

Genetic Influence on the Development of Food Allergies

Abstract

This paper purports that genetic factors assume the greatest influence over the development of food allergies in humans. A number of researchers argue that environmental factors play a more dynamic role, especially in the early development of the allergic condition. Through an examination of the process of an allergic reaction and an analysis of statistics and recent findings concerning food allergies, this paper intimates that genetic predispositions exacerbate environmental susceptibility to acquiring allergies. Thus, environmental variables exact leading roles only because the genetic makeup of certain individuals facilitates such an occurrence. The culmination of years of research lends support to the incontrovertible quality of this claim. Observation indicates that, essentially, during an allergic reaction, seemingly harmful substances called antigens bind to immunoglobulin E (IgE) antibodies on mast cells and basophils, which are inconveniently located in respiratory and lung tracts (Dawson, 2005). The IgE antibody facilitates the allergic reaction; as soon as the antigens find these antibodies, the mast cells release damaging chemicals known as histamines (Gordon, 2006). Research concerning IgE has linked the production of this antibody to the fifth chromosome and other allergy-related mutations to the second chromosome. As such, the key entities involved in an allergic reaction relate to the intrinsic genetic nature of the body.

In addition to scientific study, much statistical investigation has contributed to connecting food allergies firmly to heredity. Numerous studies allude to some sort of genetic influence in the development of food allergies. Significantly, atopic individuals tend to possess greater amounts of IgE than students who are non-atopic (Bachman, 1992). The more parents who display allergic symptoms, the greater chance that offspring will inherit such a condition (Engel, 1998). Thus, studies conducted at face value, without extensive biological analysis afterwards, also tend

to support the allegation that genetic influence constitutes the principle determinant in the procurement of the allergic condition.

The allergic condition embodies a state of being that many misunderstand and under appreciate. In a broad sense, an allergy disorder induces an adverse reaction of the immune system to certain substances that are characteristically harmless (Clayman, 1989). Allergic reactions occur essentially because the body demonstrates a predisposition to classifying commonplace materials as dangerous (Brody, 1990). In the case that such normally innocuous entities are food substances, such as milk, eggs, and shellfish, a food allergy assumes form (National Institute, 2009). Critically, that which applies to all allergies applies to food allergies since a food allergy fits under the allergy umbrella (National Institute, 2009). According to a study by the American Academy of Allergy, Asthma and Immunology, food allergies have become increasingly prevalent in recent years (Gordon, 2006). However, although a significant portion of Americans think they may be allergic to certain foods, only one to two percent of adults and five percent of children have a veritable food allergy (The American Dietetic, 1998). A thorough understanding of the underlying causes of their acquisition will benefit mankind's efforts to combat these hypersensitivity reactions to food antigens. Although various mitigating factors contribute to the development of food allergy in the early life of humans, genetic predispositions constitute the principal determinants in the acquisition of this condition.

The condition of allergy inherently relates to the immune system, because a breakdown of the latter essentially causes an allergic reaction (Engel, 1998). The immune system's normal role in the body entails forming immunoglobulins (antibodies) and lymphocytes (special white blood cells) that destroy foreign and dangerous proteins called antigens (Clayman, 1989). According to Brostoff and Gamlin (2000), special lymphocytes called B cells manufacture all types of antibodies, including immunoglobulin E (IgE). This one particular antibody actually works as a debilitating force in the body by facilitating hypersensitivity reactions such as allergic

responses to food (Van Gasse, 1995), instead of functioning like a true antibody. As such, an understanding of the great influence IgE exerts on an allergic reaction proves vital to linking the allergy condition to a genetic basis.

IgE antibody molecules attach to FcεRI receptors present on mast cells and basophils as well as lower affinity receptors on non-significant entities (Metcalfe & Sampson, 1992). Mast cells and basophils, which are types of tissue, line sensitive areas of the body such as the lungs and the gastrointestinal and respiratory tracts (Dawson, 2005). IgE antibodies must cross-link to the mast cells and basophils in order for the allergic reaction to occur when the allergen binds to the IgE (Dawson, 2005). In an allergic reaction, once food enters the body, the “foreign” food substance attaches to the IgE antibodies on the mast cells, effecting a chain reaction that results in the ejection of harmful chemicals called histamines (Gordon, 2006). Histamines essentially mediate the common and visible allergic reaction symptoms such as hives, itchiness, sneezing, and anaphylactic shock (Mader, 1990) as a consequence of the aforementioned unfortunate location of the mast cells along sensitive areas of the body. Since IgE profoundly connects with the general process of an allergic reaction, and because a food allergic reaction produces effects equitable to those of a normal allergic reaction, any sort of verified association of the IgE antibody with human genetic nature bolsters the notion that genetic foundations underscore the primary causes of the allergy condition.

