental control; (iii) better management of the disease; (iv) in particular cases, we can dream about the opportunity of personalized medicine, especially when we studied the patient drug response to control the disease having greater knowledge of asthma pathogenesis; (v) to refine the classification of asthma; (vi) enable new tools for asthma diagnosis [6-8].

In sum, we have had many progresses in this field of asthma genetics, but they still need to be understood by the physician and the basic researcher. The clinician makes the diagnosis and treat the disease, the researcher uncovers the mechanisms that cause and modulate it. Nevertheless, if one looks at the big picture, one still finds the lack of interaction between these two areas: the painting is still not uniform. We need the interaction between these two areas: the meeting of the genotype with the phenotype.

Today we have all the necessary technology to study asthma genetics. Many groups have been collaborating and were able to achieve an increase in sample size. In the long run, such group collaboration - alongside with the joint understanding of asthma environmental and genetic features - have allowed for greater asthma clinical understanding and better asthma monitoring. In addition, the clinician and the basic researcher, it is important to think about the patient’s point of view. Asthma manifests uniquely in each patient. The diagnosis is clinical and the signals and symptoms are associated with positive allergy tests and lung function evaluation. Therefore, the younger the patients included in asthma genetics studies, the better will be the understanding of the evolutionary process of the disease in relation to its genetics background.

Every single detail has its own importance and depends not only on individual nuances, but also on the complete comprehensive picture. Step by step, we understand the genetics of asthma and its association with clinical manifestations and severity. Perhaps, at the end, we may discover that different frames should be formed for each analyzed population. Perhaps the studies themselves are not so important when compared to the environment that may be the frame of this picture. Perhaps it is the interaction of both that is responsible for the complete landscape.

Our genome is a huge field to be slowly unraveled. It is an unfinished work, drawn as we discover every gene, every interaction, every feature unraveled. We are rapidly advancing our work, but much remains to be drawn. Asthma is a complex chronic disease with many phenotypes that reflect a clinical individuality and that can also manifest itself with acute exacerbations. Many genetic and environmental conditions are present on asthma phenotypes. The prevalence is high and constitutes a public health problem with high costs for health care [9]. Asthma should be better understood, researched, not only with the public and financial appeal, but also with regarding the particularity that each patient has his own asthma; each patient has his own painting. We have thousands of asthmas, with thousands of phenotypic and genotypic combinations possible.

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