5-2011

Do Maternal Factors During Pregnancy and Breastfeeding Have an Affect on the Development of Food Allergies in Children?

Kimberly J. Zagaros
St. Catherine University

Do Maternal Factors During Pregnancy and Breastfeeding Have an Affect on the Development of Food Allergies in Children?

Kimberly J. Zagaros

St. Catherine University

May 2, 2011

Abstract

Background: Food allergies are a growing concern in the pediatric population. They affect over 3 million children in the US and its incidence has risen 18% from 1997 (Branum and Lukacs, 2008).

Methods: A literature review was conducted in order to find the most recent studies regarding maternal factors during pregnancy and breastfeeding associated with the development of food allergies in children. Eight studies were found and analyzed for their results.

Results: Three studies concluded that there is a link between maternal factors during pregnancy and breastfeeding and the development of food allergies. Two studies concluded that there is insufficient data to make a definitive correlation between maternal factors during pregnancy and breastfeeding and the development of food allergies. Three studies concluded that there is no correlation between maternal factors during pregnancy and breastfeeding and the development of food allergies.

Conclusion: Based on the findings more research needs to be done in order to change current recommendations about a correlation between maternal factors during pregnancy and breastfeeding and the development of food allergies.

Introduction

Food allergies are a major factor in disease prevention and health promotion of pediatric patients. The incidence of food allergies continues to be a growing concern in this population. In 2007, the Centers for Disease Control (www.cdc.gov) reported an estimated three million children under age 18 years (3.9%) had a reported food allergy; an 18% increase from 1997. This drastic increase emphasizes the need for further evaluation of the factors associated with food allergies and how they can be effectively prevented. There are currently three known types of allergy prevention; primary prevention blocking the initial immunologic sensitization (the development of IgE antibodies to an allergen), secondary prevention reducing the further development of the disease following initial sensitization, and tertiary prevention reducing the symptoms following exposure and allergic response (Fleischer, 2011). This report will evaluate the effects of maternal factors during pregnancy and lactation associated with the development of food allergies in infants by critical analysis of existing studies evaluating food allergies and exposures to infants.

Problem

Food allergies are a potentially severe immune reactions to specific foods or food ingredients. Eight foods or categories of food account for 90% of food allergies in children and include milk, eggs, peanuts, tree nuts, fish, shellfish, soy, and wheat. Physical symptoms can range from mild to severe and occasionally resulting in death depending on the severity of the allergy (Branum and Lukacs, 2008). The most common symptoms involve the gastrointestinal, integumentary, and respiratory systems. Gastrointestinal symptoms include nausea, vomiting, abdominal pain, distension, flatulence, and diarrhea. The integumentary symptoms frequently are urticaria, angioedema, and eczema while respiratory symptoms include allergic rhinitis, asthma,

and anaphylaxis (Kimber and Dearman, 2002). Children with allergies to these types of foods are also two-four times more likely to have additional related conditions including asthma and eczema resulting in approximately 300,000 hospital visits per year (Branum and Lukacs, 2008). Currently, the exact cause of food allergies and the increase in children who continue to be affected by these antigens is unknown. It has been identified that early events including nutritional options during pregnancy, lactation, and diet may be an important component in the development and prevention of a food allergy. Thus, no specific guidelines or recommendations for practice have been developed, leaving little help for providers working with patients/mothers and their questions.

Pathophysiology

Maternal Fetal Immunity

The fetal immune system functions on naturally acquired passive immunity. This is the transfer of antibodies from mother to fetus via the placenta or from mother to infant during breastfeeding. Initial immunity is formed in a completely sterile environment therefore lacks the exposure of unwanted antigens (Dalal and Roifman, 2011). The only immunities transferred from the mother's bloodstream to the fetus through the placenta are immunoglobulin G (IgG) antibodies that protect the infant from infection while in utero and for the first 6 months of extrauterine life. These antibodies are progressively replaced by the infant's own antibodies causing the newborn to rely on their own T cells, plus elements of the innate immune system to fight infections, respond to vaccinations, and reject unwanted pathogens after birth. Infants who breastfeed also receive additional antibodies, immunoglobulin A (IgA), through the mother's breast milk assisting them in further immunity (Abrahams, 2011).

