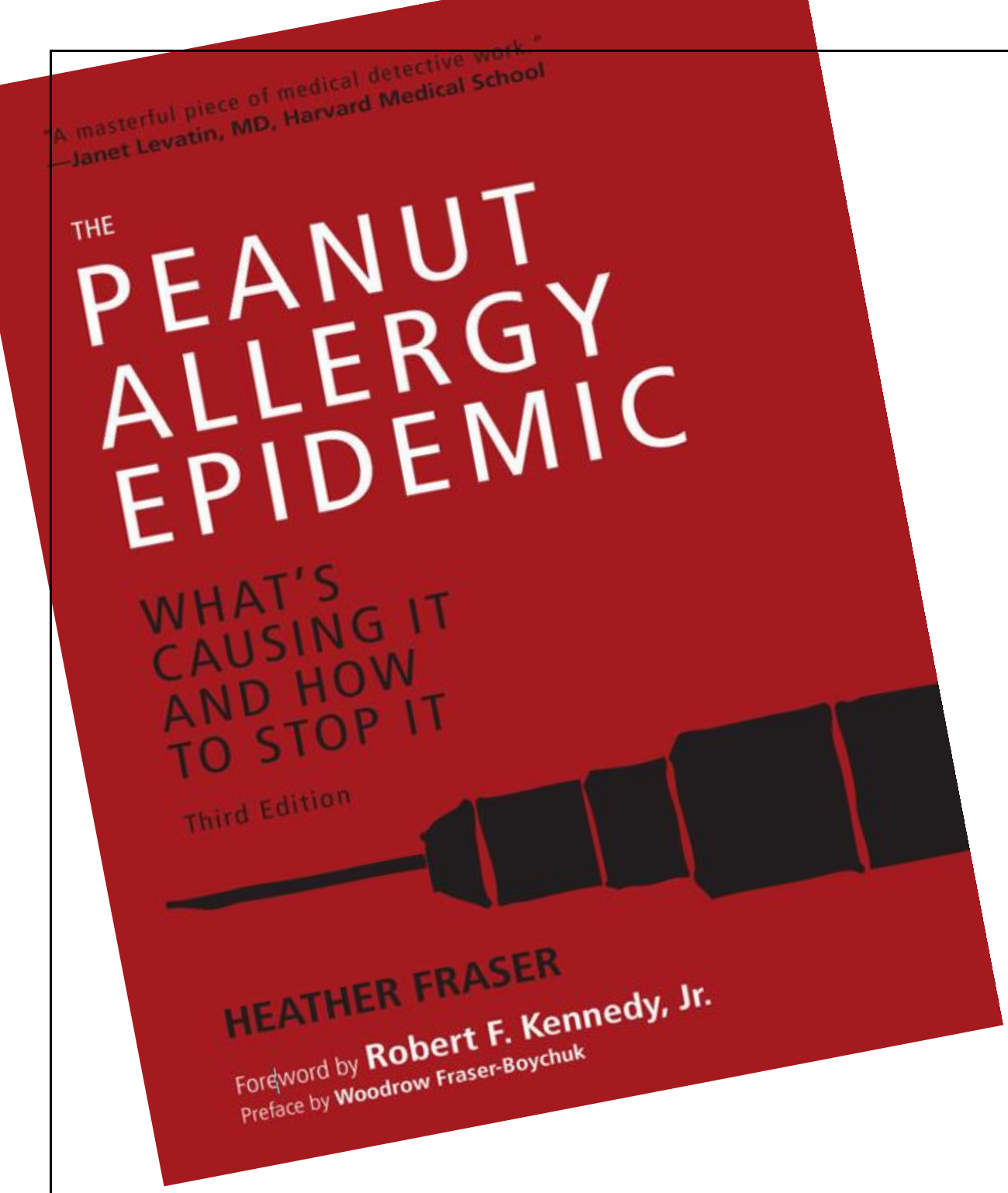

The Peanut Allergy Epidemic

What's causing it and how to stop it

By Heather Fraser

Book Summary by **Lies are Unbekoming**

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Cover letter

Hello there

If you are reading this, you've probably already realised that you haven't been told the whole truth about vaccines and you are curious to find out more.

The reason I produce these summaries is to make the subject matter a bit more accessible, to act as a gateway to the whole book (please buy it and support the author) and/or further reading on the subject.

The model I have used to summarize each part of the book is:

- Executive Summary
- Key takeaways
- Excerpts

Here is the Author's Note from the original book:

> "Only puny secrets need protection. Big discoveries are protected by public incredulity."
>
> —*Marshall McLuhan*
>
> Information is instant, constant, and exists all around us. This book is a product of the era of information and communication: an individual can find an answer to any question, if he is motivated.
>
> I was motivated to write this book by an event for which I was completely unprepared. In 1995, my firstborn son at thirteen months of age had an anaphylactic reaction to peanut butter.
>
> I wanted to know why.

I have left the Foreword, Preface, and Introduction un-summarized.

With that, I hope you enjoy this summary, you might want to visit my Substack and/or share this summary with others.

Regards

Lies are Unbekoming

unbekoming.substack.com

August 2023

Foreword

My son Conor was born in 1994. He developed chronic asthma, food allergies, and anaphylaxis that required twenty-nine emergency room visits before he was three years old. His brother Finn, born four years later, also developed anaphylaxis. What were the chances? Neither their mother nor I had any allergies. I'm not even allergic to poison ivy!

Epidemic allergy in our children emerged suddenly, then ramped up inexorably, through the 1990s, in every American community. Its impacts affect our businesses, public spaces, transportation, and schools. Without explanation, children with severe allergies to peanuts, milk, fish, bee venom, latex, pollens, and more were filling our classrooms. Peanut allergy is now so prevalent that it impacts everyday consumer behavior; 30% of polled mothers said that nut allergy influenced a food purchase decision and 67% considered nut allergy when buying snacks for an event with children.

We have all watched in bewilderment as peanut allergies among children skyrocketed from 0.8% in 2002 to 1.4% in 2008. Just three years later in 2011, another study indicated an incidence as high as 2.8%. Upwards of two million children in the United States are now allergic to peanuts. Schools learned to protect vulnerable kids with cupboards filled with EpiPens and puffers, hand-washing protocols, food bans and exempt spaces; inconveniences that sometimes annoy non-allergy families, but provided some reassurance to worried parents like me.

Initially, researchers believed peanut allergy was unique to western countries, specifically the United Kingdom, Canada, the United States, and Australia. Then, suddenly, similar epidemics exploded in Singapore, Hong Kong, and Africa. Where was this coming from? I knew the answer could not be solely genetic. As my friend, Dr. Boyd Haley, chairman emeritus of the University of Kentucky Department of Chemistry, says, "Genes don't cause epidemics. Genes can provide the vulnerability, but you need an environmental toxin." But what toxin had launched the allergy cascade?

In 1998, I was among a group of New York parents who co-founded the Food Allergy Initiative (FAI) to research cures for food allergies. FAI did extraordinary work in developing treatments, but I remained intensely curious about identifying the cause. In my own research, I learned that a host of other childhood epidemics—autism, ADD, ADHD, SIDS, OCD, ASD, narcolepsy, sleep and seizure disorders, neurodevelopmental delays, autoimmune diseases, and tics—all began rising in the early 1990s. Coincidentally, this is the time period during which the Center for Disease Control and Prevention (CDC) dramatically expanded the vaccine schedule, raising children's exposure to mercury, aluminum, and other toxic vaccine ingredients. Public health authorities expanded the schedule, including the battery of metal-laden vaccines, first in the United States, the United Kingdom, and Canada—and subsequently in Asia and Africa—along a timeline exquisitely correlated with the explosion of these new childhood disease epidemics, in these locales.

Abundant peer-reviewed science linked mercury, a known neurotoxin, to the neurodevelopmental injuries that now, according to the CDC, afflict one in six American children. In 2013, I wrote a book assembling and synthesizing hundreds of these studies. As I searched the medical literature on PubMed, I occasionally came across studies linking these contaminants to anaphylaxis, asthma, allergies, and eczema. While the timing of the anaphylaxis explosion suggested a link to the sudden expansion of the vaccines schedule, the published science on its origins was surprisingly sparse.

Scientists and public health officials have offered numerous theories purporting to explain the epidemic: the hygiene hypothesis, GMOs, glyphosate-based pesticides, environmental pollutants, or peanut mold, for example. But the proliferations of these exposures did not

neatly coincide with the allergy epidemics. None of these potential causes explained the sudden meteoric rise of anaphylaxis in the early 1990s.

Seeking answers that might help end the epidemic, the National Peanut Allergy Board, along with Food Allergy Research and Education (FARE) and the National Institutes of Health (NIH), funded the LEAP study (Learn Early About Peanut Allergy) in 2010. However, the study, which was published in 2015 in the *New England Journal of Medicine,* sheds little light on causation. We learned only that children at risk of developing peanut allergy were those who already had severe egg allergy and/or eczema. Serious allergy can lead to more allergies? That struck me as a circular conclusion; it brought us no closer to answering the riddle: What was triggering the cascade of allergy epidemics in the first place?

In the summer of 2016, I asked Dr. James Baker, head of the nonprofit FARE—formerly the FAI—and former senior vice president of Merck's vaccine division, whether vaccines could be a cause of the food allergy epidemic. He told me that it was a possibility that should be looked into. But, "unfortunately nobody is studying that at the moment." Dr. Baker's candor was refreshing. Most scientists understand the radioactive peril of raising questions that implicate vaccine safety. Scientists, like doctors and journalists, know that vaccine debate can be a career-ending minefield. For that reason, scientists interested in allergies tend to cluster around safe harbors like genetic studies or behavioral research. Funding is easier to find in these arenas, scientific journals are willing to publish such works and careers remain on trajectory. Unfortunately, such research has gotten us no closer to understanding the etiology of the epidemic.

