



EAACI position paper on diet diversity in pregnancy, infancy and childhood: Novel concepts and implications for studies in allergy and asthma

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Abstract

To fully understand the role of diet diversity on allergy outcomes and to set standards for conducting research in this field, the European Academy of Allergy and Clinical Immunology Task Force on Diet and Immunomodulation has systematically explored the association between diet diversity and allergy outcomes. In addition, a detailed narrative review of information on diet quality and diet patterns as they pertain to allergic outcomes is presented. Overall, we recommend that infants of any risk category for allergic disease should have a diverse diet, given no evidence of harm and some potential association of benefit in the prevention of particular allergic outcomes. In order to harmonize methods for future data collection and reporting, the task force members propose relevant definitions and important factors for consideration, when measuring diet diversity in the context of allergy. Consensus was achieved on practice points through the Delphi method. It is hoped that the definitions and considerations described herein will also enable better comparison of future studies and improve mechanistic studies and pathway analysis to understand how diet diversity modulates allergic outcomes.

KEYWORDS

asthma, atopic dermatitis, Diet diversity, food allergy, prevention, rhinitis

1 | INTRODUCTION

To increase our understanding of the complex relationship between nutrients and other essential components of food, there has been a growing interest in taking a whole diet approach when studying disease outcomes. In particular, research focusing on diet diversity has received significant attention due to its potential role in the prevention of allergic diseases. Diet diversity is defined as the number of different foods or food groups consumed over a given reference period.¹ Diet variety, a term often used in the literature, is considered synonymous with diet diversity.¹ A list of nutritional definitions relevant to this paper are summarized in Box 1.

As nutrients and foods are not eaten in isolation, and an intake of one food or nutrient may inadvertently lead to reduced intake of another, dietary diversity is a challenging area of research.

The task force appreciates that intake of single food or nutrients may also play an important role in the development of allergic diseases. One such example is the study by Bisgaard et al,¹⁰ showing that supplementation with omega-3 fatty acids in the third trimester of pregnancy reduced the risk of persistent wheeze or asthma. The control group was, however, taking olive oil, a key component and indicator of the Mediterranean diet, which could be attributable to reduced allergy outcomes even in the control group.^{11,12}

The mechanistic basis for how diet diversity potentially affects allergy outcomes need further clarification,¹³⁻¹⁵ but may ultimately be mediated through a multitude of immune tolerance mechanism including T and B regulatory cells, immune-regulatory cytokines and suppressed IgE antibodies as demonstrated in other allergen

tolerance models.¹⁶ Possible mechanisms by which diet diversity could affect allergy outcomes are summarized below:

1. It is postulated that a more diverse diet may indirectly affect tolerance development via an effect on the microbiome. This is supported by studies showing that increased diet diversity leads to increased microbial diversity in the elderly¹⁷ and in infants during the introduction of solid food.¹⁸ Increased microbial diversity¹⁹ or abundance of certain bacteria²⁰ has been associated with reduced allergy outcomes. However, there is a paucity of information linking diet diversity, microbial diversity and allergy outcomes.
2. Diet diversity does not provide a finite indication of diet quality, but it may be associated with increased nutrient intake whereas a more diverse diet may indirectly affect allergy outcomes by providing nutrients associated with prevention of allergic diseases such as omega-3 fatty acids and nondigestible fibres.^{21,22}
3. A more diverse diet may also lead to exposure of different food antigens that impact development of immune tolerance, though this may be "low dose" exposure,²³ supporting recent randomized controlled trials regarding early allergen introduction.²⁴⁻²⁷

The focus of this paper is to systematically review and critically address the current knowledge of the association between *diet diversity and allergy outcomes*. To fully understand the role of diet diversity on allergy outcomes and to set standards for conducting research in this field, this review will explore the concept of diet diversity and describe this in the context of other dietary measures, such as diet quality and diet patterns. This is followed by a systematic search

Box 1 Definitions: Dietary diversity, dietary variety, dietary quality, dietary patterns

Diet: Food intake

Nutritional adequacy: The comparison between the nutrient requirement and nutritional intake of an individual or population.²

Dietary diversity: The number of different foods or food groups consumed over a given reference period. It is not a proxy for nutrient adequacy.¹

Dietary variety: A term often used in the literature, is which considered synonymous to dietary diversity.¹

Dietary quality: No official definition in the literature. Definitions vary widely, and many different measurement tools are used. Diet quality may reflect nutrient adequacy but it is not always the case.¹

Dietary patterns: This term reflect an individual's food choice and differ across nationality, culture, socio-economic class and religion. Dietary patterns represent an overall view of food intake and may be a better indicator of disease risk than studying specific foods and nutrients.³

Complementary food/Wearing foods: "When breastmilk is no longer enough to meet the nutritional needs of the infant, complementary foods should be added to the diet of the child. The transition from exclusive breastfeeding to family foods, referred to as complementary feeding, typically covers the period from 6 to 18-24 months of age, and is a very vulnerable period."⁴

Sensitization to food allergens: Sensitization is defined as the presence of allergen-specific IgE (sIgE) to food allergens without having clinical symptoms on exposure to those foods.⁵

IgE-mediated food allergy requires both the presence of specific IgE to the food (sensitization) and the development of signs and symptoms when exposed to the food.⁵

Food Frequency Questionnaire: A questionnaire which enquires about the frequency of consumption of a specific list foods over a period of time.⁶

Semi-quantitative/quantitative food frequency questionnaire: Asks about portion size, gathered either by using free description, as standardized portions or a choice of portion sizes in addition to frequency of consumption.

Quantitative food frequency questionnaire: Uses measures of food eaten, for example measures such as grams or mls as opposed to using measures such as portion sizes/cups/spoons.⁷

24-hour recall: Gathers information about all foods and drinks (and nutritional supplements in some cases) consumed over the previous 24 hours.⁸

Processed food: Considered to be natural foods manufactured with the addition of salt or sugar.⁹

and review of the association between diet diversity and allergy outcomes. An overview of information on diet quality and diet patterns as they pertain to allergic outcomes is also given. Finally, this European Academy of Allergy and Clinical Immunology (EAACI) Task

Force proposes factors for consideration and relevant definitions when measuring diet diversity in the context of allergy, with the aim to standardize methods for future data collection and reporting. The authors of this taskforce hope that this paper and it's recommendations (Table 1) will also better enable comparison of future studies and improve mechanistic studies and pathway analysis.

2 | METHODS

The EAACI taskforce was formed in 2017 and met in person in 2017 and 2018 at the EAACI annual Congress to discuss proposed publications on the role of diet diversity and allergic outcomes. A search of the literature regarding diet quality, diet diversity and food patterns to inform future practice and research relating to allergic outcomes was performed. The full literature search and terms are listed in File S1. Studies on single nutrients or single foods were excluded from the search as the review focused on diet diversity and quality opposed to nutrient/food outcomes.

Searches 1 and 2 were conducted to give background information on the use of diet diversity and diet quality in characterizing nutrition intake and healthy/nutrition outcomes. Search 1 focused on diet diversity in pregnancy, infancy, childhood and in households compared to health/nutrition outcomes (Section 1). Search 2 focused on diet quality in pregnancy, infancy, childhood and households compared to nutrient/food outcomes (Section 2). Searches 3 and 4 included a systematic literature search regarding diet diversity, diet quality and food patterns compared to allergy outcomes. Search 3 focused on diet diversity and diet patterns during pregnancy and allergy outcomes in the infant (Section 3), whereas search 4 focused on diet diversity and food patterns during infancy and childhood and allergy outcomes (Section 4). Key opinion leaders in the field of diet diversity and diet quality were contacted to enquire about guideline papers or book chapters in this field. A modified Delphi panel was used among the task force members to provide consensus on key themes and potential recommendations.²⁸

2.1 | Section 1: Overview of the use of diet diversity to measure nutrition intake in pregnancy, infancy or childhood

Historically, studies on diet diversity investigated (1) child nutrient intake or (2) growth status, and (3) a number of other health outcomes. Studies from both developed²⁹ and developing countries^{30,31} have noted that an increase in diet diversity was associated with an increase in nutrient adequacy. Studies focusing on diet diversity have shown that an increase in diet diversity was associated with better growth indices in young children using height, weight, skin fold thickness and length for age or height for age z-scores to describe stunting, underweight and overweight,³²⁻³⁹ though this might be confounded by socio-economic status. The association between diet diversity and other health outcomes in pregnancy, infancy and childhood (eg obesity)⁴⁰⁻⁴² has also been studied and is summarized in online supplementary Tables S1a-d.