Cell-Mediated Immune Response

Tortora and Derrickson (2009) describe a cell-mediated immune response in a cascade of steps. Initially, the response begins with the activation of T cells by an invading antigen. This activation occurs in a 2-step signal process involving surface proteins on T cells and CD4 or CD8 proteins. First, receptors on the surface of the cell known as T-cell receptors (TCRs) recognize and bind to the surface of specific foreign antigen-MHC complexes. When an antigen enters the body only a small number of T cells have TRCs with the ability to recognize and bind to the specific antigen. The TCR and CD4 or CD8 proteins interact with the antigen-major histocompatibility complex (MHC) to create the first signal in the T cell activation process. Secondly, the T cell adheres to the surface of the antigen for a short period of time through co-stimulators, some known as interleukin 2 (IL-2), on the surface of each of these cells initiating the second signal causing the T cell to become active. Once this activation occurs the T cells that display CD4 undergoes a process known as clonal selection. This process is where the cell divides several times and forms highly specialized cells that can recognize the same antigen as the original cell. Clonal selection results in the formation of T helper and T helper memory cells. The activated T helper cells begin secreting the important cytokine IL-2 needed for most immune responses and is the main trigger for T cell proliferation or cell division. Memory helper T cells are not currently active in this initial immune response. However, when the same antigen reenters the body memory helper T cells proliferate and become additional helper and memory helper T cells. Activation of the T cells that contain CD8 develops into cytotoxic T cells (CD8 T cells). These cells recognize foreign antigens containing a MCH class I (MCH-I) on the surface of body cells infected by microbes, some tumor cells, and cells of tissue transplantation. TCRs and CD8 connect with MCH-1 following antigen recognition leading to stimulation and

activation of the cell. Following activation these cells also go through clonal selection resulting in the formation of active cytotoxic and memory cytotoxic T cells. Active cytotoxic T cells attack other body cells that have been infected with the antigen and eliminate the invaders by recognizing, attaching, and killing the specific targeted cells. Memory cytotoxic T cells do not act in the initial exposure to the antigen. These cells respond to the same antigen reentering the body at a future time by quickly proliferating and differentiating into more cytotoxic and memory cytotoxic T cells.

Antibody-Mediated Immune Response

Tortora and Derrickson (2009) illustrate an antibody-mediated immune response as a process involving the reaction of B cells. These cells stay put in the presence of an unwanted antigen and become activated in a lymph node, spleen, or mucosal lymph tissue. Activation process of B cells begins when the antigen binds to the B cell receptors (BCRs) and antigen processing begins. The antigen is taken into the B cell, broken down and combined with MCH class II (MCH-II) self-antigens and then moved into the plasma membrane. The helper T cells recognize the antigens and assists in co-stimulation required for B cell proliferation and differentiation. Following activation the B cell begins clonal selection resulting in the formation of clone B cells and memory B cells. The clone B cells known as plasma cells secrete antibodies and memory B cells proliferate and differentiate into additional plasma cells and memory B cells used for future exposure to the same antigen. Antibodies, also known as immunoglobulins, can be differentiated into five different classes; IgG, IgA, IgM, IgD, and IgE. Although each class has a different structure and specific role in immune responses they all respond by disabling antigens. The actions of antibodies include neutralizing an antigen, immobilizing bacteria, agglutinating and precipitating an antigen, activation a complement, and enhancing phagocytosis

Complement System

The complement system assists in immunity by destroying microbes causing phagocytosis, cytolysis, inflammation, and damage to the cells. Complement proteins act in a cascade effect; one reaction triggering another until the desired outcome has been achieved. Complement proteins are categorized by an uppercase C and numbered 1-9. These proteins remain inactive until triggered by enzymes to split into fragments. Activation occurs in three different ways:

a. The classical pathway begins with an antibodies binding to antigens activating C1 and eventually activating C3 leading to phagocytosis, cytolysis, and inflammation.

b. The alternative pathway is initiated by an interaction between lipid-carbohydrate complexes and the interaction between surface microbes and complement proteins activates C3.

c. The lectin pathway is activates C3 when macrophages that digests microbes release chemicals causing the liver to produce lectin, which binds to the carbohydrates leading to C3 activation. Once C3 is activated it splits into C3a and C3b. C3b enhances phagocytosis and initiates additional reactions leading to cytolysis. C3a and C5 bind to mast cells that assist and enhance the inflammatory process (Tortora and Derrickson, 2009).