Around the time of my discussion with Dr. Baker, I stumbled on Heather's book. I devoured it ravenously. Heather has done for allergies what Jared Diamond did for broader concepts of human history in his book, *Guns, Germs, and Steel*. Although she is not a scientist, she used her gift for synthesizing a wide range of scientific disciplines and history to arrive at a simple, coherent, integrated, and commonsense hypothesis that, for the first time, convincingly explains the sudden onset of the allergy epidemic. Her theory accounts for its steep rise in America in the early 1990s, and its subsequent movement from western nations into the developing world.

Heather tracks the allergy epidemic's causes to US political, legal, and medical reforms in the late 1980s. In 1986, the US National Childhood Vaccine Injury Act (NCVIA) and National Vaccine Injury Compensation Program (NVICP) eliminated liability for vaccine manufacturers; thereby creating a gold rush among vaccine makers. Any company shrewd or connected enough to persuade the CDC to add their products to the recommended schedule could cash in on the $30 billion vaccine bonanza. The vaccination schedule expanded rapidly without anybody performing comprehensive safety analyses or testing for possible negative synergies from the battery of multiple early immunizations. Nor did public health regulators make any effort to perform a mass loading calculation of the combined mercury and aluminum exposures, from so many new vaccines.

Having read Fraser's analysis, it seemed almost strange to me that no one had previously connected the dots between the greatly expanded vaccine schedule and the anaphylaxis crisis that preceded. Heather reveals how the answer to the anaphylaxis puzzle has always been buried in plain sight—in the accounts, detailed observations, and comments made by late nineteenth- and twentieth-century vaccine pioneers—concerning the tendency of vaccines to initiate anaphylaxis and allergy. Those pathbreaking vaccinologists had quickly discovered that injecting an allergen directly into the bloodstream, bypassing the digestive process, often provokes anaphylaxis. Medical literature shows how powerful adjuvants (most notably, aluminum), added to vaccines in order to increase the immunological response, also amplify the allergic response. Adjuvants promote allergic sensitization not just to vaccine antigens, but also to other "bystander" proteins and allergens in the ambient environment at

the time that the adjuvanted vaccines were administered. Thus, a child vaccinated with aluminum adjuvants during a spring ragweed outbreak, may develop a lifelong allergy to ragweed. Similarly, a child immunized with a vaccine containing peanut oil, or similar proteins, may develop a deadly allergy to peanuts. Further, and as noted by the LEAP study, once a child has developed allergies there is a tendency to develop more.

It did not elude me that the same pharmaceutical companies that have transformed vaccination into a lucrative enterprise have also profited with additional billions on the back end, selling $600 EpiPens (two-pack), as well as puffers, nebulizers, steroids, Benadryl, and other medicines to injured children.

In August, 2016, I invited Heather to give a presentation to the Food Allergy Science Initiative (FASI) at the Broad Institute in Cambridge, Massachusetts. A prestigious group of well-funded and experienced scientists had gathered at MIT to find a way to "turn off" the allergic reaction by identifying genetic precursors to allergy. This is good and important work, but I wanted them to hear and understand Heather's story of the peanut allergy from a historical perspective, one that was supported by a rich library of medical literature. Using a series of vivid slides, Fraser clearly explained the provocative role of aluminum adjuvants, conjugate vaccine technology, and combination vaccines in the allergy explosion.

Fraser's presentation was strong and convincing, but it raised only mild interest among the scientists. They were not the least bit skeptical, but, they explained to us, "This is not what we do." Their expertise, they said, were human genes. When I questioned them, they acknowledged that the allergy epidemic must have been triggered by some environmental toxin, and that their research was unlikely to identify the culprit. They told me that they did not need to know what caused the peanut allergy in order to find a treatment. I began thinking that the refusal to look at the possible causal role of vaccines was like trying to find a genetic cause for sunburn without considering the role of the sun. Their work is important but while these scientists were trying to save the children downstream, I wanted to prevent a new generation of children from being pushed into the river in the first place. I believe that the reluctance among scientists to look at the role of vaccines is prolonging the epidemic and damaging the public's health.

Finally, for those of you who, like me, want to solve the food allergy mystery, Heather's book cracks the code.

Robert F. Kennedy, Jr.

President of Waterkeeper Alliance

Chairman of the World Mercury Project

Preface

Peanut" has been a dirty word in my house for longer than I can remember; as if we were a family of wizards cowering in the shadow of Voldemort; as if by speaking the profanity we would bring down a mortal curse on our first-born son (myself). My mother can barely stand to imagine myself and a peanut in the same mental frame. It's no wonder; she's seen me on the brink of death four times (too many) and swollen like a balloon, with my dignity battered, after losing a duel with the dastardly plant. Of course, the taboo around the word has softened as I've grown older and more capable of self-defense, and am out of the home for most of the year. Even so, my mother's mind is still visibly set on edge if I tell her I've forgotten to take my EpiPen anywhere with me.

As few as twenty-five years ago, only at the absurdist theatre would one expect to meet such a family—a family which lives in constant fear of a small, crunchy hunk of cellulose. Unfortunately, this has become strange reality for many more families than my own.

"Anaphylactic" literally means defenseless; giants though we are, the smallest particle has the power to topple us. For an anaphylactic, the shadow of death skulks in the silliest places: vending machines, refrigerators, ice-cream parlors, and parks where nice old ladies feed squirrels. The mundane randomness of the peanut, as well as milk, eggs, and other embarrassingly benign foods that our bodies lethally reject, allow them to slip beneath the radar of the common mind. People appear to have an easier time empathizing with a diabetic or cancer patient—diseases where the enemy is one's own body, which must be constantly moderated, used gingerly, and plied with medicine; a situation we've all experienced in times of illness. However, to the average onlooker I imagine an anaphylactic must look like a barefoot Achilles carefully avoiding sharp stones on the ground, or King Stefan of *Sleeping Beauty* obsessively purging his kingdom of doomful spinning wheels. Oftentimes, I feel embarrassed about how silly my own requests for accommodation sound. After a new acquaintance has invited me into his home for the first time, I find myself asking him to move a bag of mixed nuts from the living room to the kitchen. If he doesn't answer my request, instead of insisting I just move to the far end of the couch, and take care not to touch my face with my fingers for the rest of the night.

My best anaphylaxis story (I say that only half-ironically) is about the first time I had dinner at my girlfriend's house. Like the careful person she was, she instructed her parents not to cook with any nuts. Needless to say, it is rather uncouth to kill your daughter's boyfriend. We sat down and I began to endure the sincere rituals of meeting the parents. I then noticed that I had begun to have an allergic reaction—how awkward. Since my girlfriend had assured me that no nut products (and especially, no peanuts) had entered the meal, I suspected that it was likely just an accidental sliver of almond that had snuck into the lamb or the salad, and I resolved to silently weather my reaction for the sake of my relationship with her parents. They were native Chinese; China being a country where allergy rates have historically been miniscule. I didn't want to cause a fuss over an itchy throat, so I would just eat around … whatever it was that I had eaten. Almost half an hour later, after the meal (with dessert, no less!), I felt nauseous, so I excused myself to the upstairs bathroom. Looking in the mirror, I observed that I was bright red. I was up against no mere almond.

Five minutes later, her father was racing me to the hospital. I did my best to keep her calm as we sat in the back seat, even as my face gradually became more inflated and hideous in front of her.

The instigating food item had been a precooked onion pancake that been cooked in peanut oil. I had eaten the same dish, cooked in different oil, a week earlier, and presumed this time it would also be safe. It was an honest mistake on their part; perhaps they had never experienced eating with an allergic person before.

I survived the event, as any adequately prepared anaphylactic would (thanks for reminding me to bring my EpiPen, mum). I was able to keep cool the entire time because I had done it before (I hadn't died any of the other times), and I figured my luck would hold out this time as well. And hold out it did—my girlfriend later told me I had impressed her father with my stoicism.

That incident was the fourth time I had almost been killed by my condition. (There has since been a fifth, which my mother won't know about until she reads this preface I've written here.) I've already accepted it as something that would happen to me from time to time, like a monkey learning that tigers are a fact of the forest. I've been content my entire life constrained in my circumstances simply because I've known nothing else.

Besides, there are plenty who have it much worse than me. There are cases of children who must be carefully isolated, usually entailing homeschooling, because they are sensitive to such a vast array of things (they will likely live in a bubble for their entire lives). At least I have the luxury of being able to visit my girlfriend's parents' house—and the luxury of being unafraid to touch doorknobs.

Whereas I've always been very nonchalant in my approach to my allergy, my mother has refused to accept my absurd reality at every turn. For many years, she's been battling the notion that this is the way that things must be.

Young people, myself included, tend to think of ourselves as unkillable protagonists covered in impenetrable plot armor. Parents are smarter than their children; they have been the narrators that kept their main characters from dying throughout their childhood and they have known just how easy it can be to lose something. Any risk factors must be eliminated; that's why my mom cares so much about getting to the root of this phenomenon. Even if it's too late for her to live a motherhood without the constant threat of losing a child, and too late for me to live a childhood where I get to eat peanuts (I expect they taste amazing, or you all would have no excuse for eating them), perhaps we can help the parents of the future and make a world without bubble children or Sabrina Shannons.