TABLE 1 Factors to consider when measuring diet diversity to improve the validity and reliability of diet diversity measured

Current state of knowledge	
Definitions	See Box 1
Factors to consider in order to measure diet diversity	
Population studied	The majority of studies on diet diversity have been carried out in children. The tool used should take into account measurements in developed vs. developing countries ^{1,126}
Food vs. food group	It is unclear if foods, food groups or both best describe diet diversity since some studies favour foods, others favour food groups or favour both ^{30,43,127,128}
Defining food groups	<p>Food group selection and whether these food groups should be selected based on their nutritional value. This decision should be dependent on the outcome being studied, for example nutritional intake, food security or disease outcome^{1,126}</p> <p>Allergen intake has been used as a marker for diet diversity.¹²⁹ The study found no consistent association between timing and diversity of allergenic solid food introduction and allergic sensitization, physician-diagnosed food allergy or eczema up to 10 y of age.</p> <p>“Food biodiversity is defined as the diversity of plants, animals and other organisms used as food, covering the genetic resources within species, between species and provided by ecosystems” Food biodiversity therefore measures food intake based on their scientific classifications and may be useful to determine nutritional intake.⁴⁴</p> <p>Microbial diversity of food: Studying foods with a high biodiversity of microbial content such as unpasteurized milk (atopic dermatitis and atopy)¹³⁰ and fermented foods such as cheese or yoghurt (food allergy and atopic dermatitis)¹³¹ as an indicator of intake of microbial diversity, and a source of short chain fatty acids, gives an indication that these foods may be associated with allergy prevention.</p> <p>Studies focusing on Westernized diets^{116,117,132} (also high in advanced glycation end products, soda, fruit juice,^{133,134} cured meats¹³⁵ and fast food¹³⁶) suggest that increased intake of advanced glycation end products (AGEs) is associated with reduced diet diversity and increased allergy outcomes. A number of studies have indicated that certain nutrients (eg antioxidants) of foods (fruit and vegetables or fish) may have a beneficial effect on prevention particularly of infant wheeze¹³⁷⁻¹⁴⁰ or eczema in the infant^{77,139}</p>
Portion size	<p>It is unclear if portion size should be included when measuring diet diversity and it will depend on the outcome being studied. Two points are of particular importance.^{1,126,142}</p> <ol style="list-style-type: none"> 1) Should portion size be included to better describe nutritional intake. 2) What is the minimum portion size to include as including foods eaten in small amounts (<15 g of the food) can falsely inflate diet diversity, particularly if nutritional intake is being studied?
Frequency of intake	Whether frequency of intake should be measured, will depend on the question asked ^{1,126,142}
Scoring system	For simple diet diversity, the number of foods or food groups consumed over a given time is usually stated. Weighted scores can be assigned to indicate how often a food or food groups were consumed over a given period of time. More complex scoring systems that include more detailed information such as the number of portions or portion size, usually form part of a dietary index as opposed to indicating diet diversity ^{58,60,143}

Recommendations regarding measuring diet diversity based on current literature

Diet Diversity, diet quality and diet patterns are distinct entities of food intake and these terms should not be used interchangeably

The age group studied must be clearly specified.

The diet diversity tool should preferably be validated in that age group, and sociodemographic information should also be collected.

The diet diversity tools required may be different depending on the populations studied.

Either foods or food groups can be studied to measure diet diversity.

Foods and/or food groups that are studied should be clearly specified and identifiable, for example fresh fruit vs. fruit juice.

Food groups chosen should be selected based on the outcome to be studied.

Practice points regarding measuring diet diversity based on Delphi consensus from the EAACI task force: Voting: >80% agreement

In future, there may be an index that better describes the allergenic potential of foods or food patterns within the context of diet diversity better

Diet diversity scores should be weighted depending on the types of food being measured (eg a diverse fast-food diet is not equal to a diverse fruit and vegetable diet).

Diversity of intake of specific foods, for example range of fruits eaten, different types of cheese consumed, different types of home-cooked or processed foods cannot be used to describe diet diversity but the terms "diversity of fruit intake," "diversity of cheese intake" or "diversity of processed food intake" can be used. These foods can, however, be included in combination with other foods to determine overall diet diversity.

For allergy (asthma, food allergy, eczema, rhinitis, allergic sensitization), there is the opportunity to measure allergenic food diversity, which relates to the number and/or amount of food allergens introduced in a given period of time, for example the first year of life. It is important to understand that allergenic food diversity may not reflect diet diversity, as an emphasis on allergenic food intake may lead to an overall reduced diet diversity.

There is the opportunity to measure food biodiversity, which relates to the taxonomic classification of food intake, for example classifying tomatoes as *Solanum* (genus) opposed to vegetables.

There is the opportunity to measure food microbial diversity which relates to the microbial content of foods.

Diversity of intake of foods containing specific nutrients or ingredients, for example foods containing advanced glycation end products or foods containing omega-3 fatty acids cannot be used to describe diet diversity but the terms "diversity of advanced glycation end products intake" or "diversity of foods containing omega-3 fatty acid intake" can be used. These foods can however be included in combination with other foods to determine overall diet diversity. In these instances, diversity may have a positive (fruit and vegetables) or a negative (advanced glycation end products) connotation. This list may continue to grow as we gain more knowledge about foods/nutrients with immunomodulatory potential, for example bovine trans fatty acids which are isomers of linoleic acid¹⁴¹

Portion size should be defined and measured using quantitative or semi-quantitative questionnaires when required to describe diet diversity

It is not possible at present to define the minimum amount of allergenic protein that should be consumed in order to be sufficient within the allergenic food diversity measure

Frequency of specific food or food group exposures should be measured in diet diversity studies

Scoring systems for diet diversity are simple and measure the number of foods or food groups consumed over a given timeframe

TABLE 1 (Continued)

	Current state of knowledge
Cut-off values	It is difficult to define what constitutes high or low diversity of foods or food groups and cut-off points should be determined in the context of why and in which population the diet diversity is measured. In terms of determining nutrient adequacy, it is recommended that cut-offs should take local food systems and dietary patterns into account. In some instances, sensitivity-specificity analysis ³⁰ or receiver-operating characteristics (ROC) curves ¹⁴⁴ can be used to determine cut-off points
Recall period	The optimal recall period required to assess dietary diversity will depend on daily variety of intake, risk of recall errors, and for intake data can be used at individual or population level. On an individual level, the number of foods being eaten reaches a plateau after 10-15 d. ¹⁴⁵ In addition, as the curve increases steeply for the first 3 d, measuring diet diversity based on a 1-day recall may underestimate dietary diversity. On a household or population level, the reference period is still unclear, but seems to be shorter than measuring nutrient intake at an individual level. Ultimately, the number of days included in the recall period should take participant burden and feasibility into account ^{1,146-148}
Primary source of food procurement and food preparation	Information about the primary source of food procurement may be important to determine intake of either for the whole or certain specified foods/food groups (fruit, vegetables, dairy), for example. ¹⁴² 1 = Own production, gathering, hunting, fishing. 2 = Purchased. 3 = Borrowed, bartered, exchanged for labour, gift from friends or relatives. 4 = Food aid. 5 = Other. Food preparation can also affect the microbial content of food. Food preparation can therefore potentially affect the allergy preventative potential of foods ^{9,62,149-151}
Consumption of fortified foods	If digestion of fortified foods is important for the outcome studied, then the questionnaire may need to address this or at least consider the local availability of these ^{126,142}
Mixed dishes or foods with multiple ingredients	As with foods consumed in small amounts, it is important to err on the side of caution and not inflate what was eaten ^{126,142}
Open recall methods or predefined list of food	There is anecdotal evidence that predefined lists of food eaten may collect less complete information than when food intake is determined in an open/unstructured fashion ^{126,142}
Age of weaning and introduction of infant formula	Data suggest that age of introduction of solid foods, ¹⁵² food allergens ^{24,25} and infant formula ¹⁵³ may affect allergy outcomes. Introduction of solid foods during the weaning period increase the diversity of the gut microbiome ¹⁸
Other factors that may affect allergy outcomes	Studies looking at growth, nutritional intake and health outcomes other than allergic diseases, do not need to control for other risk factors of allergic diseases, but these will need to be controlled for in diet diversity studies ¹⁵⁴

Recommendations regarding measuring diet diversity based on current literature

Cut-off values of high/low diversity can only be correctly ascertained at a population level, where the sample is robust and representative of the population to allow generalizability. Consequently, these may differ in different populations

The recall period and method of data collection should be clearly specified, but age of introduction for each food during infancy should also be reported given this may have a potential significant confounding effect

Investigators should consider ways to ensure measuring food procurement in their study design, and if/when possible, this should be detailed for both diet diversity and diet quality

If fortified foods are consumed, the study should aim to capture these data, and the amount quantified.

If any vitamin or mineral or nutritional supplements is consumed in the study, this should be reported, including brand and the amount consumed. Detail of probiotic and prebiotic supplements should also be recorded. Foods already supplemented with any of the above should also be clearly recorded.

If mixed dishes are consumed, this should be clearly specified, and the method for how individual food or allergen content quantification was achieved should be fully detailed for both diet quality and diet diversity.