Allergic reactions

An allergic reaction occurs when a person has a specific response to a substance causing adverse effects such as flushing, itching, urticaria, nasal discharge, choking, cough, wheeze, nausea, vomiting, cramping, dizziness, tachycardia, or hypotension and tissue injury (Siomons,

2010). Four different types of allergic reactions can occur including antibody-immune responses and cell-mediated immune response.

A Type I anaphylactic reactions are the most common type of allergic reaction and usually occurs within a few minutes after a sensitized person is re-exposed. In response to the first exposure an antibody-mediated response occurs causing no reaction to the antigen, preparing the body for future exposure. IgE antibodies are present during re-exposure and a release of histamine, prostaglandins, leukotrienes, and kinins occurs. The release of these chemicals initiates an inflammatory response leading to vasodilation, increased capillary permeability, contraction of the airways, and increased mucous secretion. Anaphylactic shock, a life threatening response, can cause wheezing, shortness of breath, and shock caused by increased vasodilation and fluid loss from blood. Common triggers of an anaphylactic reaction include food allergies, insect stings or bites, or medications (Tortora and Derrickson, 2009).

Antibodies IgG and IgM directed towards a person's blood cells or tissues cause a Type II cytotoxic reaction. The reaction between antibodies and antibodies triggers the activation of the complement system. Blood transfusion reactions are commonly the cause of cytotoxic reactions (Tortora and Derrickson, 2009).

A Type III immune-complex reaction is an immune-complex reaction involving antibodies IgA or IgM, and complement. In conditions such as glomerulonephritis and rheumatoid arthritis some of the immune complexes escape phagocytosis and cause complement system activation leading to inflammation (Tortora and Derrickson, 2009).

A Type IV cell-mediated or delayed hypersensitivity reaction usually occurs 12-72 hours after exposure to an allergen. The allergen is taken up by antigen-presenting cells that migrate to the lymph nodes and introduces the antigen to the T cells resulting in T cell activation,

proliferation, and differentiation. Bacteria such as *Mycobacterium tuberculosis* or poison ivy toxin can trigger a cell-mediated immune response (Tortora and Derrickson, 2009).

Although all of the above are known physiologic responses to allergies, they are not all associated with maternal factors related to food allergies. The most common type of reaction found in food allergies is a type I anaphylactic reaction (Tortora and Derrickson, 2009).

Food Allergy vs. Food Intolerance

Kimber and Dearman (2002) emphasize the importance of distinguishing between food allergies and food intolerances when evaluating food allergies. Intolerance to food other than a food allergy can be defined as a group of reproducible adverse reactions mediated by non-immunological actions that are aggravated in those who are susceptible to intolerance following the consumption of a particular food or ingredient in a food. Food allergy can be described as adverse reactions and health effects that elicit the induction of a specific immune response. Food allergies are therefore characterized by and dependent on the exposure to one or more food proteins leading to an immune reaction. Allergic sensitization requires the production of the specific IgE antibodies to stimulate an allergic response.

Diagnostic Testing

Diagnosing a food allergy begins with a detailed history in addition to laboratory and/or elimination testing. There are two common laboratory tests used to diagnose a food allergy, yet they may not by definitive (Burns, Dunn, Brady, Starr, and Blosser, 2009). A skin prick test (SPT) is the standard form of testing and is thought to be somewhat sensitive, it is estimated at a 50% accuracy rate (Nolte, 2011). However, the cutaneous response may not be the same as the systemic response (Burns, Dunn, Brady, Starr, and Blosser, 2009). A serum IgE and eosinophilia count can be done if the child is unable to have a SPT. Theses results are used to diagnose an

allergy if the eosinophilia level is greater than 400/mm3. Results need to be carefully interpreted because findings can reflect an exposure to a different allergen. Food elimination and challenge is another way to diagnose a food allergy. After a food has been identified as a potential source of the problem the elimination of that food is used to confirm diagnosis. Typically, foods are completely eliminated from the diet for a minimum of 2 weeks and then very gradually reintroduced. An allergy is confirmed if the child's symptoms reappear after the food has been reintroduced (Burns, Dunn, Brady, Starr, and Blosser, 2009).