Since 1990, the peanut-allergic population of the United States has more than tripled, now numbering a tragic three million individuals. We know how to treat it, we know how to mitigate it, but we still haven't agreed on what causes it, let alone figured out how to cure it. Large-scale anaphylaxis has not always been a fact. Anaphylaxis occurs rarely in many parts of the world; this may indicate a short-term, likely human cause historically unique to westernized countries. If this is the case, it is baffling that we haven't discovered it already.

Woodrow Fraser-Boychuk holds a BA in creative writing & English language and literature from Western University.

Introduction

THE PROBLEM OF PEANUT ALLERGY

By 2012, as many as 2.3% of Canadian children under 18 and 2% to 3% of children in the US, the UK and AU were allergic to peanut. (See Appendix). And as children born during the first wave of the epidemic in the early 1990s have aged, the statistic of adults with peanut allergy is increasing. In 2008, an estimated 1% of the US population was allergic to this one food, about 3 million people. Four years later by 2012, that number jumped to an estimated 4 million living with a life threatening allergy to peanuts.

Peanut allergy began as a phenomenon largely affecting children living in Western countries, the US, Canada, Australia and the UK. The alarm sounded for Americans when between 1997 and 2002 the number of peanut allergic children doubled and then tripled reaching an astonishing one million in 2008. In 2010 one study put that number at 2%, an additional 500,000 children in just two years. As this book unfolds it will become evident that there is a pattern in the way in which the peanut allergy in Western and now non-Western countries has emerged—epidemic levels of peanut allergy in children are now also documented in mainland China, Hong Kong, Singapore, Israel and parts of Africa.

While the exact numbers are a matter of debate, it is clear through statistics, scientific inquiry, and simple anecdotal evidence (the parental refrain “no one had a peanut allergy when I was at school”) that the prevalence of the allergy among children has increased at an *alarming* rate. This development has altered the fabric of societies now forced to accommodate life-threatening allergies to common foods.

Families with children allergic to peanuts (or any of the other top 8 allergenic foods—tree nuts, fish, shellfish, wheat, soy, dairy, egg) live in a state of constant tension. If these families eat at restaurants, they do so with extreme caution. Not knowing the severity of the allergy, parents are vigilant about smears of peanut butter left on tables or on grocery cart handles. Trace amounts on the skin or lip or even the scent of the food could trigger a reaction. Parents, the child, caregivers, and teachers are fearful. Children are segregated in school cafeterias at designated tables or left out of play because friends have peanut butter in the house. Every school now tackles the peanut question, whether to ban peanut butter sandwiches and how to educate staff and students about the deadly nature of this ubiquitous childhood food.

Public awareness of peanut and other severe food allergies has impacted education systems and social norms, provoked legal reform, and made billions of dollars for those active in the food-allergy industry. This industry’s infrastructure consists of many overlapping allergy awareness groups, international allergy associations, medical researchers, pharmaceutical companies, allergy doctors, “free from” food makers, and government regulators, all of which support or are supported by the growing legions of food-allergic children.

The inherent inertia of this industrious leviathan, however, has pushed the salient questions into the background: How has the peanut allergy epidemic developed, and why is it continuing?

It is difficult to accept the startling increase in peanut allergies in children in just the last twenty years as a coincidence or to chalk it up to genetic fluke. The challenge for any concerned medical professional has been to unearth the precise practical mechanism of sensitization common to these children—how did they become sensitized to peanut in the first place? And while there are a limited number of proven ways of “how to” make someone anaphylactic—ingestion, inhalation, through the skin, injection—no hypothesis of *mass sensitization* has yet connected any of these functional mechanisms to all the specific characteristics of the peanut-allergy epidemic.

Researchers have considered skin creams that contain peanut oil, peanut consumption, parasite burden, and more without satisfactorily explaining why there has been a rise of the allergy in children. Why peanut? Why has it happened so suddenly, and why just in certain countries, most of them Western? Risk factors for developing the allergy have been explored without conclusion. These include the following: maternal age, mode of delivery, levels of intestinal flora, heredity, and even birth month and socioeconomic status. Confusing matters further is a debate over the basic concept of allergy: Is allergy the outcome of a roulette-style genetic predisposition to immune dysfunction, or is allergy an innate, purposeful immune defense?

An important and clear distinction must be made between *sensitizin*g someone to peanut and *launching* the allergic reaction. Sensitization is believed to occur when a protein bypasses the detoxifying process of the digestive system and becomes bonded with blood serum. This prompts specific blood cells to create antibodies that are then programmed to recognize the threatening protein—in this case, peanut protein. The launching of an allergic reaction, on the other hand, occurs when the body is subsequently exposed to the protein and the antibodies trigger the biochemical players in the allergic reaction.

Lack of a standardized definition of anaphylaxis has hampered some studies where categories of "true" anaphylaxis mediated by Ig antibodies are compared with non-Ig anaphylaxis. This is less of a concern with peanut allergy where apparent consensus is that it is almost always Ig mediated.

Immunoglobulins epsilon (called IgE) are sentries of the body. The job of the IgE is to patrol the fortress walls—mucous membranes—looking for peanut protein intruders. When they detect one of the many peanut protein epitopes (strings of amino acids that are numbered 1 through 8 and all called Ara h after *Arachis hypogea*, Latin for peanut) they alert the body, which in turn lets loose the army—the body's immune system. A biochemical cascade is deployed that is damaging and potentially dangerous. It is typically characterized by coughing, shortness of breath, itchy skin hives, systemic leaking of blood vessels that causes swelling and potential asphyxia, vomiting, and diarrhea. In severe reactions, blood pressure drops, draining vital organs and causing the heart to stop.

Scientists have shown that the anaphylactic condition in all mammals can be achieved by inhaling peanut protein if it is combined with a toxic additive. For example, doctors have created anaphylaxis in lab animals that inhaled a mixture of peanut and cholera. The toxic bacteria functions as an adjuvant, an additive that excites the immune system to form antibodies. It is suggested that the toxin and benign food can become in this way linked and both remembered by the immune system. One wonders then at the idea of an allergy to bacteria and the toxins produced by them. Allergy to bacterial toxins has been acknowledged for many years and can result in inflammation of the tonsils and adenoids and anaphylaxis.

Researchers have not explored the role of adjuvants in peanut sensitization. They have preferred to focus only on the peanut proteins, their allergenicity, and the ingestion of them as the most obvious elements in sensitization. They seemed to think that if they could simply pinpoint the initial oral exposure to these proteins, they could stop the epidemic. To this end, they have considered the ways in which peanuts are prepared (boiled versus roasted), age when they are introduced to the child's diet, maternal diet and breast milk, and even peanut oil used in nipple creams. Although it is possible to create the condition through simple ingestion, it is difficult. A healthy digestive system will neutralize any potentially sensitizing protein.

In fact, a 2006–2007 study stated that it did not matter whether mothers ate peanuts or not—the same percentage of children developed the allergy. Some children whose mothers did not eat peanuts before, during, or after pregnancy still developed a peanut allergy. Kids who

had never been exposed to peanuts exhibited anaphylaxis on their first or second taste of it—suggesting that they were already sensitized either to peanut proteins or to proteins similar enough to them leading to cross-reactivity. Adding to the allergy mystery is the fact that Sweden, which has a low level of peanut consumption, has a higher prevalence of the allergy than the United States. Israel, which has a high level of peanut consumption, has a low prevalence of peanut allergy in Jewish children at 0.6% in 2012 (but a high prevalence of sesame allergy) and a high prevalence of peanut allergy in Arab children (2.6%) living in the same country.

Another puzzling feature of the epidemic is the sudden emergence of peanut allergy in non-Westernized countries like Ghana, China and Singapore. It was suggested previously by Sampson et al in 2001 that children living in China did not have peanut allergy because their peanuts are boiled which partially destroys and reduces sensitizing peanut proteins. However, the sudden and increasing prevalence of food allergy in children living in mainland China and peanut allergy Hong Kong upends this theory and deepens the seeming mystery of this allergy.

Today, thousands of research articles by doctors on the biology of the allergic reaction, clinical observations, and allergy management are available in prestigious periodicals. From this mound of information, doctors have developed and tend to favor two explanations for the current epidemic of peanut-sensitized children. They are the helminth hypothesis and the hygiene hypothesis.

Helminths are worms that live in the human intestinal tract. It surprised researchers in the 1980s to discover that people heavily infected with worms had few allergies. One study confirmed that most Venezuelan Indians living in the rainforest had worms but no allergies while very few of the wealthy Venezuelans living in the cities had worm infections, but many had allergies.

From this observation, researchers developed an explanation for all allergies: because parasites and humans have coevolved, they have an apparent symbiotic relationship in which parasites suppress allergic reactions while enjoying their human host. Without worms, the theory states, humans are unable to achieve homeostasis. In other words, immune dysfunction occurs due to lack of worms.

As an explanation for peanut allergy, the helminth hypothesis is inadequate. It cannot explain why there has been a rise of peanut allergy just in children. And given that Western countries have been largely unburdened by major helminth infections for decades, it does not explain the sudden increase of food allergy that shocked school systems in the early 1990s.

Another popular explanation for the rise in childhood allergies grew from an apparent correlation between this rise and the general decline in family size. It was proposed that unhygienic contact in large families—lots of siblings bringing illness home from school—was important for the development of a healthy immune system. The greatly expanded and much-touted hygiene hypothesis suggests that overzealous cleaning, germ-killing products, chlorinated water, antibiotics, (vaccination is specifically avoided by researchers) have “protected” Western children unnaturally. And as a result, the immune systems of First World children, in particular, are sheltered from a natural microbial burden. Their immature immune systems are under stimulated, dysregulated, and therefore prone to random allergic sensitization. This malfunction is a product of an unburdened lifestyle.