Open recall, for example 24-h recall, is the preferred method of ascertaining foods or diet quality index. Predefined lists are less preferable, but if used, these should be clearly defined in the methods or in a File S1

There is a nonexhaustive list of factors that may affect allergy outcomes, but potential confounders should be corrected for analysing associations between diet diversity and allergy outcomes

Practice points regarding measuring diet diversity based on Delphi consensus from the EAACI task force: Voting: >80% agreement

When possible, studies reporting diet diversity data should include sensitivity, specificity, true and false positive/negative values to assist future meta-analyses and comparisons. They should also de-emphasize reporting of a cut-off value for a sample

For pregnant women, we recommend a minimum recall period of 2 weekdays and 1 weekend day (ie 3 d), measured at repeated intervals throughout pregnancy. When possible, portion sizes should be collected and reported. For infants, we recommend food recall periods within the first year of life. These periods may include the first four months of life, the first six months of life or the first year of life. Repeated measure of intake is recommended to improve the quality of data, rather than just recording consumption of a particular food/food group once. When possible, portion sizes should be collected and reported and data should ideally be recorded prospectively

Investigators should consider ways to ensure measuring how food is prepared/cooked (eg Raw, home cooked, processed, ultra-processed and fermented) is recorded in their study design, and if/when possible, this should be detailed for diet diversity. Ideally, foods should be defined as raw, home-cooked, processed or ultra-processed

Age of introduction of solid foods, allergenic foods and infant formula should be clearly indicated in studies investigating the association between diet diversity and allergy outcomes.

Introduction of infant formula cannot be classified on its own as diet diversity but should be included in measuring diet diversity of food and food groups. Introduction of infant formula may in fact reduce diet diversity as breastmilk potentially exposes an infant to the diversity of the maternal diet, whereas formula is uniform

Diet diversity can be measured by counting individual foods,^{31,37,42-46} food groups^{30,34-36,38,39,41,43,47-54} or foods within a group,^{32,33,55,56} and can be measured over a time period ranging from the previous 24 hours (24-hour recalls)^{31-35,38,40,44,46,48} or over a 7-day period^{33,39,42,47,53,55-57} to intake over one year. Food intake data may be collected once^{32-34,38,40,44,48} or a number of times^{31,35,46} as summarized in Table S1a-d. Factors that should be considered when measuring diet diversity are summarized in Table 1.

2.2 | Section 2: Overview of the use of diet quality in measuring nutrition intake

The term diet quality is broadly used as an umbrella term to describe a healthy, balanced, nutritious diet for optimal health characterized by limited amounts of fat, saturated fat, cholesterol, sodium and refined sugars, and meeting the recommended amounts of fruits, vegetables and whole grain products.⁵⁸ Diet quality is often described using a nutrition index, although food patterns, for example the Mediterranean diet, are also often referred to as indicators of diet quality. The purpose of diet indices is to synthesize a large amount of dietary information into a single useful indicator.⁵⁹ Over the years, various indices have been developed to measure diet quality, amended and validated to evaluate healthy dietary patterns such as the healthy eating index (HEI) score,⁶⁰ the Mediterranean diet score⁶¹ and more complex indices.⁶²⁻⁶⁴ Scoring is typically based on consumption of nutrients, foods or both⁶⁵ though there is wide heterogeneity in methods used. Table S2 summarizes the most commonly used dietary indices in pregnancy, infancy and childhood.

2.3 | Section 3: Diet diversity and diet quality (indices and diet patterns) during pregnancy and allergy outcomes in the infant

Diet diversity and diet quality may be used to find associations between dietary intake and allergy outcomes in either pregnancy or infant's early life or both. We conducted a systematic review of the literature to identify relevant studies, investigate the measures used and describe any associations found with allergy outcomes.

2.3.1 | Diet diversity in pregnancy and allergy outcomes

While this is an important relationship to understand, no studies have been identified exploring an association between diet diversity in pregnancy and allergy outcomes.

2.3.2 | Diet quality in pregnancy and allergy outcomes

Studies on diet quality in pregnancy and allergic diseases have used the Mediterranean diet score⁶⁶⁻⁷⁰ or a modified version of the HEI in pregnancy^{71,72} to investigate an association with allergic outcomes.

2.3.3 | Mediterranean diet in pregnancy and allergy outcomes

Four papers from longitudinal birth cohort studies^{66-68,71} (two from Spain, one from Greece and one from the USA) studied the association between Mediterranean diet patterns and atopic outcomes in the offspring (Table S3). Typically, a diet score is developed and dietary information, obtained from food recalls (food diaries, 24-hour recalls, food diaries), is used to calculate the Mediterranean diet score.⁶¹ In two studies, dairy intake^{67,68} was added to the Mediterranean diet score. All studies adjusted their analyses for common confounders, though these varied between studies. Two studies included the use of vitamin/mineral supplements in their dietary analysis^{66,71}; in one study, it was unclear if the use of supplements was included in the analyses⁶⁷ and one study adjusted for mineral/vitamin intake only in the statistical analyses.⁶⁸

Outcomes reported in the infants were wheeze in all studies,^{66-68,71} asthma in one study,⁷¹ and rhinitis, atopic dermatitis and/or eczema in three studies.⁶⁶⁻⁶⁸ Sensitization to food/aero-allergens was determined in two studies.^{68,71} None of these studies reported on food allergy as an outcome, highlighting a true deficiency in the literature. All outcome measures were parental reported and/or doctors diagnosed.

In 3 of the 4 studies,^{66,67,71} a Mediterranean diet pattern during pregnancy was not associated with the development of atopic diseases in the offspring. However, one study⁶⁸ indicated that the Mediterranean diet was associated with reduced prevalence of persistent wheeze, atopic wheeze and atopy.

2.3.4 | Other dietary patterns or indices used in pregnancy

Investigating dietary patterns other than the Mediterranean diet, we found four longitudinal birth cohort studies:⁷²⁻⁷⁵ three studies using semi-quantitative FFQs, from which the diet patterns were analysed, and one used a 24-hour recall⁷³ (Table S4). In three studies, the use of vitamin/mineral supplements was not reported, and in one, the use of supplements was excluded.⁷²⁻⁷⁵ All studies adjusted their analyses for common confounders, although confounders varied widely between studies.

In one study, the Alternate Healthy Eating Index modified for pregnancy (AHEI-P) developed by Rifas et al⁷⁶ was used to examine associations with allergic outcomes,⁷² and in three studies, food patterns were studied.⁷³⁻⁷⁵ In these three studies, principal component analysis was used to assess individual food intake and the following food patterns emerged as associated with allergic outcomes:

- Seafood and noodle pattern (noodle soup, noodles with sauce, fish, seafood and seafood products);
- Vegetable, fruit and white rice pattern (vegetables, fruit, whole grain bread and white rice with minimal processed foods/meats);
- Pasta, cheese and processed meat pattern (pasta, processed grains, cheese and processed meats)⁷³;

- Healthy pattern (high consumption of green and yellow vegetables, seaweed, mushrooms, white vegetables, pulses, potatoes, fish, sea products, fruit, and shellfish and low intake of confectioneries and soft drinks);
- Western pattern (high consumption of vegetable oil, salt-containing seasonings, beef and pork, processed meat, eggs, chicken, and white vegetables and low intake of fruit, soft drinks, and confectioneries);
- Japanese pattern (high consumption of rice, miso soup, sea products, and fish and low intake of bread, confectioneries and dairy products)⁷⁴;
- Health conscious pattern (salad, fruit, fruit juices, rice, pasta, oat/bran, fish);
- Traditional pattern (vegetables, red meat and poultry);
- Processed pattern (meat pies, sausages, burgers, fried food);
- Vegetarian pattern (meat substitutes, pulses, nuts, herbal tea);
- Confectionary (chocolate, sweets, biscuits, cakes and puddings).⁷⁵

Outcomes measured were asthma,^{73,75} eczema,^{73,74} rhinitis⁷³ and sensitization to food/aero-allergens.^{72,73,77} The only positive associations reported were reduced sensitization due to the seafood and noodle pattern⁷³ and reduced wheeze due to the Western diet pattern.⁷⁴ The seafood and noodle pattern⁷³ was associated with a reduced risk of developing allergen sensitization at both 18 months [odds ratio (95% confidence interval): 0.7 (0.5-0.9)] and 36 months [odds ratio (95% confidence interval) 0.7 (0.6-0.9)]⁷³; the maternal Western diet pattern was associated with a reduced risk of wheeze, and in adjusted analysis, the OR between extreme quartiles was 0.59 (95% CI: 0.35-0.98, *P* for trend = 0.02).^{74,75} Once again, no study looked at food allergy as an outcome.