Review of Literature- Dietary factors for Women who are Pregnant and/or Lactating

A literature search was conducted using the medical databases PubMed and Medline using the terms 'infant', 'child', 'maternal factors', 'breastfeeding', 'peanut', 'egg', 'cows milk', and 'wheat' all combined with the term 'food allergy' and 'food hypersensitization'. A further Google search was done using the term 'maternal factors associated with food allergies' to obtain additional potentially useful documents and information. These searches were initiated in January 2011 and completed in April 2011. All articles and studies used are in English and were published from 2005-2010.

The unborn fetus is able to make immunologic responses to food or other allergens during pregnancy, whether it is due to a normal immunologic response or it is related to the development of an allergy is currently unknown (Fleischer, 2011). Important factors affecting the development of food allergies include; exposure to the allergen during pregnancy, in breast milk or during infancy, the possible protective effect of breast milk, the immune status of the mother during pregnancy and lactation and the age at which foods are introduced to the infant (Kimber, 2002).

DesRoches, Infante, Paradis, Paradis, and Haddad (2010), Sichere et al (2010), and Sausenthaler et al (2007) all found data to indicate a link between maternal consumption and the development of a peanut allergy. DesRoches, et al. (2010) conducted a case-control study of 403 infants 18 months or younger. Two hundred and two infants were chosen to be in the case group and two hundred and one were chosen to be in the control group. Data was collected using a self-administered questionnaire filled out by the infant's mother and involved evaluation of food consumption during pregnancy and breastfeeding. They were asked specifically which foods were eaten, during what periods, and how often. Food history of the infant was also investigated and the presence or absence of peanut sources in the infant's environment and a list of peanut containing foods. Skin prick testing and total and specific IgE antibody levels were measured. Statistical analysis was used to estimate odds ratios and 95% confidence intervals associated with maternal peanut consumption. A reaction to peanuts occurred at first known exposure in 75% of the case infants. The mean age at which the first reaction to peanuts was observed was 12.1 months. Urticaria (81%) and angioedema (47%) were the two most common manifestations and vomiting and diarrhea were present in 12% of the infants. More severe reactions also occurred such as respiratory reactions (16%), altered consciousness (4%), and hypotension (0.5%). Greater than 56% of the reactions involved at least two body systems. This study indicates that early exposure to peanut proteins in utero or in human breast milk is a potential risk factor for the development of a food allergy. In this study breastfeeding alone did not appear to be a factor, however, consumption habits during both pregnancy and lactation seemed to have had the greatest impact (DesRoches, Infante, Paradis, Paradis, and Haddad, 2010).

Sicherer et al (2010) conducted a study of 512 infants enrolled at 3-15 months of age with enrollment requirements that included; a history of a positive reaction to cow's milk or egg, a

positive skin prick test, and moderate to severe atopic dermatitis were included. Maternal ingestion of peanuts during each trimester and during breastfeeding was then evaluated based on the following criteria: a) avoided, b) <2 times per week, c) >/= 2 times per week, but less than daily, d) daily or e) unknown. Frequent ingestion was termed as 2 times per week or more. Skin prick testing and IgG and IgG4 antibodies were measured. Results of the study indicated maternal ingestion of peanuts during pregnancy had a dose-dependent association with peanut sensitization and likely peanut allergy in infants with an additional egg or milk allergy. Logistic regression was used in this study with a stepwise selection procedure retaining variables that were significant (p<.5) in the resulting model. Of the 512 infants enrolled 503 were further analyzed. Overall, 270 (53.7%) of the 503 infants had a positive skin prick test to peanuts, 305 (60.6%) had a detectable IgE to peanuts (>/= 0.35), and 346 (68.8%) had sensitization detected by at least 1 of the test methods at enrollment. There were 140 (27.8%) children who had detectable IgE peanut levels >/= 5. This clinical study denotes that the emphasis was focused on sensitization to peanuts, not a clinical allergy (Sicherer et al, 2010).

Sausenthaler et. al. (2007) indicated a link between maternal diet during pregnancy and the development of allergic sensitization. The subjects were chosen from the LISA (Influences of Lifestyle-related factors on the Immune System and the Development of allergies in Childhood) database. The sample included 3097 newborns and their mother's. Data was collected using a questionnaire focused on atopy, prenatal education, smoking, and maternal diet during the last 4 weeks of pregnancy shortly following delivery, and blood samples for specific IgE analysis at 2 years of age. Repeated questionnaires were given to mothers at the intervals of 6 months, 12 months, 18 months, and 24 months. Options for answering the questions were no exposure, less than 2 times per week and greater than 4 times per week. Statistical analysis was used to estimate