The hygiene hypothesis is problematic in explaining peanut allergy. It does not consider the possibility that the immune systems of these children are not under stimulated but rather overstimulated by Westernized approaches to toxic chemicals, drugs, and vaccinations. In addition, the theory does not indicate a practical mechanism of mass sensitization that would explain the sharp rise in food allergy just in children that was first noticed in the early 1990s

in specific countries when a flood of affected children arrived for kindergarten. This is a primary clue to causation that researchers have either missed or dismissed altogether.

In addition, these two favored explanations for the epidemic assume that allergy is a dysfunction, that the body has made a mistake in attacking a benign substance. And yet, the opposite may be true. Some suggest that allergy has an evolved purpose seen before the twentieth century but provoked increasingly today by drugs and noxious pollutants in our air, water, and food.

American researchers Rachel Carson (1907–1964) and Theron G. Randolph (1906–1995) and evolutionary biologist Margie Profet (b. 1958) proposed that allergy is an evolved protective response. In 1991, Profet stated in *The Function of Allergy* that allergy is a final and often risky natural defense against toxins linked to benign substances. The IgE antibody is not, as it is generally characterized in medical literature, a rogue immune factor. It is more akin to a hero provoked by toxins the body has deemed a deadly threat. The scratching, vomiting, diarrhea, and sneezing are desperate attempts to eject a toxin as fast as possible. It is a risky reaction but one the body is programmed to unleash as a last-ditch effort to protect itself. This event occurs when the general defenses have been insufficient in preventing a specific toxin from accessing the bloodstream for a second time.

This is a provocative concept. However, because it was developed before the rise in peanut allergy, it lacks specificity—again, why peanut and why the sudden increased prevalence in children?

Conspicuous by its absence from current theories is the one mechanism that has an actual history of creating mass allergy—injection. Injection is examined in this book in some detail since it was the means by which the founder of anaphylaxis, Dr. Charles Richet, stumbled on alimentary (food) anaphylaxis in humans and animals over one hundred years ago. Richet concluded in 1913 that food anaphylaxis was a response to proteins that had evaded modification by the digestive system. Using a hypodermic needle, he was able to create the condition in a variety of animals—mammals and amphibians—proving that the reaction was not only universal but also predictable using the method of injection followed by consumption or another injection.

There are two lines of thought in the medical literature regarding injection as a mechanism of sensitization. The first is that injection, in the form of vaccination or other injections such as the neonatal vitamin K1 prophylaxis, merely *unmasks* genetic predispositions or tendencies to allergic disease. In short, there is something wrong with the child and not the injection(s).

The second line of thought is that there is a causal relationship between the injected ingredients and allergy—and although the proven allergenicity of vaccines is widely acknowledged, medical literature carefully avoids the question of what kinds of allergies vaccines can and do create to substances that are coincidentally or subsequently inhaled, ingested or injected. One exception to this unwritten rule was an unusual admission by Japanese doctors that an outbreak of gelatin allergy in children starting in 1988 and continuing through the 1990s was caused by pediatric vaccination. In that year, changes to the vaccination schedule in Japan meant that the DTP was replaced by an acellular version containing gelatin, the age at which it was administered to children was dropped from two years to three months, and this new vaccine was given before the live virus MMR vaccine that also contained gelatin. When children began reacting with anaphylaxis to the MMR vaccine as well as gelatin-containing foods (yogurt, Jell-O, etc.), doctors investigated. Finally, they concluded that the aluminum adjuvant in the DTaP had helped sensitize children to the “minute amounts” of proteins in the refined gelatin in the vaccine. Removal of gelatin from the DTaP vaccines was “an ultimate solution for vaccine-related gelatin allergy.” Subsequently, new cases of gelatin allergy in Japanese children dropped.

Quantities and qualities of adjuvant and other vaccine ingredients injected into children changed dramatically between 1989 and 1994 in 'mature markets' for vaccines including the United States, United Kingdom, Canada, and Australia. During those years, at least five new vaccine formulations for the same bacteria, *Haemophilus influenzae* type b (Hib) were introduced within an expanded and intense vaccination schedule. Like the gelatin allergy that emerged from a changed schedule of pediatric injections, was there some mix of ingredients that included powerful aluminum additives in the new Western schedule that was sensitizing children to peanut? The fact that refined peanut oil was a documented vaccine ingredient in the past is a subject of concern equal to the potential of sensitization to body tissues or even of cross-reactivity between dietary peanut and homologous injected proteins. These cross-reactive proteins may include those in the Hib cellular membrane or legume oil in a popular brand of the vitamin K1 prophylaxis. Cross-reactivity explains why a person who is allergic to peanuts, legumes like soy and castor beans, may also react to nuts or citrus seeds, which belong to different plant families—their proteins have similar molecular weights and structures.

As ingredients changed, the number of shots increased for kids in their first eighteen months of life from ten to as many as twenty-nine. The increase meant inconvenience to parents who would have to make more trips to the doctor and discomfort to the children who would have to experience multiple injections. To overcome these obstacles to compliance with the new schedule, the vaccines for diphtheria, pertussis, and tetanus (DPT); polio (OPV); and *H. influenzae* b (Hib) were administered to children in a single visit with two injections and an oral polio dose starting around 1988. By 1994 starting in Canada, these five were rolled into a single needle. Few parents realize that by design, immunization provokes both the desired immune response and allergy at the same time. These natural defenses are inseparable and the more potent the vaccine, the more powerful the two responses. This is an outcome of vaccination the medical community has understood at least since Charles Richet won the Nobel Prize (1913) for his research on anaphylaxis. Anaphylaxis, Richet observed, is one of three outcomes of vaccination.

Paul Offit, chief of Infectious Diseases at Children's Hospital in Philadelphia in 2008, dismissed concerns that the vaccination schedule was overwhelming children. To Offit, this was just not good science. Other doctors disagreed. In respected medical journals such as *The Journal of the American Medical Association* and *Allergy: European Journal of Allergy and Clinical Immunology*, doctors expressed concern over the long-term effects of early vaccinations. Some doctors state that excessive vaccination is ineffective and dangerous.

But vaccination is a complex subject, and its role in the food-allergy epidemic is difficult to address because of the heated political, social, and economic implications. It is a subject doctors avoid. And so, despite the continuing intense attention given to the peanut allergy in children, an answer to its cause(s) has not yet been found. What has emerged, instead, is a robust economy of doctor fees, nut-free foods, ongoing medical research, and pharmaceutical sales. Peanut and other food allergies have become enormously profitable. It is so much so that one market analyst has suggested that an "autoimmune index" would be a great tool for investors. This index, tagged as "save the children and make money," would monitor the profitability of pharmaceutical stocks relative to the continued rise in peanut allergy and other childhood epidemics.

Peanut allergy began as a mere idiosyncrasy after World War II. Today, its epidemic proportions help fuel a multibillion-dollar food-allergy industry.

Chapter 1 – From idiosyncrasy to multibillion-dollar industry

Executive Summary:

The chapter traces the rise of peanut allergy from an obscure and poorly understood condition in the mid-20th century to a multibillion-dollar industry by the 21st century. Initially, peanut allergies were often dismissed or trivialized, but a small outbreak of anaphylaxis to cottonseed oil in the late 1930s drew attention. Subsequently, peanut allergy emerged and gradually grew in prevalence, particularly affecting children.

Media coverage of tragic cases, public outcry, and the need for proper food labelling increased awareness. The medical community focused on post-sensitization treatments, leading to the development of products like EpiPens.

However, the root cause of the rapid rise in peanut allergy, particularly among children, remained unknown, and the focus shifted to managing the allergy through various means, including allergen-free products, research, and guidelines.

Key takeaways:

1. Peanut allergy cases in the US increased gradually but more significantly in the late 1980s and early 1990s, primarily affecting children.
2. Schools and governments responded with policies, protocols, and allergy-free zones to protect allergic children.
3. Media coverage and public awareness led to increased funding for research, allergy centers, and the development of allergen-free products.
4. Pharmaceutical companies capitalized on the growing allergy market, selling EpiPens and exploring various treatments, such as oral immunotherapy and vaccines.
5. Legal systems faced challenges, with lawsuits related to allergic reactions and accommodations for allergic individuals becoming necessary.
6. Allergy bullying became a concern, leading to felony cases against offenders.
7. Some doctors disagreed on the severity of the peanut allergy problem, but the industry continued to grow.
8. The exact cause of the rapid increase in peanut allergy, particularly in children, remains unknown, despite numerous research efforts.
9. The allergy industry encompasses organizations, conferences, research, and lobbying groups, involving significant funding and pharmaceutical contributions.
10. Guidelines and registries helped track allergens and reactions, but some challenges arose with labelling practices and pharmaceutical ingredients.
11. Despite the industry's growth, the chapter concludes by emphasizing the unresolved issue of what causes children to become sensitized to peanuts and the need for continued research.

Excerpts:

1. "Thirty-year-old Dr. Walter Teller disembarked from the Holland American liner Maasdam at New York City in December 1954... The men went to dinner in midtown. Five hours later, Dr. Teller was 'nearly strangled' when his esophagus closed. He had eaten peanuts for the first time."
2. "Until the last decade of the twentieth century, the US press typically met the rare and curious reactions to peanut with surprise and a shrug of the shoulders... Intense scrutiny in medical literature of this outbreak peaked during the late 1940s and sharply declined during the 1950s."
3. "Peanut allergy tipped into critical mass in the early 1990s when a 'sudden surge of severely allergic children entering school systems … caught many educators off guard.' A 2000 article published in a magazine for Canadian teachers recounted the surprising phenomenon of the unexpected flood of four- and five-year-old food-allergic children."