2.4 | Section 4: Diet Diversity during infancy and allergy outcomes in the infant

2.4.1 | Diet diversity and allergen sensitization

IgE sensitization is a commonly used (but limited and imprecise) marker of clinical allergy. Roduit et al¹⁶ investigated the association between diet diversity and allergic disease and collected sensitization data based on specific IgE (Table 2) in the Protection Against Allergy Study in Rural Environments (PASTURE) prospective cohort study, which enrolled children from Austria, Finland, France, Germany and Switzerland. Diet diversity in this study was defined as the 15 foods commonly eaten by 80% of the children in the study in the first year of life: any cow's milk, yogurt, other milk product; eggs; nuts; vegetables or fruits; cereals; bread; meat; fish; soya; margarine or butter; cake; and chocolate. A second definition was also used, including the 6 major foods introduced in the first 6 months or first 12 months of life: vegetables or fruits; cereals; bread; meat; cake; and yogurt. Children with a low-diet diversity, as defined as above, had an increased risk of sensitization to food allergens at age 4.5 or 6 years, but no significant associations were found with sensitization to inhalant allergens. The association remained significant,

albeit weaker, in a subgroup analysis of children without food allergy, respiratory disorders or both. Subgroup analysis was carried out to consider the potential risk of reverse causality, given early-onset allergy or family history of allergy could lead to low-diet diversity due to different feeding practices in those infants. The German LISAPlus study assessed the impact of diet diversity on allergic disease.⁷⁸ That study concluded that children in the highest quartile of food group diversity had lower odds of allergic sensitization to aeroallergens. This finding was also supported when food group diversity was treated as a continuous variable. Similarly, in a Finnish birth cohort of over 3500 children, Nwaru et al⁷⁹ studied the association between diet diversity (defined as the number of foods introduced at 3, 4 and 6 months of age) and sensitization to food and aeroallergens at the age of 5 years (see Table 3 for definitions used). After adjustment for several demographic and parental factors, they found that reduced diet diversity as early as 3 months was associated with an increased risk of sensitization to specific food and aeroallergens. While the adverse risk estimates became much stronger with increased diet diversity at 4 and 6 months, the authors found that, compared to non-high-risk children, the at-risk children (ie those with atopic eczema by 6 months of age or those with a parental with history of allergy) had a greater risk of allergic sensitization.

2.4.2 | Diet diversity and food allergy

Only one study has focused on the potential association of the diversity of food intake and the development of food allergy (Table 3). In the aforementioned PASTURE study, Roduit et al¹⁶ showed that children with a more diverse diet had a lower prevalence of food allergy, measured as a report of a doctor-diagnosed food allergy but not necessarily an allergy proven by oral food challenge. In this study, the inclusion of 0-6 items from vegetables/fruits, cereals, bread, meat, cake and yogurt within the first 6 months or first year of life, respectively, was recorded. The study showed that children with a low-diet diversity score (consumed fewer of the items listed above) had an increased risk of food allergy up to six years of age, compared to children having more food items in their diet.

2.4.3 | Diet diversity and atopic dermatitis

A limited number of studies have investigated the association between diversity of complementary foods and the risk of atopic dermatitis (AD) in children. In all studies, diet diversity was defined as the sum of the number of complementary foods that had been introduced into the child's diet (even if eaten only once) up to a specified time point, usually within the first year of life. We identified eight unique studies (seven birth cohorts and one matched case-control study), reporting their data in 12 publications originating from Germany, Italy, New Zealand, Finland, Austria, France and Switzerland (Table 4). The pooled data from the GINIPlus and LISA studies revealed that the early introduction of solids with a high diversity before the end of the fourth month was associated with an increased risk of AD at two and six years, but

TABLE 2 The association between diet diversity and sensitization to food and aero-allergens

Study and country	Definition and assessment of diet diversity	Results	Comments
Roduit et al (2014) ¹⁶ Prospective birth cohort study Protection Against Allergy Study in Rural Environments (PASTURE/ EFRAIM). Stratified in farm and non- farm children Sample Size: Baseline N: 1133 Analytic N: 856 Attrition: 75% Sex: 49.5% girls and 50.5% boys Race/Ethnicity: Rural areas of 5 European countries (Austria, Finland, France, Germany and Switzerland). Atopic Disease Risk Status: 53.6% had at least 1 al- lergic parent Background Diet: 47.4% were breastfed for more than 6 mo (not exclusively)	Intervention/Exposure: Parental administered questionnaires for asthma, rhinitis and food allergy within the third trimester of pregnancy and when the children were 2, 12, 18 and 24 mo of age and then yearly up to age 6 y Asthma: at least one either doctor-diagnosed asthma or at least 2 doctor-diagnosed episodes of obstructive bronchitis in the last 12 mo in the year 4, 5, or 6 questionnaires independent of a diagnosis reported in the first 3 y of life. Food allergy: at least once been given a diagnosis of food allergy by a doctor up to age 6 y. Allergic rhinitis: Reported presence of symptoms (itchy, runny, or blocked nose without a cold and associated with red itchy eyes) or doctor-diagnosed allergic rhinitis in the 6- year questionnaire. DD was assessed through food items in monthly food diaries from 3rd-12th months. DD scores were calculated as follows: (1) using major food items introduced in the first year of life (including vegetables or fruits, cereals, bread, meat, cake and yogurt); (2) with the same major food items but introduced in the first 6 mo of life; and (3) with all food items introduced in the first year of life (including any cow's milk, yogurt, other milk product, eggs, nuts, vegetables or fruits, cereals, bread, meat, fish, soy, margarine, butter, cake and chocolate) Assessment Methods: parent self-administered questionnaire on dietary intake. Levels of allergen-specific IgE antibodies (Dermatophagoides pteronyssinus, Dermatophagoides farinae, alder, birch, hazel, grass pollen, rye, mugwort, plantain, cat, horse, dog, Alternaria species, hen's egg, cow's milk, peanut, hazelnut, carrot and wheat flour) were measured in blood among children at age 4.5 and 6 y. Sensitization was defined as ≥ 3.5 KU/L Assessment of DD age: at 6 and 12 mo of age	Complementary food introduction associated with asthma, food allergy, allergic rhinitis and atopic sensitization up to 6 y of age Assessment Methods: DD score and parent reported asthma, rhinitis and food allergy. Specific IgE levels to indicate sensitization Results Sensitization to any allergen at age 4.5 and/or 6 y was present in 25.5% of children, sensitization to food allergens was present 10.7%, and sensitization to inhalant allergens was present in 22.1%, as measured at 4.5 or 6 y. Increased DD within the first year of life was negatively associated with sensitization to food allergens at 4.5 or 6 y of age. The children with a low DD had an increased risk of sensitiza- tion to food allergens at age 4.5 or 6 y. The analysis with children having doctor-diagnosed food allergy combined with positive food sensitization showed an even stronger negative association with DD (adjusted OR for each ad- ditional food items: 0.55, 95%CI: 0.40-0.76). Children with a low DD, had an increased expression of marker for antibody isotype switching to IgE and a reduced expression of the regulatory T cell-associated gene Foxp3 measured at 6 y of age	Confounders: Confounders adjusted for in this publication. (in particular atopic dermatitis) Limitations: Study population were se- lected from rural Europe and may not reflect general population. Outcomes of asthma, food allergy and rhinitis were based on parental reports. Reverse causality cannot be ruled out in this study DD score used was not validated and made use of allergens and foods like chocolate and margarine as part of the diversity score, which may not be representa- tive of true DD.
Markevych et al (2017) ⁷⁸ Population-based German birth cohort LISAPlus (Influences of Lifestyle related Factors on the Immune System and the Development of Allergies in Childhood) Sample Size: Baseline N: 3097 Analytic N: 2518 Attrition: 81% Sex: See original LISAPlus study Race/Ethnicity: See original LISAPlus study Atopic Disease Risk Status: 57.2% had atopic family history	Intervention/Exposure: Impact of DD within the first 6 mo of life on 5 allergic outcomes: doctor-diagnosed ec- zema, asthma and allergic rhinitis and sensitization to aeroallergens and food allergens. Assessment Methods: Self-administered biannual parental questionnaires on the children's health and on life- style factors from birth to 2 y and at 4 y and 6 y. Blood samples for specific IgE at 2 and 6 y, levels > 0.35 KU/L were defined as sensitization. At 12 mo of age, parents were asked about breastfeeding practices and about the timing of solid food introduction. Possible answer choices included: "1st until 4th month," "5th/6th month," "7th to 12th month," and "solid food item not yet introduced." Forty-eight food items were grouped into 8 food groups: (1) vegetables/avocado, cauliflower, beans, broccoli, peas, cucumbers, carrots, potatoes, white cabbage, turnip, cabbage, lenses, celery, asparagus, spinach, tomatoes, onion, vegetable juices; (2) fruit (apples, pineapples, apricots, bananas, pears, strawberries, peaches, citrus fruit, fruit juices); (3) cereal (bread/pretzels/rolls, cookies/cakes/rusk, rolled oats, muesli, millet, cornmeal/corn starch, wheat semolina/starch, noodles, rice/rice starch, spelt); (4) meat (poultry, lamb, veal/beef, pork, sausages); (5) egg; (6) dairy products (cow milk/cream, yoghurt/quark/cheese); (7) fish; and (8) other (nuts, soy products, cocoa/ chocolate). Assessment of DD age: DD at 6 mo and 12 mo of age	Allergic outcomes at 2 and 6 and 15 y of life Assessment Methods: Several definitions of DD (food group and food item diversity, each treated in quartiles and continuously) during the first year of life. All analyses were stratified by the presence of a wide range of early skin symptoms to test the impact of reverse causality. Allergic outcomes were assessed at 6, 12 and 18 mo and age 2, 4, 6, 10 and 15 y. Results Children in the highest quartile of food group diversity had lower odds 0.61 (0.44-0.85) of allergic sensitization to aeroallergens. When food group diversity was treated as a continuous vari- able, those with a higher DD had statistically significantly less allergic sensitization to aeroallergens in the total population and in children with early skin symptoms. 0.88 (-0.80-0.95) When food item diversity was used as an exposure variable in- stead of food group diversity, similar associations for allergic sensitization to aeroallergens were observed. Highest quartile 0.82 (0.52-0.98); Continuous variable 0.99 (0.97-1.00)	Confounders: Confounders adjusted for: atopic eczema Limitations: Information on frequency and amount of food intake not available. Reverse causality could not completely be ruled out DD score used not validated