The term “atopic” describes any individual that could develop traditional allergic disorders based on excessive amounts of immunoglobulin E in the body (Engel, 1998). According to Astor (1996), every two out of three individuals conducts normal manufacturing of the IgE antibody, preventing excess production and thereby making it impossible for them to develop allergies. Simple math leads to the conclusion that approximately one out of every three

individuals expresses atopy. However, basophils react to the IgE-dependent histamine-releasing factor (HRF) in only 50 percent of atopic people; this phenomenon suggests a heterogeneous nature of the origins of the IgE-related flaw that those with allergic diseases have (Langdon, Lichtenstein, MacDonald, & Rafnar, 1995). Such apparent discrepancy (not every atopic individual exhibits allergy) alludes to the genetic influence on allergies, since variation asserts itself as an intrinsic concept of the branch of heredity. As Table 1 illustrates, there exists a strong correlation between any given child's atopic nature and the number of atopic relatives. The final statistic in Table 1, that if both parents are atopic, then 40-75% of their offspring will be as well, heavily indicates the predominance of genetics in determining whether an individual will develop a food allergy. Interestingly, although studies show that atopy strongly connects with food allergies, 90% of individuals allergic to food substances do not possess any other allergic conditions (Dreskin, 2007). Nonetheless, this statistic does nothing to belittle the concept that atopy indicates a genetic vulnerability to obtaining food allergies.

According to analysis by Brostoff and Gamlin (2000), an atopic individual's incapability of stifling the production of IgE to the harmless substances mistaken as foreign invaders forms the foundation of the allergy condition. Thus, the development of the allergic state boasts a direct function relationship with the amount of IgE in the body, or lack thereof. Bachman (1992) sheds a telling light on the correlation between IgE levels and atopy—IgE generally predominates in individuals with allergy than in those without this condition. Based on these findings, one could deduce that IgE must be inherently associated with some sort of genetic entity in the body, such as a chromosome. In fact, the fifth chromosome of a human's DNA contains the gene that regulates the production of IgE; therefore, a flaw in this gene sets off the chain of events that culminates in an allergic reaction (American College, 2000). Additionally,

two mutated variants of an amino acid IgE building block chain associated with a gene on human chromosome two aptly underlie a genetic inclination to developing food allergies (Pennisi, 1994). Mutations on genes thus instigate the unfettered manufacturing of IgE, which subsequently predisposes an individual to acquiring an allergy. Therein lays substantial proof that genetic factors constitute the prime determinants in developing a food allergy.

Table 1. Predisposition to atopic allergic states

This table emphasizes the relationship between family atopic history and the genetic makeup of children by showing that if more family members exhibit the allergic condition, then a child has a greater chance of displaying atopy.

Atopic family members	% of Atopic Children
None	5-10%
One sibling	25-35%
One parent	20-40%
Parent and sibling	40-45%
Both parents	40-75%

Note. These data are adapted from Shan, 2007.

Some researchers contend that environmental factors such as hygiene and early exposure play more superior roles than genetic variables in determining whether or not an individual acquires allergies, including food allergies. Dr. Robert Wood of the Johns Hopkins Medical Institution purports that the prevalence of allergy will rise in developed countries because the absence of germs in these regions will, in a sense, leave allergens more prone to attack by a hyper immune system (Gordon, 2006). Additionally, some studies have shown that the earlier an individual consumes a certain food substance, the more likely that this individual will develop an adverse reaction to this food (Gordon, 2006). The latter assertion stems from the accurate observation that first-time contact with potentially harmful food materials serves the purpose of sensitizing the immune system for successive encounters with these allergens (Engel, 1998), while the second and subsequent exposures induce the actual allergic reaction (Arms & Camp,

1982). However, when such statements advertise themselves to be the principal determinants in the development of food allergies, they assume a false guise. Belief in the influence of environmental factors has been lost to the receding wave of history; modern research downplays their significance by pointing out that environmental variables contribute to allergy development, but only if genetic mechanisms create a predisposed immune system (Brostoff & Gamlin, 2000). A genetic foundation opens the door for environmental influences to take hold of those individuals likely to develop food allergies. Ironically, the same Wood (2001) who suggested that food allergies might link to hygiene conducted a study of twins with veritable histories of food allergies that yielded interesting results: in 64.3% of the pairs of monozygotic (identical) twins tested, both children had a peanut allergy, whereas in only 6.8% of the dizygotic (fraternal) twin pairs did both children have a peanut allergy. Since monozygotic twins develop from the same egg, these results provide considerable evidence of the extensive influence of genetics in the development of not only a peanut allergy, but also all food allergies. The environment plays second fiddle to the powerful laws of genetics.