Chapter 2 – Risk Factors

Executive summary:

This chapter delves into the significant increase in peanut allergies among children in Western countries during the 1990s and early 2000s. Various risk factors, such as atopy, maternal age, cesarean birth, and heredity, have been identified but none fully account for this sudden surge. The allergy, initially believed to be primarily located in certain Western countries, later appeared in unexpected regions like Hong Kong and Singapore, challenging the geographic risk factor. The chapter explores various potential causes, including peanut consumption and preparation methods, although evidence has been inconsistent.

Researchers have also considered the "oral allergy syndrome" or pollen-food allergy, where inhaled birch pollen can trigger peanut reactions, as well as dysregulation of the Th1/Th2 immune response paradigm. Other factors such as delivery method, maternal age at delivery, socioeconomic status, and immune system overload have also been studied but offer only partial understanding of the situation.

Moreover, this chapter discusses the possible role of vaccination, ear infections and antibiotic use, genetically modified foods, and the nocebo effect in the surge of peanut allergies. It evaluates concerns about thimerosal, a mercury preservative in vaccines, and the rise in the number of vaccines. It also looks into the influence of antibiotics on gut flora, the potential role of genetically modified foods and herbicides like glyphosate in disrupting the gut microbiome, and the potential contribution of the nocebo effect, where negative beliefs lead to negative physical symptoms. However, no single factor has been identified as the definitive cause for the rise in peanut allergies.

Key takeaways:

1. Vaccinations, particularly pertussis and polio vaccines, were linked to an increased risk of atopy and allergic illnesses in some studies.
2. The LEAP study suggested that early introduction and regular consumption of peanuts in high-risk children could decrease the risk of peanut allergy.
3. The sudden development of anaphylaxis only in children and specific Western countries pointed to a more specific and powerful determinant of peanut allergy.
4. While peanut consumption may play a role in peanut allergies, it does not fully explain the epidemic's scale and specificity.
5. Swedish researchers propose that cross-sensitization to inhaled birch pollen may cause peanut allergies through "oral allergy syndrome" or pollen-food allergy.
6. The age of onset for peanut allergies in children has decreased over time, with cases being detected at younger ages.
7. Children born during specific months, such as January to March or October to December, have a higher risk of experiencing their first reactions to peanuts during the same months.
8. Peanut allergies exhibit a gender disparity, being more prevalent in boys than girls, echoing a similar trend observed in autism.
9. Children born via cesarean section may have an increased risk of developing allergies due to delayed intestinal flora colonization and its impact on the immune system.
10. Maternal age at delivery has been examined, but the relationship between older maternal age and peanut allergies remains inconclusive.

11. The concept of a genetic "allergy gene" is debated, with some studies suggesting heredity plays a role in peanut allergies, but genetics alone cannot explain the epidemic.
12. Immune system overload resulting from poor diet, fungal overgrowth, antibiotic use, and vaccinations has been proposed as contributing to allergies, including peanut allergies.
13. The sudden prevalence of peanut allergies since the 1990s remains inadequately explained by the identified risk factors, indicating that other factors may be at play.
14. Concerns have been raised about thimerosal in vaccines as a possible factor in the rise of autism, ADHD, asthma, and allergies in children, but definitive studies of vaccinated and nonvaccinated populations have been lacking.
15. The prevalence of peanut allergies increased significantly in areas with higher vaccination rates and decreased in regions with lower vaccination rates, suggesting a potential link between vaccination and peanut allergies.
16. The use of antibiotics in early childhood has been associated with an increased risk of food allergies, potentially due to their impact on gut flora and subsequent disruption of the immune system.
17. Genetically modified foods and the herbicide glyphosate have been questioned for their impact on gut health, but their correlation with the sudden rise of peanut allergies remains uncertain.
18. The nocebo effect, wherein negative beliefs lead to negative physical symptoms, might play a role in sensitization to allergens and allergic reactions.
19. Some children may "outgrow" peanut allergies, but the reasons for this resolution are not fully understood, and the allergy's recurrence in some cases raises questions about its permanence.

Excerpts:

1. "In successive waves through the 1990s, hundreds of thousands of peanut-allergic children arrived for kindergarten in Western countries, catching educators off guard and prompting sudden changes in social behavior, shopping, and eating habits."
2. "In Sweden, peanut consumption is low but the allergy to peanut is high. This being difficult to explain, researchers suggested that cross-sensitization to inhaled birch pollen was causing peanut allergy—this is known as ‘oral allergy syndrome’ or pollen-food allergy."
3. "Doctors have religiously cited a general malfunction of the immune system as a risk factor for peanut allergy in children. The Th1/Th2 paradigm neatly organized the immune system by splitting it into two sides with two distinct thymus (T) white blood cell responses to pathogens and allergens."
4. "As the numbers of allergic children climbed, the age of onset dropped. A review of pediatric peanut-allergic patients at Johns Hopkins University indicated a median age of peanut exposure and reaction were twenty-two and twenty-four months respectively for children born between 1995 and 1997."
5. "For scientists, it was difficult to confirm the role of vaccination because there were no studies of nonvaccinated populations in the United States—there were so few children who had not been vaccinated."

Chapter 3 - Theories

Executive Summary:

The chapter investigates several hypotheses attempting to explain the rise of peanut allergies, primarily in children in Western countries. Some of these theories include the broken-skin hypothesis, ingestion hypothesis, helminth hypothesis, hygiene hypothesis, and the expanded hygiene hypothesis. Each of these theories operates on the assumption that allergies are a genetically determined disorder. In contrast, the toxin hypothesis suggests that allergies, such as peanut allergies, are an evolved immune defense against acute toxicity. While each of these hypotheses provides some insight, they all have limitations in fully explaining the peanut allergy epidemic.

The chapter particularly focuses on two prominent theories: the "Helminth Hypothesis" and the "Hygiene Hypothesis". The Helminth Hypothesis proposes that parasitic worms (helminths), which used to be common in human populations, offered protection against immune-mediated diseases and allergies. With the advent of modern hygiene practices that eliminated these worms, allergic reactions have become more prevalent. Conversely, the Hygiene Hypothesis posits that reduced exposure to infections, particularly in early childhood due to smaller family sizes and improved sanitation, has resulted in an imbalanced immune system that is prone to allergic responses. Despite these theories, the cause of the rise in peanut allergies remains unclear.

Key Takeaways:

1. Existing hypotheses generally consider allergy as a genetically determined disorder.
2. The toxin hypothesis challenges the prevailing view, suggesting allergies function as a defense against acute toxicity.
3. Peanut proteins, particularly Ara h 1 and Ara h 2, have high allergenic epitopes and are resistant to digestion.
4. Theories based on ingestion alone cannot fully account for the rapid rise of peanut allergies in children.
5. Refined peanut oil used in skin creams and oral medications is a potential sensitization route.
6. Skin prick testing and the Th1/Th2 paradigm are also considered in the context of peanut allergies.
7. Profet's toxin hypothesis, introduced in 1991, proposes allergies as a last line of defense against toxic substances.
8. Helminth Hypothesis: Certain parasitic worms used to protect humans from immune-mediated diseases and allergies, but their absence in modern society may lead to an increase in allergies. The Helminth Hypothesis does not fully explain the complexities of allergy, and other factors may play a role.
9. Hygiene Hypothesis: Reduced exposure to infections, particularly in childhood, due to improved sanitation and smaller family sizes may lead to an imbalanced immune system that favors allergic responses.
10. Antibiotic use and vaccination are factors that can influence the immune system and may contribute to the rise in allergies.
11. Clostridia and bacteroides fragilis are gut bacteria that may play a role in reducing allergic sensitization.

12. Antibiotic use in early childhood may increase the risk of developing food allergies.
13. Vaccination may be a factor in allergy development, but it remains a controversial topic in research.

Excerpts:

1. "By 2000, doctors had matched general risk factors with clinical observations to produce several explanations for the general rise in allergy. Each disparate theory, however, was a bad fit for the peanut allergy. None could adequately explain its sudden and increased prevalence initially only in children living in certain Western countries."
2. "The toxin hypothesis, developed before the peanut-allergy epidemic emerged, provided a lone counterpoint to the official hypotheses. This hypothesis was alone in its proposal that allergy is not a dysfunction at all. Within this new concept, allergy serves a purpose as an evolved immune defense against acute toxicity."
3. "First proposed in 1991 by biologist Margie Profet, the toxin hypothesis provided an alternative framework for understanding allergy. It offered, for the first time, a purpose for this disturbing immune response."

Chapter 4 – Rediscovering Anaphylaxis

Executive summary:

This chapter investigates the historical origins of anaphylaxis, allergies, and vaccinations. It discusses how the introduction of hypodermic syringes for mass vaccinations in the late 19th century coincided with a significant rise in severe allergic reactions, particularly among children. Vaccination, while crucial in combating infectious diseases such as smallpox, also introduced new risks with adverse reactions. Despite these risks, vaccination was widely adopted given the severity of the diseases it prevented. This era marked a time of fast-paced scientific and technological progress.

The chapter further discusses the development of vaccines and pharmaceuticals, particularly after the US Civil War. The invention of the hypodermic needle made administering vaccines and medications more straightforward, leading to an increase in pharmaceutical companies. Vaccines, especially the smallpox vaccine, had a significant role in the war's outcome. Yet, medicine shortages during the war spurred creative drug innovation and the rise of pharmaceutical businesses, which would eventually morph into today's modern pharmaceutical companies. The chapter then explores the link between vaccination, immunity, and allergy, underscoring the unintended repercussions of mass injections via the hypodermic needle.