(Continues)

TABLE 2 (Continued)

Study and country	Definition and assessment of diet diversity	Results	Comments
Nwaru et al (2013) ⁷⁹ Finnish Type 1 Diabetes Prediction and Prevention study Prospective Cohort Study Finland	Intervention/Exposure: The child's diet was assessed by using age-specific dietary questionnaires at ages 3, 4, 6 and 12 mo and a follow-up "age at introduction of new foods" form for recording the age at introduction of complementary foods. Looked at the number of complementary foods introduced at 3, 4 and 6 mo of age. 4 categories of food diversity at each time point were defined based on the distribution of the data at each time point: at 3 mo, these were "no food item," "1-2 food items," and ">2 food items"; at 4 mo, these were "no food item," "1-2 food items," "3-4 food items," and ">4 food items"; at 6 mo, these were "0-4 food items," "5-6 food items," "7-8 food items," and ">8 food items". Food groups used as DD definition: cow's milk and formula (as a combined variable); potatoes; carrots; turnip; fruits and berries (as a combined variable); cereals (rye, wheat, oats and barley as a combined variable); other cereals (maize, rice, millet and buckwheat as a combined variable), meat; fish; egg; cabbage; spinach; and lettuce. Assessment Methods: Prospective Parent questionnaires Assessment of DD age: birth to 12 mo of age	Sensitization to the following food and aeroallergens were analysed at the age of 5 y: egg, cow's milk, fish, wheat, house dust mite, cat, timothy grass and birch. These outcomes were based on analysis of IgE concentration using ImmunoCAP fluoroenzyme immunoassay. Atopic sensitization was defined as IgE \geq 0.35 kU/L. Less DD as early as 3 mo was associated with an increased risk of allergic sensitization at 5 y. The associations were stronger with DD at 4 and 6 mo and more prominent in at-risk children compared to no-high-risk children. Less DD as early as 3 mo was associated with an increased risk of allergic sensitization at 5 y. Food Allergen sensitization ⁷⁹ : No food at 3 mo vs > 3 foods 1.39 (1.07-1.81); 1-2 foods at 3 mo vs > 3 foods 1.36 (1.06-1.74) P value .021	Confounders: Confounders adjusted for: sex of the child, number of siblings, parental asthma, parental rhinitis, delivery hospital and maternal smoking during pregnancy. Limitations: The study recruited children carrying genetic risk (HLA-conferred susceptibility) for type 1 diabetes. Therefore, may reduce generalizability of findings to general population The number of statistical tests performed could mean that the type 1 error rate might not be at the 0.05 level. DD definition was not validated and could be improved with increasing the variety
Sample Size: Baseline N: 4074 Analytic N: 3781 Attrition: 93% Sex: 1646 boys (52%), 1496 (48%) girls Race/Ethnicity: not mentioned Atopic Disease Risk Status: 16% and 62% of parents had asthma and rhinitis respectively			

TABLE 3 The association between diet diversity and food allergy

Study and country	Definition and assessment of diet diversity	Results	Comments
Roduit C et al JACI 2014 ¹⁶ Prospective birth cohort study Protection Against Allergy Study in Rural Environments (PASTURE/EFRAIM). Stratified in farm and nonfarm children See—food sensitization	See—food sensitization	Food allergy: parental report of ever doctor-diagnosed food allergy up to 6 y of life (overall proportion: 7.4%). Food diversity score (0-6 items): 0-3: FA 21.9% 4-5: FA 9.2% 6: FA 5.7% Food diversity score (0-6 items): 0-3: food sens. 26.9% 4-5: food sens. 13.1% 6: food sens. 8.5% Increased diet diversity in the 1st year of life: reduced risk of reported doctor-diagnosed food allergy up to 6 y (adjusted OR for each additional food items: 0.70, 95%CI: 0.57-0.86) and food sensitization at 4.5/6 y (adjusted OR for each additional food items: 0.72, 95%CI: 0.57-0.90)	Outcome assessment mainly based on parental reports of doctor-diagnosis. Adjustment for the potential confounders was performed: farmer, centre, duration of breastfeeding, parents with allergy, maternal education, sex and number of siblings. Reverse causality was taken into account to some extent, that is—analysis with exclusion of children with FA within the 1st year of life analysis with exclusion of children with AD within the 1st year of age. However did not account for lactose/food intolerances as lack of information and also lack of information on subclinical manifestations of food allergy (colic, gastroesophageal reflux etc)

interestingly not at four years.⁸⁰ Why this statistical anomaly was seen is not clear, and diversity before four months might be highly influenced by breast/formula feeding practices. Conversely, the delayed introduction of solids and reduced diversity beyond 6 months of age was not beneficial for allergy prevention. Prior to the pooling of the GINIplus and LISAPlus studies, the LISAPlus study reported an increased risk of AD at the age of 2 years associated with less diet diversity at four months of age, but they found no association between diet diversity at four months of life and AD at six years.^{81,82} Data from the 15-year follow-up of the LISAPlus birth cohort indicated that children in the highest quartile of diet diversity who were introduced to all 8 food groups during the first year of life had lower odds of developing eczema up to age 15 years when compared with children in the lowest quartile.⁷⁸

A birth cohort from New Zealand found that a more diverse diet during the first 4 months of life was associated with an increased risk of developing AD both at 2 and 3 years and an increased risk of recurrent AD at the age of 10 years.⁸³⁻⁸⁶ In this study, diet diversity was defined as the sum of six food groups (cereals, vegetables, dairy products, meat, fruits, and egg or related products). The Finnish birth cohort showed that lower diet diversity at six months was associated with an increased risk of AD at 5 years and a same tendency was observed with the diet diversity at 12 months of age.⁸⁷ In the European PASTURE study, an increased diet diversity within the 1st year of life was associated with a reduced risk of developing AD through 4 years of age (even after excluding children with AD onset within 1st year of life).⁸⁸ This finding was supported by the outcomes from the matched case-control study from Italy, in which the authors reported that more diverse diet at 4 and 5 months was associated with a reduced risk of AD by 2 years of age.⁸⁹

2.4.4 | Diet diversity and asthma and allergic rhinitis

Data investigating these relationships were limited to two large European prospective birth cohorts (Table 5), the PASTURE study¹⁶ and the Finnish Type I Diabetes Prediction and Prevention Study Prospective Cohort Study.⁸⁷ While these cohorts were recruited for different purposes, similar assessment of diet diversity was recorded during the first year of life, as a variable to help predict allergic outcomes at either ages 5 (Finnish Type I Diabetes Prediction and Prevention Study) or 6 (PASTURE). In the PASTURE cohort, Roduit et al¹⁶ noted that increasing diet diversity in first year of life was associated with a linear protective trend against the development of reported asthma, resulting in a 26% reduction for the introduction of each successive food. However, no protective association was noted for allergic rhinitis or inhalant sensitization. In the Finnish Type I Diabetes Prediction and Prevention Study, Nwaru et al⁸⁷ noted less diversity at 12 months of life was associated with a greater risk of development of any form of asthma at age 5 for 0-7 foods incorporated in the diet and for 8-9 foods incorporated into the diet compared to > 11 foods ($P < .001$), as well as increased risk for any wheeze for 0-7 foods vs. > 11 foods ($P = .004$). For allergic rhinitis, lower diet diversity at both 6 and 12 mo of life was significantly associated with later risk of developing allergic rhinitis. At 6 months, the risk of developing allergic rhinitis was significantly greater with incorporation of 0-4 foods and 5-6 foods vs. > 8 foods ($P = .02$). Similarly, at 12 months the risk was significantly higher with incorporation of 0-7 foods and 8-9 foods vs. > 11 foods ($P < .001$) (see Table 6 for a summary of all allergy outcomes).