odds ratios and 95% confidence intervals associated with maternal consumption and fetal exposure. The prevalence of doctor-diagnosed eczema totaled 446 (17.7%) of patients and allergic sensitization against food allergens totaled 200 (9.3%) of patients. Cow's milk totaled 110 (5.1%), eggs 116 (5.4%), and peanuts 37(1.7%) of patients. The results of this study suggest that maternal diet during the last 4 weeks of pregnancy had an effect on the development of allergic disease in their infants. It suggests that the high ingestion of certain foods high in 6 polyunsaturated fatty acids such as margarine, vegetable oils, celery and citrus fruits were possible associated with sensitization at the age of 2 years. While high fish ingestion and foods high in 3 polyunsaturated fatty acids may decrease the risk (Sausenthaler, 2007).

Articles by Heine and Tang (2008) and Greer et al (2008) indicate the need for further evaluation and research. Heine and Tang (2008) evaluated dietary approaches to food allergy prevention by reviewing several previous studies and two recent meta-analyses these indicated that maternal elimination diets during pregnancy and lactation generally are not considered to be effective and may cause nutritional deficits to the mother and fetus. The analysis of their data signifies doubtful effectiveness of dietary avoidance during pregnancy and similar findings during breastfeeding. This review evaluated multiple dietary approaches to the prevention of food allergies with maternal exposure being only one component. They concluded that further research is needed to clarify the effectiveness of dietary interventions both maternally and nutritionally for the infant (Heine and Tang, 2008).

Greer et al (2008) indicated a need for further evaluation of the effectiveness of dietary intervention in the prevention of food allergies. It also evaluated the effectiveness of nutritional interventions on the development of atopy with the role of maternal diet being only one factor. According the American Academy of Pediatrics (AAP) (2008), there is a lack of evidence that

supports maternal dietary restrictions during pregnancy or lactation playing a significant role in the prevention of atopy and additional studies are need to conclude an association between these factors (Pediatrics, 2008).

Studies by Venter (2009), Host and Halken (2005), and Kramer and Kakuma (2009) indicate no link between maternal dietary modification and food allergies in infants or children. Venter (2009) assesses factors associated with maternal dietary intake and the development of food sensitivity published in 2009. Data was obtained from 969 families regarding food frequency ingested by the pregnant mother's at 36 weeks gestation and at 3 months, 6 months, 9 months, 1 year, 2 years, and 3 years of life. During infancy and early childhood information regarding feeding practices, immunizations, and symptoms of atopy were obtained. All children were skin prick tested at ages 1 year, 2 years, and 3 years of life. A positive reaction was a 3 mm diameter greater than the control group. At 1 year a total of 17 children were sensitized to any of the predefined food allergens and 23 children at age 3 years of life. In this small number of children frequency of food intake during pregnancy did not appear to influence the development of food hypersensitivity. A total of 614 mothers who breastfed for greater than 1 week were asked about food avoidance during breastfeeding 265/614 reported avoiding one of the main foods from their diets with 39 mothers avoiding more than one of the main food allergens. No difference was found between mothers with and without a history of atopy. Statistical conclusion could not be made due to the small number of mothers avoiding each of the major allergens. Results of this study indicate maternal intake during pregnancy and during breastfeeding did not appear to influence the development of sensitization. This observational study does not support the impact of maternal factors on allergic disease (Venter, 2009).

The review by Host and Halken (2005) evaluates the primary prevention of food allergies in infants. This critical review focuses specifically on the prevention of food allergies in infants with maternal factors being only one component. In the prospective observational controlled studies reviewed findings revealed no evidence for preventative dietary intervention is linked to food allergies in infants or children therefore, no need for a special diet during pregnancy or lactation is required (Host and Halken, 2005).

A Cochrane Review (2009) concluded that an avoidance diet for women who are pregnant or lactation is unlikely to reduce the risk the infant developing atopic disease. The objective of this study was to assess the effects of prescribing an avoidance diet during pregnancy pr lactation to prevent food allergies or atopic disease in the infant. All studies evaluated were controlled comparisons of maternal dietary antigen avoidance at any time during pregnancy. Participants were pregnant or lactation women who were at risk for having an atopic infant. Exclusion diets avoided cow's milk, eggs, peanuts, fish and chocolate. Data was collected through electronic database searches and analyzed for their results. Results indicated maternal factors are not associated with atopy and further research is need. Implications for further research recommended are studies with larger sample sizes, longer trials, and stricter adherence to the studies guidelines (Kramer and Kakuma, 2009).