The discovery of food anaphylaxis by French immunologist Charles Richet in 1901 is also covered. Richet found that an initial exposure to certain proteins could sensitize animals, causing them to have violent and sometimes fatal reactions upon subsequent exposure. This response depended on the time between injections. His work suggested that food anaphylaxis was the body's defensive reaction to proteins that entered the bloodstream directly, bypassing the digestive system. However, the medical community later focused on the incomplete ingestion hypothesis, which attributed food anaphylaxis primarily to poor digestion. By the mid-20th century, a food anaphylaxis outbreak linked to cottonseed oil in vaccines and processed foods led to its decreased usage and the subsequent adoption of peanut oil as a replacement.

Key takeaways:

1. Anaphylaxis, allergies, and serum sickness became prevalent with the mass application of vaccinations using hypodermic syringes in the late 19th century.
2. The emergence of germ theory revolutionized medicine and contributed to the understanding of microorganisms causing diseases.
3. Vaccine development in the 19th and 20th centuries included various ingredients to enhance effectiveness and shelf life, leading to new challenges and safety concerns.
4. Commercial vaccine makers fiercely guarded their proprietary formulae to protect shareholders and maintain economic frameworks supporting mass vaccination.
5. The invention of the hypodermic needle facilitated the administration of vaccines and pharmaceuticals, leading to the growth of pharmaceutical companies.
6. Vaccines played a critical role in the outcome of the US Civil War, with smallpox being a significant factor in deciding the war's result.
7. Shortages of medicines during the war led to creative drug innovation and the rise of pharmaceutical businesses.
8. The hypodermic needle allowed for convenient, cost-effective, and relatively sanitary mass administration of vaccines.

9. Mass administration of injected sera caused serum sickness, leading to the study of altered reactivity, later termed "allergy."
10. Austrian pediatricians Von Pirquet and Schick studied serum sickness, laying the groundwork for the concept of allergy.
11. The convenience of the hypodermic needle with vaccines led to unintended allergic reactions and chronic degenerative diseases.
12. The British government switched to calf lymph suspended in glycerin for vaccination to appease public concerns.
13. The hypodermic syringe and mass injections contributed to the first man-made allergic phenomenon in children, known as serum sickness.
14. French immunologist Charles Richet discovered food anaphylaxis during 1901 vaccination experiments on dogs.
15. Initial exposure to certain proteins made animals hypersensitive, leading to violent reactions upon subsequent exposure.
16. Food anaphylaxis occurs when proteins bypass the digestive system and enter the bloodstream directly.
17. The medical community later focused on the incomplete ingestion hypothesis, attributing food anaphylaxis to poor digestion.
18. Cottonseed oil was widely used in vaccines and processed foods, leading to an outbreak of food anaphylaxis in the mid-20th century.
19. The decline in cottonseed oil usage led to the adoption of peanut oil as a replacement in vaccines and drugs.

Excerpts:

1. "The illness characterized by hives, inflammation, vomiting, shock, and drop in blood pressure was a side effect of a technology and procedure never before used together in mass application—vaccination with the hypodermic syringe."
2. "Research and development in vaccines was aided by the invention of a new device making their administration easier and more effective, the hypodermic needle. At the same time, laying the framework for the production, distribution, and sale of both vaccine and needle were the business-minded makers of patent medicines and pharmaceuticals."
3. "Gradually, the hypodermic needle began to replace the unsteady and messy transdermal tools that were used to puncture or scratch the skin. There were few obstacles to the widespread use of the needle for vaccination. The cost was reasonable at $2.50 per device in 1897 (about $67 in 2008). Improvements on the design were required—the glass barrels tended to crack, tips leaked, and needles easily snapped."
4. "Richet expected that the first exposure to the poison would have created a certain amount of immunity in the dogs. Instead, the initial exposure made the dogs hypersensitive."
5. "Alimentary or food anaphylaxis, Richet had discovered through experiments, was the body’s defensive response to proteins that had bypassed the modifying process of the digestive system and been introduced directly to the bloodstream."

Chapter 5 – The History of Peanut Allergy

Executive summary:

This chapter delves into the historical use of peanut oil in penicillin production during World War II and its possible link to the rise of peanut allergies. Dr. Monroe J. Romansky innovatively used peanut oil in the penicillin formula, extending its duration and allowing for single daily injections. The wide use of penicillin and its derivatives, which contained peanut oil, potentially led to side effects such as the development of peanut allergies in some patients. While it's unclear how many individuals developed peanut allergies during this period, it's suggested that peanut allergy began to surface as a growing issue for children.

The chapter also discusses the use of vaccine adjuvants, specifically Adjuvant 65-4, which contained peanut oil and was used in flu vaccines in the late 1960s. Adjuvants are additives that enhance the immune response to vaccine antigens, improving their effectiveness. Despite the benefits of Adjuvant 65-4, its use raised concerns about inducing peanut allergies. A notable surge in peanut allergies among preschool children in the early 1990s coincided with increased vaccination efforts.

Additionally, the chapter discusses the Canadian vaccine PENTA, which combined five vaccines into one needle. PENTA was administered to Canadian children in 1994 without official approval or proper safety studies, which led to reports of adverse reactions. The chapter also mentions the introduction of a vitamin K1 prophylactic injection for newborns, which contained potentially allergenic ingredients. During this same period, there was an increase in peanut and food allergies among children, leading to a concerning rise in severe food allergies.

Key takeaways:

1. Penicillin, a crucial antibiotic, faced challenges in mass production and short-term effectiveness during World War II. Dr. Monroe J. Romansky's innovation of using peanut oil in the penicillin formula extended its duration, leading to single daily injections. The widespread use of penicillin and its derivatives containing peanut oil may have contributed to the rise of peanut allergies.
2. Peanut oil was chosen for penicillin due to its availability, stability in heat, relative affordability, and patriotic connotations.
3. The postwar years saw a growing awareness of peanut allergy in medical circles, but it did not significantly impact public consumption or industry revenues.
4. The peanut industry experienced a decline in the 1990s due to the rise of peanut allergies and a decrease in the snack foods market share.
5. Penicillin administration in the mid-20th century likely contributed to the development of peanut allergies in an estimated 2.5% of children injected with penicillin.
6. The exact number of peanut-allergic individuals from the postwar years remains unknown, but it may have cut across all demographics.
7. Adjuvant 65-4, containing peanut oil, was introduced as a vaccine additive in the late 1960s, promising to improve vaccine efficacy and reduce production costs.
8. The use of adjuvants like Adjuvant 65-4 was associated with allergic reactions, including peanut allergies, due to the presence of peanut proteins in the oil.
9. In the 1990s, efforts to increase vaccination coverage rates among preschool children were intensified, leading to the sudden surge of peanut allergies among four- and five-year-olds in Canada, the United Kingdom, the United States, and Australia.

10. The introduction of Hib conjugate vaccines in the late 1980s and early 1990s contributed to the rise of peanut allergies, as they were administered alongside other vaccines and could potentially depress the immune system.
11. The sudden increase in peanut allergies among children correlated with extensive vaccination campaigns and changes in vaccination schedules during the 1990s.
12. Different countries adopted vaccination schedules recommended by the World Health Organization, incorporating vaccines like Hib conjugates into their pediatric vaccination programs.
13. The use of adjuvants in vaccines has been a topic of concern due to the delicate balance between vaccine potency and safety, particularly regarding allergic reactions.
14. Combination vaccines offer advantages such as reduced injections, improved timeliness, and lower shipping costs, but concerns about increased adverse events exist.
15. Vitamin K1 prophylactic injections for newborns contained potentially allergenic ingredients, and the injected vitamin K remained in the body, overlapping with vaccination schedules.

Excerpts:

1. "Penicillin POB actually doubled penicillin blood levels according to research at the Montreal General Hospital in 1947. But this success was not without side effects. The amount of beeswax and oil used was reduced 'in an attempt to eliminate undesirable reactions.' And in 1950, a study of penicillin treatments in over one hundred children at the Philadelphia Children's Hospital reported additional obstacles to the formula. The Romansky formula had created peanut allergy in an undisclosed number of children."
2. "Adjuvant 65-4, containing up to 65% peanut oil, was introduced as a vaccine additive in the late 1960s, showing promise in creating safer and more effective vaccines. However, the peanut oil in the adjuvant also had the potential to induce peanut allergies, leading to a growing concern about its safety."
3. "The sudden surge of peanut-allergic four- and five-year-old children in the early 1990s caught educators and health authorities off guard, coinciding with intensified vaccination efforts among preschool children in several Western countries."
4. "The introduction of Hib conjugate vaccines in the late 1980s and early 1990s contributed to the rise of peanut allergies, as they were administered alongside other vaccines and could potentially depress the immune system."
5. "In 1994, five vaccines were packaged into a single needle. The first was a Canadian-made vaccine called PENTA (DPT-Polio-Hib PRP-T) by Connaught [...] This product had neither license nor NOC nor product monograph when it was administered to Canadian children starting in 1994."
6. "Immediately, reports of adverse reactions to the combination began to pour into Health Canada. In less than three years, there were more than eleven thousand 'adverse events following immunization' (AEFI) reports [...] These side effects included meningoencephalomyelitis (brain inflammation), convulsions, anorexia, infections, anaphylaxis, inconsolable screaming, and death according to Health Canada records."

Chapter 6 – Absorbing the Costs

Executive summary:

The chapter discusses the potential link between childhood vaccinations and the rise of allergies, particularly peanut allergies. Vaccines contain various ingredients, including adjuvants, stabilizers, and preservatives, which can stimulate the immune system and increase efficacy. However, they may also lead to adverse effects like allergies. The chapter explores the concern that the homology of proteins in vaccines, particularly the Hib vaccine, and peanuts could cause cross-reactivity and sensitization to allergens. Furthermore, it raises questions about the potential role of toxins in vaccines, which might promote allergies to both environmental and food proteins. The lack of transparency in disclosing vaccine ingredients and the potential risks they pose is also discussed.