TABLE 4 The association between diet diversity and atopic dermatitis

Study and country	Definition and assessment of diet diversity	Results	Comments
Zutavern A et al <i>Pediatrics</i> (2006) ⁸¹ Zutavern A et al <i>Pediatrics</i> (2008) ⁸² Germany LISA study N = 2612 up to 2 years LISA study N = 2073 up to 6 y	Solids diversity was defined by the total number of different food groups (48 single food items were asked in a questionnaire) and classified in 8 groups of solid food: vegetables, cereal, fruit, meat, dairy products, egg, fish and others (like soybean, nuts, cacao, chocolate) introduced in the child's diet at 4 and 6 mo of age Assessment method: parental interview on infant's diet at 6 mo and parental interview on infant's diet at 12 mo	Assessment of AD: parental reports on doctor-diagnosis and symptoms of AD, questionnaires at birth, 0.5, 1, 1.5, 2, 4 and 6 years Introduction of a high number of different solid foods by 6 mo of age reduced the risk for doctor-diagnosed AD up to 2 y in all children (aOR 5-8 food groups versus no solid food: 0.66, 95%CI: 0.46-0.94) and also in the children having early skin or allergic symptoms Increased diet diversity within first 6 mo: reduced risk of AD at 2 y (no significant association with DD at 4 mo and AD) No association between diet diversity at 4 mo and AD at 6 y, for all population. Exclusion of children with early skin or allergic symptoms: increased DD was associated with an increased risk of doctor-diagnosis of AD, but not symptoms of AD at 6 y	Families with higher educational background and high proportion of atopic parents were included. Reverse causality was taken into account: separate analysis with AD children with and without early skin symptoms Confounding factors due to the children included in the analysis; recall bias due to retrospective information regarding introduction of solids—misclassification of feeding history possible; Information on eczema from questionnaire
Filipiak B et al <i>J Pediatrics</i> (2007) ¹⁵⁵ Germany GINI study N = 4753	Solids diversity was defined by the total number of different food groups (48 single food items were asked in a questionnaire) and classified in 8 groups of solid food (vegetables, cereal, fruit meat, dairy products egg, fish, others like soybean, nuts, cacao, chocolate) introduced in the child's diet at 4 and 6 mo of age Assessment method: parental interview on infant's diet at 12 mo	Assessment of AD: parental reports on doctor-diagnosis and symptoms of AD, yearly up to 4 y No association between doctor-diagnosed or symptomatic AD and time point of solid food introduction and diversity of solids Intervention group: family history of allergy, prospectively and randomly assigned to either hydrolysed formula or cow's milk formula; Nonintervention group: no family history of allergy or parents did not wish to participate in intervention trial No association between diet diversity at 4 or 6 mo and AD up to 4 y	Selection bias due to loss of follow-up (nonintervention subgroup 25%, intervention subgroup 14%); Misclassification due to recall bias (exposure was assessed retrospectively at 1 y of age)
Sausenthaler S et al <i>Am J Clin Nutr</i> (2011) ⁸⁰ Germany GINIplus and LISA plus study N = 9088	Solids diversity was defined by the total number of different food groups (48 single food items were asked in a questionnaire) and classified in 8 groups of solid food (vegetables, cereal, fruit meat, dairy products egg, fish, others like soya bean, nuts, cacao, chocolate) introduced in the child's diet at 4 and 6 mo of age; high diversity of solid foods: 3-8 groups Assessment method: parental report	Assessment of AD: LISA study: parental reports on doctor-diagnosis and symptoms of AD, up to 4 y in GINI study and up to 6 y in LISA study No association between diversity of solid foods at the age of 4 mo and 6 mo and doctor-diagnosed AD during first 4 y of life. At 6 y of age significant association between AD and high diversity of solid foods. Symptomatic eczema at 2 y of age was associated with high diversity of diet at 4 mo → conclusion: early introduction of solid foods and high diversity before 17 wk of age may increase allergy risk. Increased diet diversity at 4 mo: associated with increased risk of symptoms of AD at 2 y and doctor-diagnosed AD at 6 y, but not at 4 y	Reverse causation was not controlled for as in the GINIplus study early symptoms of eczema was not assessed; results on association between high diversity of solids in infants within the first 4 mo and increased risk of eczema was not consistent throughout different outcome measures and different time points

(Continues)

TABLE 4 (Continued)

Study and country	Definition and assessment of diet diversity	Results	Comments
Markevych et al (2017) ⁷⁸ Population-based German birth cohort LISAPlus (Influences of Lifestyle related Factors on the Immune System and the Development of Allergies in Childhood)	Intervention/Exposure: Impact of DD within the first 6 mo of life on 5 allergic outcomes: doctor-diagnosed eczema, asthma and allergic rhinitis and sensitization to aeroallergens and food allergens. Assessment Methods: Self-administered biannual parental questionnaires on the children's health and on lifestyle factors from birth to 2 y and at 4 y and 6 y. Blood samples for specific IgE at 2 and 6 y, levels > 0.35 KU/L were defined as sensitization.	Allergic outcomes at 2 and 6 and 15 y of life Assessment Methods: Several definitions of DD (food group and food item diversity, each treated in quartiles and continuously) during the first year of life. All analyses were stratified by the presence of a wide range of early skin symptoms to test the impact of reverse causality. Allergic outcomes were assessed at 6, 12 and 18 mo and age 2, 4, 6, 10 and 15 y. Results Children in the highest quartile who were introduced to all 8 food groups during the first year of life had lower odds of developing eczema up to age 15 y compared with children in the lowest quartile with a maximum of 5 food groups. When food group diversity was treated as a continuous variable, those with a higher DD had statistically significantly less eczema	Confounders: Confounders adjusted for: atopic eczema Limitations: Information on frequency and amount of food intake not available. Reverse causality could not completely be ruled out DD score used not validated
Sample Size: 3097 Analytic N: 2518 Attrition: 81% Sex: See original LISAPlus study Race/Ethnicity: See original LISAPlus study Atopic Disease Risk Status: 57.2% had atopic family history	At 12 mo of age, parents were asked about breastfeeding practices and about the timing of solid food introduction. Possible answer choices included: "1st until 4th month," "5th/6th month," "7th to 12th month," and "solid food item not yet introduced." Forty-eight food items were grouped into 8 food groups: (1) vegetables (avocado, cauliflower, beans, broccoli, peas, cucumbers, carrots, potatoes, white cabbage, turnip, cabbage, lentils, celery, asparagus, spinach, tomatoes, onion, vegetable juices); (2) fruit (apples, pineapples, apricots, bananas, pears, strawberries, peaches, citrus fruit, fruit juices); (3) cereal (bread/pretzels/rolls, cookies/cakes/rusk, rolled oats, muesli, millet, cornmeal/corn starch, wheat semolina/starch, noodles, rice/rice starch, spelt); (4) meat (poultry, lamb, veal/beef, pork, sausages); (5) egg; (6) dairy products (cow milk/cream, yoghurt/quark/cheese); (7) fish; and (8) other (nuts, soy products, cocoa/ chocolate). Assessment of DD age: DD at 6 mo and 12 mo of age	Diagnosis of AD by dermatologist, at the age of 3-24 mo Weaning at age 4-5 mo was inversely associated with AD risk. Introduction of a high number of different solid foods at 4 and 5 mo was associated with a reduced risk of AD (OR: 0.30, 95% CI: 0.11-0.81 \geq 3 foods vs 0 foods at 4 mo; OR: 0.44, 95% CI: 0.21-0.91 \geq 8 foods vs 0 foods at 5 mo)	Maternal recall bias, reverse causation by prolonged exclusive breastfeeding due to mothers considering children at risk for allergies; lack of information on maternal diet during pregnancy and lactation.
Turati F et al Allergy (2016) ⁸⁹ Italy Matched case-control study on incident physician-diagnosed AD (451 cases and 451 controls)	The total number of food items included in the infant's diet at 4 and 5 mo of age; no solids, 1-2 foods, 3-22 foods Assessment method: face to face questionnaire		

(Continues)

TABLE 4 (Continued)