Summary of Findings

A total of eight articles were analyzed for this review. The findings of these articles show inconsistencies in findings and no direct link or strong correlation regardless of fetal exposure to the allergen or complete elimination from the diet. Based on the data maternal factors associated with the development of food allergies remains inconclusive. The most recent studies indicate a link, but further research on maternal transmission and diet modification is still required.

The two most recent articles evaluating maternal peanut consumption during pregnancy and lactation and an additional study published in 2007 evaluating a variety of foods consumed during the last 4 weeks of pregnancy all indicated a strong link between maternal diet and the development of an allergy in the infants. The findings are consistent with an association between the development of an increased likelihood of allergy development or sensitization following regular early exposure during pregnancy and lactation. Although the three studies found different strengths of association between external maternal factors and infant allergies or sensitization, results focused on a consistent theme. Two articles indicating a need for further evaluation of the issue found inconclusive data signifying the need for further research before a definite recommendation would be made. These two articles looked at a variety of dietary approaches to the prevention of a food allergy with maternal factors being only one component. The remaining three articles do not link any maternal dietary factors to the development of food allergies in infants.

Implications for Practice and Recommendations

The American Academy of Pediatrics currently states there is a lack of evidence surrounding maternal dietary modifications during pregnancy and lactation and more data needs to be collected. Until more research is done and there is concrete evidence that maternal factors play a significant role in prevention, current practice and recommendations should be maintained. The American Academy of Pediatrics is known as the gold standard for pediatric patient care in the United States and providers continue to look towards their recommendations for the most accurate and current information. Due to the inconsistency in the findings it is difficult for providers to make recommendations based on the most recent data. Recommendations for the future include the following:

- More research needs to be done in order to conclude a definitive answer regarding the affects of maternal factors on the development of food allergies in children
- The sample sizes of the studies reviewed were of moderate size and would have benefited from a larger cohort studied
- To meet the needs of the diverse population in the US it would be beneficial to also research ethnic populations such as, but not limited to African Americans, Native Americans, Asians, Hispanics, and Somalis.

The impact of these findings resulting in a new guideline regarding the effects of maternal nutrition during pregnancy and lactation on food allergies would greatly benefit the pediatric population. The ability to advise and educate mothers on how to prevent the development of food allergies is a primary focus of health promotion and disease prevention.

Food allergies are a public health concern. Children of all ages are affected by this potentially life-threatening reaction. Raising the awareness of food allergies and food allergy prevention would reduce the risk of unknown and unwanted exposures. Action should be taken to inform others the impact food allergies have on society both socially and economically.

As nurse practitioners promoting evidence-based practice and standards of care, follow up care should be emphasized. Childhood food allergies should be evaluated on a regular basis because often time's allergies can be outgrown. Follow-up and management of food allergies is completed with the patient, family, and primary care provider; others involved may also include a pediatric allergist, dietitian, or childcare providers. The main goal is to maintain nutrition levels adequate for normal growth and development while preventing nutritional deficits and exposure to the offending food. If an allergic reactions does occur education on prompt, appropriate response is also essential. If the child has a severe or life threatening reaction self-

administered epinephrine is prescribed. Parents and caregivers should be taught on how and when to administer the medication and the child should have it with them at all times. It is also important for the provider to offer recommendations for on going support, resources, and advocacy for the families (Burns, Dunn, Brady, Starr, and Blosser, 2009).

Recommended Online Resources

- www.app.org: The American Academy of Pediatrics website is an excellent resource of the latest recommendations in pediatric care. Most of their articles are available on line for viewing, printing, and reference.
- www.aafa.org: The Asthma and Allergy Foundation of America is a non-profit organization aimed at informing patients and provider's practical information regarding asthma and allergies, community services, and support groups.
- www.cdc.gov: The Centers for Disease control is a national website offering information and statistics regarding the current status and recommendations of common illnesses in the US
- www.health.state.mn.us: The Minnesota Department of Health provides local information regarding the current status of health in Minnesota.