Observations of family history and the allergic tendencies of the offspring of allergic parents also lend key support to the theory that genetic mechanisms exact crucial roles in the development of food allergies. According to Gordon (2006), an individual expresses more susceptibility to acquiring a mutated immune system inclined to triggering a hypersensitivity reaction to an allergen if any ancestor has allergies. Figure 1 illustrates the chances that a child will develop a food allergy if none, one, or two of the parents has an allergy. Significantly, Figure 1 aptly shows the sharp increase in chances between one and two parents exhibiting allergic tendencies (30% to 70%)—this suggests a firm link between heredity and allergies since a combination of atopic genes would produce more atopic offspring. Although family history

plays a critical role in establishing a vulnerability to allergies in posterity, exposure to a particular allergen determines the specific allergy in a child—that is, an allergy does not necessarily mirror the allergy of the allergic parent (Astor, 1996). Hourihane, Dean, and Warner (1996) conducted a study in which they analyzed family history of peanut allergy, the most common and fatal variety of food allergies. This research yielded significant results: peanut allergy was more predominant in successive generations and it appeared that offspring inherit it more strongly through the maternal line (Hourihane et al., 1996). Such findings hint heavily at a genetic mechanism at work, such as recessive heredity, in which the tendency towards acquiring food allergy passes down through the line but does not display itself in an individual's phenotype (Hourihane et al., 1996). These conclusions would also explain the recent rise in the number of reported cases of peanut allergy in industrialized countries (Brandtzaeg, 2007).

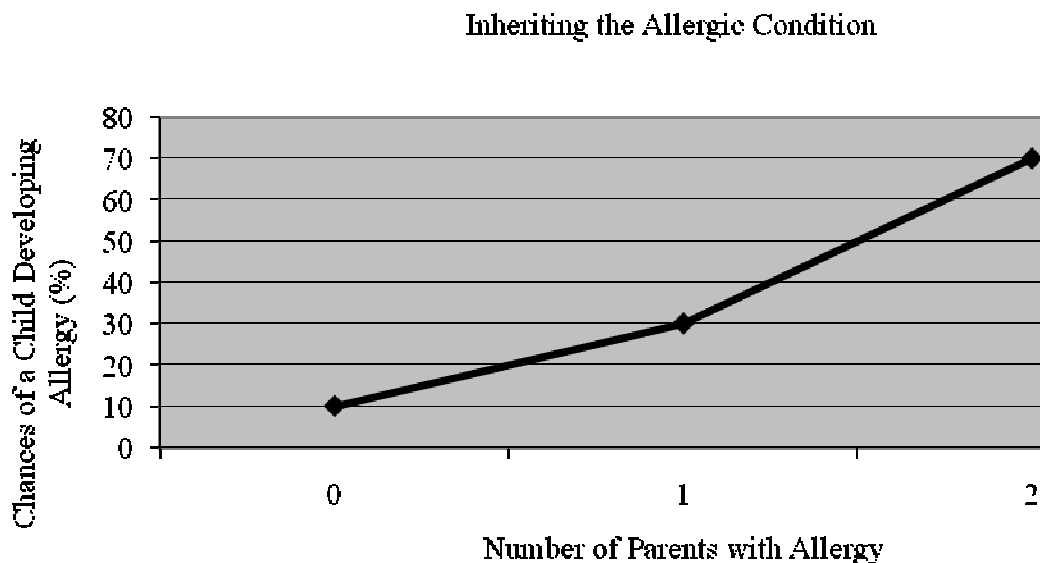


Figure 1. This graph depicts how the number of parents with allergy significantly relates to the expected percentage of children who will inherit this condition by illustrating the notable and non-linear increase in the chances of a child developing allergy between having no parents and both parents with allergy.

Note. These data are adapted from Engel, 1998.

Zwirn (2009) contends that all types of allergic diseases, including food allergies, have become increasingly prevalent since the early 1990s. A comprehensive analysis of the genetic-based workings of an allergic reaction, a disproving of the argument that environmental factors play more critical roles, and a consideration of statistical and unbiased studies all allude to the reality that genetic variables assume the lead roles in facilitating the development of food allergies in humans. This contention contains several implications for the future of dealing with food allergy. Currently, the best remedy for the condition necessitates complete avoidance of any food substance that may trigger a hypersensitivity reaction (Bachman, 1992). Thus, by concentrating on the specific flaws in the genetic makeup of atopic individuals, allergists may eventually find a panacea for all types of food allergies, or at least different remedies for each kind. As the instances of food allergy become increasingly bountiful, more reliable research will assert itself at the forefront to accurately pinpoint the precise regions of chromosomes two and five that associate with predisposition to food allergies. Furthermore, continuing study of IgE could lead to so-called anti-IgE therapy, in which treatment reduces IgE levels in the body (Gordon, 2006). Objectively, finding a cure for food allergies would signify a novel epoch in the human pursuit of establishing further control over genetic inconsistencies and a better understanding of the human body. Subjectively, establishing that genetic vulnerabilities account for the primary determinants in the development of food allergy constitutes the first step towards liberating a suffering group of individuals from a debilitating and life-threatening condition.

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