The chapter explores the emergence of peanut allergies in children and its potential link to changes in pediatric vaccination schedules and the use of certain pharmaceutical ingredients. It highlights the correlation between the rise of peanut allergy and the increase in childhood vaccinations, especially in countries like the United States, the United Kingdom, Canada, Australia, and others. The presence of common ingredients in vaccines, such as castor seed oil, soybean, and other legume proteins, may be responsible for cross-sensitization and subsequent peanut allergies. Additionally discussed, is the impact of toxic adjuvants and compromised detoxification processes in some children, leading to adverse reactions to vaccinations and an increase in allergies.

Key takeaways:

1. Adjuvants can cause side effects, including the production of IgE antibodies linked to allergies.
2. Some vaccines contained gelatin and other allergenic substances, leading to a rise in allergies in children.
3. The homology between proteins in the Hib vaccine and peanuts raised concerns about cross-reactivity and sensitization to allergens.
4. Vaccines, especially when combined, might lead to overstimulation of the immune system and an increased risk of allergies.
5. Toxicity and allergenicity were sometimes considered an acceptable compromise in vaccine adjuvants.
6. Countries that introduced the Hib vaccine in combination with DPT saw an increase in peanut allergies.
7. Lack of transparency in vaccine ingredients and potential risks posed a challenge for understanding and managing allergies.
8. Studies indicate that vaccines can induce hypersensitivity and long-term sensitization to foods and antigens.
9. The rise of peanut allergies in children appears to be linked to changes in pediatric vaccination schedules and increased vaccination rates in several Western countries.
10. Vaccines contain ingredients such as castor seed oil and soybean proteins, which may cross-sensitize children to peanuts and other legume proteins.
11. The use of toxic adjuvants in vaccines, coupled with impaired detoxification processes, may contribute to allergic sensitization.

12. The presence of aluminum in some vaccines is known to stimulate IgE antibodies and may increase the risk of allergies.
13. The introduction of prophylactic injections with vitamin K1, which contains legume proteins, may also play a role in peanut allergies.

Excerpts:

1. "Only a handful of doctors through the late 1990s looked directly at pediatric injections and asked whether a reduction of common childhood diseases through a policy of mass vaccination (and other injections) was worth the price of a higher prevalence of allergy and other adverse outcomes."
2. "While manufacturers, government, and doctors are not obliged to reveal the precise ingredients of vaccines, the CDC offered a limited list."
3. "The question about vaccination has never been whether there would be damage but rather how much and what kind in relation to the established vaccination goals."
4. "In a 2001 study, none of the 456 Tasmanian children aged seven to eight years reacted to a peanut skin prick test. By 2009, one in ninety children or 1.11% was allergic to peanuts. Changes in the vaccination schedule and the increased rate of children vaccinated in Tasmania correlated to this development."
5. "Most peanut-allergic patients also have IgE antibodies against other legume proteins, including soybean and also oil seed proteins such as castor. However, 'fewer than 15% of such patients react to other members of the legume family.'"
6. "Allergy is designed to defend against toxins that escape general detoxification. The ability of peanut-allergic children to eliminate toxins, including those from the pediatric injections, would have been challenged at the time they were administered and afterwards."

Chapter 7 - Rationalizations

Executive summary:

The chapter delves into the rationalizations used by medical professionals and authorities to deflect the growing concerns regarding the rapid rise of peanut allergies in children, especially in Western countries. It highlights the potential link between the expanded pediatric vaccination schedule and the surge in peanut allergies. The chapter discusses the economic and political motivations behind dismissing the vaccine-allergy connection, as well as the efforts to protect pharmaceutical companies from liability. It also addresses the shifting public perception of vaccination risks, the role of parents in perpetuating mass injections, and the concept of the "crossover point" where vaccine complications outweigh the benefits of disease prevention.

The chapter also discusses the concept of herd immunity, routine vaccinations, and the complexities surrounding vaccine safety and efficacy. It highlights how the state's control over citizens' health decisions, particularly routine and mass vaccination policies, raises concerns about individual rights and the potential risks associated with vaccines. The mathematical model of herd immunity, aiming to reduce disease transmission through vaccination, may not accurately reflect real-world scenarios due to numerous variables and assumptions. The chapter sheds light on adverse reactions and allergies associated with certain vaccines, emphasizing the need for informed vaccination choices and recognition of individual differences in susceptibility.

Key takeaways:

1. The rise in allergic conditions coincided with the intensified and expanded vaccination schedule starting in the late 1980s.
2. Medical professionals and authorities rationalized away the vaccine-allergy connection, deflecting concerns and protecting pharmaceutical interests.
3. The Vaccine Injury Compensation Program guidelines in the United States made it challenging to establish a causal link between vaccinations and later-onset allergies like peanut allergy.
4. The pharmaceutical industry's profits and the rise in food allergy have contributed to a booming free-from food market.
5. Parents' trust in mass injections and vaccination traditions has contributed to the current epidemic of allergies and other chronic conditions in children.
6. Pharmaceutical companies and governments have prioritized economic interests and vaccination promotion over addressing the allergy epidemic.
7. The public's changing tolerance towards vaccine risks has sparked increased activism and questioning of vaccination practices.
8. The chapter explores the complex web of factors that shape public perception, medical practices, and the role of parents in vaccine-related issues.
9. Routine and mass vaccination policies involve citizens relinquishing control over their health decisions to the state for the promise of herd immunity.
10. Allergies and contraindications to vaccines exist, which are important factors to consider for vaccination safety and individualized health choices.
11. Recent measles outbreaks in highly vaccinated populations question the efficacy of current vaccines and the concept of herd immunity.

12. Parents and individuals are becoming more aware of vaccination concerns and questioning the high coverage rates needed for herd immunity.
13. The US has introduced state bills to limit vaccination exemptions based on personal belief or religion and to increase adult vaccination rates.
14. The PENTA vaccine, a combination of five vaccines, was removed from use due to significant side effects.
15. Corporate interests and lack of accountability contribute to the persistence of vaccine injury issues.
16. Vaccination and allergy/anaphylaxis have a causative relationship, and the potency of vaccines may increase the risk of allergies.
17. Legal recourse for vaccine injury is limited in Canada, and there is still no acknowledgment of the inherent risks of vaccines.
18. Immunologists and allergists understand the relationship between immunity and allergy and the potential risks associated with vaccines.

Excerpts:

1. "And yet, medical literature illustrated that the only means by which immediate and mass allergy had ever been created was by injection. With the pairing of the hypodermic needle and vaccine at the close of the nineteenth century, mass anaphylaxis made its explosive entry into the Western world."
2. "Doctors knew that as the number and potency of vaccines increased, so, too, would the risk of side effects that included soaring IgE and atopy. Anaphylaxis immediately following vaccination had finally become an 'obstacle' to the routine jab, doctors observed."
3. "The costs of vaccine damage such as allergy were not built into the government model of disease management because they were absorbed by those affected—children and their parents. These people, in turn, have spawned a new source of revenue for business. The rise in food allergy and intolerance has contributed to an enormous free-from food market."
4. "Laws and policies related to routine and mass vaccination highlight the extent to which the state controls the bodies of its citizens."
5. "Allergies used to be straightforward contraindications for immunization through the 1960s... Eczema is still a contraindication for smallpox vaccination—eczema vaccinatum is potentially fatal. Asthma, rhinitis, eczema, food, or environmental allergies used to be red flags."

Chapter 8 - The Business of Breathlessness and Oozing Skin

Executive summary:

The chapter traces the historical development of Western medicine's understanding and treatment of allergies and related conditions, focusing on asthma, eczema, and urticaria. It highlights the shift from early vague and ineffective treatments to the modern medicalization of allergies, which led to the discovery of key substances like adrenaline and histamine and the development of antihistamines and steroids. The rise of pharmaceutical companies and the patenting of medicines further shaped the landscape of allergy treatment. The chapter also touches upon famous historical figures who suffered from allergies, such as revolutionary Che Guevara. Overall, it explores the transformation of allergies from mysterious and poorly understood conditions to medical challenges with targeted remedies.

The chapter explores the history and challenges of managing allergies, particularly anaphylaxis, through the 20th and early 21st centuries. It discusses the development of desensitization treatments, immunotherapy, and emergency care for allergic reactions. The author delves into the rising prevalence of allergies, particularly in children, and highlights the potential connection between vaccination and the allergy epidemic. Additionally, the chapter examines various approaches to allergy treatment, including traditional Chinese medicine and immunotherapy advancements. It concludes with a reflection on the limitations of the medical approach to allergies and the need to explore the role of consciousness and epigenetics in addressing allergic reactions effectively.

Key takeaways:

1. Early medicine lacked effective treatments for allergies, which were considered vague and poorly understood conditions.
2. Western medicine's focus on classifying and reclassifying diseases led to the medicalization of allergies and the pursuit of remedies to conquer symptoms.
3. Allergies like asthma and eczema were treated with various nostrums, some of which had harmful effects on patients.
4. The discovery of adrenaline and histamine paved the way for antihistamines and steroids, revolutionizing allergy treatment.
5. The pharmaceutical industry played a significant role in shaping the landscape of allergy treatment through patenting and marketing medicines.
6. Famous historical figures, like Che Guevara, suffered from asthma and utilized inhalers and injections for relief.
7. Asthma deaths in the 1960s were linked to high concentrations of inhaled asthma drugs, highlighting the importance of appropriate dosing and safety measures.
8. Allergist Robert Cooke's experiments in the early 20th century led to the development of desensitization treatments for allergies, particularly pollen-related conditions.
9. Emergency care, originating from battlefield medicine, became crucial for managing anaphylactic reactions.
10. Allergy prevalence surged throughout the 20th century, becoming a significant health concern, with billions of dollars spent annually on medication and EpiPens.
11. The EpiPen, initially developed for military use, gained widespread usage but faced controversies due to its high prices.