Study and country	Definition and assessment of diet diversity	Results	Comments
Nwaru et al (2014) ⁸⁷ Finnish Type 1 Diabetes Prediction and Prevention Study Prospective Cohort Finland Sample Size: Baseline N: 4074 Analytic N: 3781 Attrition: 93% Sex: 1646 boys (52%), 1496 (48%) girls Race/Ethnicity: not mentioned Atopic Disease Risk Status: 16% and 62% of parents had asthma and rhinitis respectively	<p>Intervention/Exposure: The child's diet was assessed by using age-specific dietary questionnaires at ages 3, 4, 6 and 12 mo and a follow-up "age at introduction of new foods" form for recording the age at introduction of complementary foods.</p> <p>Looked at: the number of complementary foods introduced at 3, 4, 6 and 12 mo of age. 4 categories of food diversity at each time point were defined based on the distribution of the data at each time point: at 3 mo, these were "no food item," "1-2 food items," and ">2 food items"; at 4 mo, these were "no food item," "1-2 food items," "3-4 food items," and ">4 food items"; at 6 mo, these were "0-4 food items," "5-6 food items," "7-8 food items," and ">8 food items"; at 12 mo, these were "0-7 food items," "8-9 food items," "10-11 food items," and ">11 food items" Food groups used as DD definition: cow's milk and formula (as a combined variable); potatoes; carrots; turnip; fruits and berries (as a combined variable); cereals (rye, wheat, oats and barley as a combined variable); other cereals (maize, rice, millet and buckwheat as a combined variable); meat; fish; egg; cabbage; spinach; and lettuce.</p> <p>Assessment Methods: Prospective Parent questionnaires</p> <p>Assessment of DD age: birth to 12 mo of age</p>	<p>Asthma, wheeze, atopic eczema and allergic rhinitis were analysed.</p> <p>AD: parental reports of doctor-diagnosis ever up to 5 y. International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire for assessment of allergy symptoms</p> <p>No association between diet diversity at the age of 3, 4 mo and AD up to 5 y.</p> <p>Reduced diet diversity at 6 mo: increased risk of AD up to 5 y (OR and 95% CI: 0-4 food items vs > 8 items: 1.39, 0.88-2.19; 5-6 items vs > 8 items: 1.32, 0.96-1.81; 7-8 items vs > 8 items: 1.38, 1.11-1.71)</p>	<p>Confounders adjusted for: sex of the child, number of siblings, parental asthma, parental rhinitis, delivery hospital and maternal smoking during pregnancy.</p> <p>Limitations: The study recruited children carrying genetic risk (HLA-conferred susceptibility) for type 1 diabetes. Therefore, may reduce generalizability of findings to general population</p> <p>The number of statistical tests performed could mean that the type 1 error rate might not be at the 0.05 level.</p> <p>DD definition was not validated and could be improved with increasing the variety</p>
Roduit C et al JACI (2012) ⁸⁸ Prospective birth cohort study Protection Against Allergy Study in Rural Environments (PASTURE/EFRAIM). Stratified in farm and non-farm children Sample Size: Baseline N: 1133 Analytic N: 1041	<p>Diversity of diet in 1st year of life: summing the number of different types of solid food, which were introduced within the 1st year of life -> 2 scores were measured:</p> <ol style="list-style-type: none"> Diversity score (score 0-6) including food items, which were introduced among about 80% of the children, in total 6 groups: vegetables/fruits, cereals, bread, meat, cake and yogurt. Score used as a continuous variable and a categorized variable (0-3, 4-5, 6 food groups) Diversity score (score 0-15), including all food items: any cow's milk, yogurt, other milk products, eggs, nuts, vegetables or fruits, cereals, bread, meat, fish, soy, margarine, butter, cake and chocolate 	<p>AD: parental report of doctor-diagnosis up to 4 y and/or positive SCORAD score during medical examination at the age of 1y (total: 27.1% with AD up to 4 y).</p> <p>Increased diet diversity within 1st year of life: reduced risk of AD up to 4 y of age (exclusion children with onset within 1st year of life) (for each additional food introduced in 1st year, reduction of 25% of AD) (aOR for each additional food item: 0.75, 95% CI 0.62-0.91)</p>	<p>Outcome assessment mainly based on parental reports of doctor-diagnosis.</p> <p>To take into account reverse causality: analysis with exclusion of children with AD within the 1st year of age</p>

(Continues)

TABLE 4 (Continued)

Study and country	Definition and assessment of diet diversity	Results	Comments
Fergusson et al (1981) ⁸³ New Zealand Baseline N: 1265 Analytic N: 1156 Atopic Disease Risk Status: 24% parental history of atopy Background Diet: 19% exclusively breastfed at 4 mo	Diet diversity assess at 4 mo: Food groups used as DD definition: cereals, vegetables, dairy products, meat, fruits, egg or related products, other solid foods Food groups categorized as: 0, 1-2, 3-4, 5+ Assessment methods: Parent interview and food diary	Atopic Dermatitis assessed at 2 y. Assessment Methods: Maternal report; some with physician follow-up Increased diet diversity at 4 mo was associated with increased eczema at 2 y (0 food groups: 13%; 1-2 food groups: 17%; 3-4 food groups: 17%; 5+ food groups: 33%; P < .05) Increased diet diversity at 4 mo was associated with an increased risk for AD at 2 y	Adjusted for: Parental history of allergic disease Limitations: No info on similarity of groups at baseline. No blinded/validated assessments of outcome. Key confounders not taken into account (education, SES, sex, race/ethnicity, feeding practices, birth size, gestational age, smoking, pets) Timing of DD assessment (most children don't eat by 4 mo)
Fergusson et al <i>J Epidemiol Community Health</i> (1982) ⁸⁴ New Zealand Birth cohort of New Zealand N = 1143	Diet diversity assess at 4 mo: DD definition: sum of food groups (cereals, vegetables, dairy products, meat, fruits, egg or related products), which the child had been given during the first 4 mo. Categorized as follows: 0, 1-3, 4+ food groups Assessment method: Parent interview and food diary	Atopic Dermatitis: maternal report or directly from the child's doctor information on doctor-diagnosis of AD, up to 3 y Increased diet diversity within the first 4 mo of age: increased risk of AD up to 3 y (proportion of AD by numbers of solid food: 0:18.4%; 1-3:20.6%; 4+: 24.3%)	Adjusted for parental AD and/or asthma, infant milk diet. Analyses stratified depending on infant milk diet (breastfeeding, bottle only or both)
Fergusson D et al <i>Pediatrics</i> (1990) ⁸⁵ New Zealand Birth cohort of New Zealand N = 1067	Diet diversity assess at 4 mo: DD definition: sum of food groups (cereals, vegetables, dairy products, meat, fruits, egg or related products), which the child had been given during the first 4 mo. Categorized as follows: 0, 1-3, 4+ food groups Assessment method: Parent interview and food diary	Atopic Dermatitis: maternal report on doctor-diagnosis of AD, up to 10 y of age (31% at least once report of medical attendance for AD). Definition of a subgroup with recurrent or chronic AD (at least 3 medical visit for AD, condition has lasted at least 3 consecutive years and regular treatment for AD) (7.5%) Increased diet diversity within the first 4 mo of age: increased risk of recurrent/chronic AD up to 10 y (2.9 times increased risk: 4+ food groups versus no foods)	Adjusted for age at onset of AD, parental and siblings history of allergy, SES, maternal education, age of mother, ethnic status, breastfeeding
Fergusson D et al <i>PAI</i> (1994) ⁸⁶ New Zealand Birth cohort of New Zealand N = 1141 (data on AD up to 2 y) N = 1067	Diet diversity assess at 4 mo: DD definition: sum of food groups (cereals, vegetables, dairy products, meat, fruits, egg or related products), which the child had been given during the first 4 mo. Categorized as follows: 0, 1-3, 4+ food groups	Atopic Dermatitis: maternal report or directly from the child's doctor information on doctor-diagnosis of AD, up to 2 and 10 y of age Increased diet diversity within the first 4 mo of age: increased risk of AD up to 2 y (1.6 times increased risk: 4+ food groups versus no foods), increased risk of recurrent/chronic AD up to 10 y (2.5 times increased risk: 4+ food groups versus no foods)	Analyses adjusted for child's milk diet (breastfeeding vs bottle), parental history of AD or asthma, maternal education

2.4.5 | Food patterns during infancy and allergy outcomes in the infant

In children, all studies focused on *current* intake vs. allergy outcomes, which does not give an indication of early intake (infancy) vs. later (childhood) outcomes. These studies therefore are not designed to inform us regarding the role of the Mediterranean diet as a proxy measure of diet quality in infancy on allergy outcomes in childhood. Nonetheless, an overview is provided of the Mediterranean diet pattern in childhood and its influence on current allergy outcomes. The majority of childhood studies have used either the KIDMED Mediterranean score^{68,90-95} or the adult EPIC score.^{69,96-99} While the KIDMED index reflects what is commonly interpreted as a “healthy and diverse” diet, many Mediterranean diet studies use an index which was developed in a time when saturated fatty acids were believed to increase the risk of cardiovascular disease. Thus, dietary intake is categorized into “pro-Mediterranean diet” (fruit, vegetables, fish, cereals, pasta, rice and potatoes) and “anti-Mediterranean diet” (milk, meat, fast foods). There is, however, growing evidence that the “pro-” and “con-” assignment is not only out of date, but that it is not evidence-based, and some of the “anti-Mediterranean diet” foods were actually found to decrease the risk of wheeze/asthma (eg meat^{66,91,100,101} and milk^{67,97,101}).