Conclusion

The growing prevalence of food allergies in children emphasizes the need for prevention. Determining whether maternal factors such as nutritional and diet modification during pregnancy and lactation are correlated to allergy development or prevention was evaluated. Eight articles were analyzed and critiqued for current findings and recommendations surrounding the issue. Based on these findings, inconclusive data indicates the need for further research before a recommendation can be made. Because maternal health during pregnancy and breastfeeding are

essential aspects of healthy infants and children accurate, conclusive findings are a crucial component to the development of a clinical guideline.

References

Abrahams, V.M. (2011) Immunology of the maternal-fetal interface. In *UpToDate,* edited by E. Richard Stiehm and Charkes J. Lockwood, published by UpToDate in Waltham, MA. As an online service.

Branum, A.M. and Lukacs, S.L. (2008). Food allergy among us children: trends in prevalence and hospitalizations. *NCHS Data Brief, 10*. Retreived from www.cdc.gov

Burns, C.E., Dunn, A.M., Brady, M.A., Starr, N.B., & Blosser, C.G. (2009). *Pediatric Primary Care*: Fourth Edition. Saunders. St. Louis, Missouri.

Dalal, I. and Roifman, C.M. (2011). Immunity of the newborn. In *UpToDate,* edited by E. Richard Stiehm, published by UpToDate in Waltham, MA. As an online service.

DesRoches, A., Infante-Rivard, C., Paradis, L., Paradis, J., and Haddad, E. (2010). Peanut allergy: is maternal transmission of antigens during pregnancy and breastfeeding a risk factor? *Journal of Investigative Allergology and Immunology, 20* (4), 289-294.

Fleischer, D.M. (2011) Primary prevention of allergic disease: maternal avoidance diets in pregnancy and lactation. In *UpToDate,* edited by Scott H. Sicherer, published by UpToDate in Waltham, MA. As an online service.

Greer, F.R., Sicherer, S.H., and Burks, A.W. (2008). Effects of early nutritional interventions on the development of atopic disease in infants and children: the role of maternal dietary restriction, breastfeeding, timing of introduction of complementary foods, and hydrolyzed formulas. *Pediatrics,* 121, 183-191.

Heine, R.G. and Tanh, A.L.K. (2008). Dietary approaches to the prevention of food allergy. *Current Opinion in Clinical Nutrition and Metabolic Care,* 11, 320-328.

Host, A. and Haulken, S. (2005). Primary prevention of food allergy in infants who are at risk.

Current Opinion in Allergy and Immunology, 5, 255-259.

Kimber, I. and Dearman, R.J. (2002). Factors affecting the development of food allergy. *Proceedings of the Nutritional Society,* 61, 435-439.

Kramer, M.S. and Kakuma, R. (2009). Maternal dietary antigen avoidance during pregnancy or lactation, or both, for preventing or treating atopic disease in the child. *Cochrane Database of Systematic Reviews*, 2009(1), Article CD000133. Retrieved March 31, 2011 from *PubMed*.

Nolte, H., Kowal, K., and Dubuske, L. (2011). Overview of skin testing for allergic disease. In *UpToDate,* edited by Bruce S. Bochner and Robert A. Wood, published by UpToDate in Waltham, MA. As an online service.

Sausenthaler, S., Koletzko, S., Schaaf, B., Lehmann, I., Borte, M., Herbarth, O., et al. (2007). Maternal diet during pregnancy in relation to eczema and allergic sensitization in the offspring at 2 year of age. *American Journal of Clinical Nutrition,* 85, 530-537.

Sicherer, S.H., Wood, R.A., Stablein, D., Lindblad, R., Burks, A.W., Liu, A.H., et al. (2010). Maternal consumption of peanut during pregnancy is associated with peanut sensitization in atopic infants. *Journal of Allergy and Clinical Immunology,* 126(6).

Simons, F.E. and Camargo, C.A. (2011) Anaphylaxis: rapid recognition and treatment. In *UpToDate,* edited by Bruce S. Bochner, published by UpToDate in Waltham, MA. As an online service.

Tortora, G.J. and Derrickson, B. (2009). *Principles of Anatomy and Phisiology, 12th edition.* John Wiley & Sons. Hoboken, NJ.

Venter, C., Pereria, B., Voigt, K., Grundy, J., Clayton, C.B., Higgens, B., et al. (2009). Factors

associated with maternal dietary intake, feeding and weaning practices, and the development of food hypersensitivity in the infant. *Pediatric Allergy and Immunology,* 20, 320-327.