12. Western medicine's focus on symptom management rather than finding a cure raised concerns about long-term patient dependence on drugs.
13. The rise of allergies in children was linked to vaccination, but medical authorities were hesitant to acknowledge this connection.
14. Immunotherapy, such as Xolair and oral immunotherapy, aimed to target IgE, but these treatments had limitations and adverse effects.
15. The war on IgE missed considering its potential role in cancer prevention.
16. Traditional Chinese medicine and herbal therapies showed potential in treating allergies, but challenges in scalability and compliance remained.
17. Epigenetics played a crucial role in the development of allergies, indicating the need to consider environmental influences on genetic expression.
18. The Cartesian mechanist view of the body limited the medical approach to allergies, leaving out consciousness and the mind-body connection.
19. Vaccination and medical practices contributed to an iatrogenic epidemic of allergies, raising concerns about the medical system's limitations.
20. Parents and caregivers are becoming more aware of the potential risks of vaccinations, leading to a growing movement against mandatory vaccination.

Excerpts:

1. "Western medicine is rooted in a division of mind and body—for which we might credit philosopher René Descartes (1596–1650) who likened the sick body to a malfunctioning clock: fix it or take out the broken parts and things will go back to normal, maybe. This clock-body metaphor became an ongoing invitation to classify, then re-classify the body and its diseases large and small—the progress of which was marked by materia medica, treatises on diseases and their remedies."
2. "The nosology of what would be known as allergy and anaphylaxis after 1901 in western medicine was first described by where and how symptoms appeared in the lungs or skin. The word asthma, it is said, came from the Greek asthmati used in Homer’s poetic saga The Iliad (c. 800BC)."
3. "At the start of the twentieth century, new understandings about the root causes of asthma, eczema, and urticaria required entirely new labels: anaphylaxis and allergy. The twin relationship of allergy and immunity had emerged from the first use of the hypodermic syringe in mass vaccination."
4. "Cooke and colleagues tried to reverse the condition through the same means in 'anti-anaphylaxis' or 'desensitization' experiments."
5. "Emergency care workers rely on drugs like Benadryl, nebulizers, and epinephrine to control anaphylactic reactions, but long-term management remains a challenge."
6. "The primary cause of the allergy epidemic in infants and children was well known—it was and is an externalized cost of vaccination."

APPENDIX

The following figures reveal an upward trend of peanut allergy in children living in affected countries.

AFRICA

• In 2013, reactions to peanut in 1.5% and sensitization in 17.5% of Ghanaian schoolchildren.[1] In 2006, Gideon Lack noted in a speech mentioned in the British Medical Journal the 0% incidence of peanut allergy in Sub-Saharan Africa suggesting the reason being the early consumption of peanuts at weaning and beyond. Either he was mistaken or the phenomenon of peanut allergy grew rapidly.[2] There was serologic evidence of sensitivity in 5% of Xhosan children in Cape Town (2007),[3] but with no reported cases of anaphylaxis. By 2011 another study of Xhosan children in an urban high school showed 1.9% sensitized and reacting to peanut.[4]

AUSTRALIAN CAPITAL TERRITORY (ACT), AUSTRALIA

• Of school-entrant ACT children, 2% (2009) were confirmed by diagnostic test to be peanut allergic.[5] ACT is a self-governing state with the highest density population and smallest area at 910 square miles. Within it is the national capital, Canberra. According to the Australasian Society of Clinical Immunology and Allergy, 1.15% of ACT children born in 2004 were peanut allergic, compared to 0.47% of those born in 1995.[6] National figures are 0.71% (2001)[7] and 0.5% (1995).[8] Children make up 26% of the Australian population. Therefore, in 2009 of almost 22 million Australians, there were about 5.7 million children, 114,400 of whom were peanut allergic.

MELBOURNE, AUSTRALIA

• 3% of children were found to be allergic just to peanut in Melbourne in a 2011 published study.[9]

TASMANIA, AUSTRALIA

• This Australian island and state's figures are 1.11% in 2009[10] and 0% in 2001.[11] Of a population-based cohort of 456 Tasmanian children aged seven to eight years, none reacted to a peanut skin prick test in 2001. By 2009, 1.11% were reactive to peanut.

MONTREAL, CANADA

• 1.21% to 2.33% of children in a Canadian nation-wide survey in 2009 were found to be peanut allergic.[12] Of children under nine in Montreal, Canada, 1.71% or more (2007) were peanut allergic.[13] In 2009 there were 7.8534 million children in Canada under nineteen. Therefore, as many as 183,000 children were peanut allergic.

NORTHEASTERN EUROPE

• Geographically close countries in northeastern Europe—Estonia, Lithuania, and Russia—appear to have a very low prevalence of peanut allergy[14]

• Data are limited for this area. A study of a Lithuanian birth cohort born between 2006 and 2007 found 2.8% of 12 month olds were sensitized to foods primarily egg and dairy. Peanut allergy was low being found in two from a group of 1553 children.[15]

• A 2014 published study of peanut allergy in children from the Tomsk Region of Russia indicated that just .08% of children were reactive to the food.[16]

• It is worth noting that the health infrastructure of Russia appears to have been weak with allergy and 'weakness' being a contraindication to immunization until about 1996. De-

worming is an ongoing initiative. The Hib B vaccine was introduced by the Russian Ministry of Health only after 2010. http://rostropovich.org.

FRANCE

• Of children under fifteen in France, 0.45% or more (2000) were peanut allergic.[17]

GERMANY

• No firm statistics are available, although peanut allergy seems of limited significance. In a 2005 analysis of physician-reported cases of 103 anaphylactic children in Germany, foods were the most frequent cause of the reaction (57%, and of this number 20% to peanut) followed by insect stings (13%) and immunotherapy injections (12%). Peanuts and tree nuts were the foods most frequently causing the reactions.[18] In a 2004 study of food allergy in Berlin children and teens, there appeared to be no self-reported symptoms brought on by peanut.[19]

HONG KONG

• Of Chinese children aged two to seven living in Hong Kong, 0.57% to 1%[20] were reactive to peanut (2009). A EuroPrevall Prague report indicated that 0.7% of Hong Kong's study participants were peanut allergic (2008). Studies published in 1994[21] and 1999[22] concluded that sensitization to peanut was rare in Chinese children living in Hong Kong. In fact, it seemed rare to find peanut allergy at all in Southeast Asia.[23] Subsequent reports in 2001/2002 reiterated that while the per capita consumption of peanuts in China is similar to that in the United States, peanut allergy was rare in China. By 2008, 0.57 to 1% of Chinese children were reacting to peanut. Chinese American children living in the United States had an incidence of peanut allergy similar to that of the general US population (2001).[24]

INDIA

• Limited data are available on peanut allergy in India although food allergy in general is believed to be increasing. There are no recent studies of peanut allergy.[25]

ISRAEL

• .6% (2012) Jewish Israeli children aged 13-14 allergic to peanut.[26] This is up from .17% (2002).[27]

• In the same country, 2.5% (2012) of Arab Israeli 13-14 year old children were peanut allergic.

JAPAN

• In 2003, population-based prevalence figures for food allergy in Japan were apparently unavailable.[28] The most common food allergen among Japanese children was hen's egg, followed by cow's milk and then wheat. These three food allergens accounted for 60% of pediatric food allergy. Peanut allergy appears generally not to be a concern.

NORWAY AND DENMARK

• In 2005, 0.5% of Danish adolescents reacted to peanuts.[29] In 2001, the numbers of peanut-allergic children in Norway and Denmark were believed to be very low.[30]

SINGAPORE

• Of children aged five to twelve living in Singapore, 1.08%–1.35% were reactive to peanut in 2007.[31] A 1997 study had alluded to reports of peanut allergy from several Asian centers including Singapore, the Philippines, Malaysia, Indonesia, Japan, Beijing, Hong Kong, and Taiwan.[32] Sensitization to peanut was second to shellfish in cohort under five according to a National University of Singapore professor (2005). Significantly, although children were

sensitized to peanut, there were no reported cases of anaphylaxis. The reason for this was not known although lack of exposure to peanuts was not a factor.[33] However, by 2007, a three-year study revealed a "worrying trend" of peanut reactivity in Asian children living in Singapore (Chinese, Malay, Indian, and Eurasian ethnic groups).[34] Peanut allergy was found in 27.3% of food-allergic children.

SWEDEN

• Of children under six in Sweden, 1.2% or more (1998) were reactive to peanut.[35] In 2000, 2.139 million people were under age nineteen. Therefore, 25,668 children were allergic to peanuts in 1998.

UNITED KINGDOM

• A total 1.8% of schoolchildren were allergic to peanuts in 2007.[36] In 2008, there were 11.5 million children under sixteen. Therefore, an estimated 207,000 children in the United Kingdom were allergic to peanuts. Statistics have ranged from 3% to 1% in a study of 70 children (1.43%).[37]

UNITED STATES

• 2% - 2.8% or 1.5 to 2 million peanut allergic children in 2010 was an apparent increase from a 2008 study in which 1.4% children were found to be allergic.[38] 0.8% children under eighteen were reactive to peanut in 2002.[40]