2.4.6 | Mediterranean diet in infancy and childhood

While this is an important relationship to understand, no studies have been identified exploring an association between the Mediterranean diet in infancy and allergy outcomes. Four systematic reviews focussing on nutrients and foods associated with asthma and allergy outcomes investigated the impact of the Mediterranean diet in childhood¹⁰¹⁻¹⁰⁴ and found an inverse relationship between eating according to the Mediterranean diet and a range of reported symptoms such as wheeze and asthma. Papamichael et al¹⁰³ summarized twelve studies reporting an inverse association between adherence to a Mediterranean dietary pattern and asthma in children. Nurmatov et al¹⁰² reported that adherence to a Mediterranean diet was protective for persistent wheeze (OR, 0.22; 95% CI, 0.08-0.58) and atopy (OR, 0.55; 95% CI, 0.31-0.97). Garcia-Marcos et al¹⁰¹ found a significant negative association between the highest tertile of Mediterranean diet score (OR 0.85, 95% CI 0.75-0.98; $P = .02$) and “current wheeze.” Lv et al¹⁰⁴ concluded that the Mediterranean diet in children may be associated with prevention of asthma or wheeze, but that randomized controlled trials are required.

Ten original papers were also identified (Table S5) from cross-sectional studies,^{66,68,90-92,95,97,98,105,106} three papers from two (birth) cohorts from Mediterranean regions^{68,94,107}; five cross-sectional studies from non-Mediterranean regions^{69,99,108-110} and one paper on using the International Study on Asthma and Allergy in Children (ISAAC) data from 29 centres in 20 countries.¹⁰⁰ These publications focused on a range of allergy outcomes: most of the studies investigated the impact of the Mediterranean diet on risk of wheeze/asthmatic symptoms,^{68,69,90-96,99-101,106-108,110} sometimes including the development

of other allergic outcomes. Only a few studies focussed on atopic dermatitis,⁹⁷ and allergic rhinitis.^{94,98,105} However, as with the maternal studies, none of the studies focused on food allergy as an outcome.

The majority of studies used FFQs completed by parents^{69,90,93,95,97} and semi-quantitative questionnaires^{68,91,94,98,99} to calculate either the KIDMED index⁹⁰⁻⁹⁵ or another index to assess adherence to a Mediterranean diet.¹⁰⁶ The analysis of the ISAAC study¹⁰⁰ assessed adherence to a Mediterranean type diet using their own food categorization. Selected food items with either positive (+) or negative scoring (-) included were meat (-), fish (+), fresh fruit (+), raw green vegs (+), cooked green vegs (+), burgers (-), fruit juice (+) and fizzy drinks (-).

Adherence to the Mediterranean diet was primarily evaluated by using two different indices. While the KIDMED index, used in six studies,⁹⁰⁻⁹⁵ reflects what is commonly interpreted as a “healthy and diverse” diet, the other Mediterranean diet studies used the EPIC index^{69,96-99} (developed by Psaltopoulou/Trichopoulou^{61,111}). The EPIC score allowed a total of 10 points from incorporation of the following foods into the diet: vegetables, legumes, fruit, dairy products, cereals, meat and meat products, fish and seafood, olive oil, monounsaturated: saturated lipids, and alcohol intake.

Summarizing the available data on the association between the Mediterranean diet and childhood allergy outcomes is difficult, primarily due to different definitions of the Mediterranean diet and allergy outcomes assessed. Some studies showed a small but positive effect on current severe asthma in girls¹⁰⁵ and a protective effect on asthma/wheeze.^{69,96,100,110} Alternatively, some studies showed no protective effect on asthma and/or rhinitis symptoms^{98,99,108,109} or atopic dermatitis.⁹⁷ None of the studies reported on food allergy as an outcome.

2.4.7 | Other dietary patterns in infants and childhood allergy outcomes

Dietary patterns other than the Mediterranean diet were also studied in children. Three birth cohorts were identified,¹¹²⁻¹¹⁵ two cross-sectional in design^{116,117} and three papers on a case-control study within a cohort.^{114,118,119} Some of the identified publications on the Mediterranean diet also studied other dietary patterns.^{91-94,97-99} Dietary intake was assessed using FFQs,^{73,116,117} 24 hour recalls,¹¹³ semi-quantitative questionnaires¹¹² and prospective food diaries.^{114,118,119} (Table S5).

Two studies showed that food intake relating to a Western dietary pattern was associated with an increased prevalence of wheeze/asthma.^{116,117} A study from Singapore identified the pattern “noodles and seafood” as protective against the development of allergen sensation in Asian infants in the second half of the first year of life.¹¹⁵ Grimshaw et al^{114,118,119} found that a diet pattern obtained from principal component analysis on prospective food diary data described as “predominantly home cooked” (fruit, vegetable, fish and poultry consumption) in UK infants was associated with a reduced prevalence of food allergy. In contrast to other studies, analysis of the dietary data of the Dutch Generation R cohort did not find that a

TABLE 5 The association between diet diversity and asthma/allergic rhinitis

<p>Roduit et al (2014)¹⁶ Protection Against Allergy Study in Rural Environments (PASTURE/EFRAIM) Prospective Cohort Study Austria, Finland, France, Germany, Switzerland Sample Size: Baseline N: 1133 Analytic N: 848 (asthma) 806 (rhinitis) Attrition: 25% asthma 29% rhinitis Sample Size Calculation: NR Sex: 49.5% female Race/Ethnicity: NR Atopic Disease Risk Status: 53.6% parental history of atopy Background Diet: 47.4% nonexclusively breastfed at least 6 mo</p>	<p>Asthma/Allergic Rhinitis assessed at 6 y Assessment Methods: Questionnaires were administered in interviews or self-administered to the mothers within the third trimester of pregnancy and when the children were 2,12, 18 and 24 mo of age and then yearly up to age 6 y. Findings: <i>Asthma</i>: Increased diet diversity in first year of life was associated with linear trend in protection against development of reported asthma, 26% reduction for the introduction of each successive food. <i>Allergic Rhinitis</i>: No significant relationship noted between diversity and the development of allergic rhinitis (linear trend $P = .31$, $P = .29$ for inhalant sensitization)</p>	<p>Confounders: Confounders adjusted for: Centre, living on a farm, atopic Fhx, breastfeeding, gender, siblings, maternal education Limitations: Use of reported doctor-diagnosis and possible lack of assessment for resolved transient childhood wheezing Use of asthma medications was not part of the asthma definition While analysis was adjusted for confounders, were these the optimal ones to choose Use of 1-month recall period in assessing new food introduction</p>
Intervention/Exposure: See table—Food Allergy		

healthy dietary pattern in early life is associated with a lower risk of allergic sensitization or atopic diseases in childhood.¹¹²

2.4.8 | Key measurement issues to address when designing studies assessing the association between diet diversity and allergy outcomes

Research into diet diversity should have three particular prerequisites; (1) The method used needs to be specific to the outcome which may include nutritional intake, growth or health outcomes; (2) The diet diversity tool needs to be able to measure food security and socio-economic status.^{120,121} This needs further clarification

as well as careful statistical guidance to disentangle the impact of socio-economic status vs. diet diversity on outcomes; and (3) Consideration should be given to healthy diet diversity vs. unhealthy diet diversity when studying disease outcomes to inform diet recommendations. Researchers must consider that diversity in diets is also strongly related to local and ethnic traditions, with regional environmental exposures leading to unmeasured/unmeasurable characteristics which could potentially impact on short-, medium- and long-term outcomes. These considerations will apply to the most studied model of diet intake, that is, the Mediterranean diet, which partly explains some “caveats” within previous paragraphs.

Allergy outcome	Increased risk with higher DD	Reduced risk with higher DD	No effect
Sensitization		+ (4.5 y) [food] ¹⁶ + (6 y) [food] ¹⁶ + (up to 15 yrs) [aero-allergens] ⁷⁸ +(year 5) ⁷⁹	+ (4.5 y) [inhalant] ¹⁶ + (6 y) [inhalant] ¹⁶
Food Allergy		+ (up to 6 y) ¹⁶	
Atopic Dermatitis	+ (2 y) ⁸⁰ + (6 y) ⁸⁰ + (2 y) ⁸³ + (3 y) ⁸⁴ + (10 y) ^{85,86,155}	+ (2 y) ^{81,82} + (1 y) ⁸⁷ + (5 y) ⁸⁷ + (4 y) ⁸⁸ +(2 y) ⁸⁹	+ (4 y) ⁸⁰ + (6 y) ^{81,82}
Asthma/Wheeze		+ (6 y) ¹⁶ + (5 y) ⁸⁷	
Rhinitis		+ (5 y) ⁸⁷	+(6 y) ¹⁶

TABLE 6 Summary of diet diversity on Allergy Outcomes

TABLE 7 Modified delphi panel regarding diet diversity statements

Statement	Agree	Disagree	Per cent Agreement between those who voted	Comments
1. Diet diversity scores should be weighted depending on the types of food being measured (eg a diverse fast-food diet is not equal to a diverse fruit and vegetable diet).	23	1	95.8%	
2. Diversity of intake of specific foods, for example range of fruits eaten, different types of cheese consumed, different types of home-cooked or processed foods cannot be used to describe diet diversity but the term diversity of fruit intake, diversity of cheese intake, or diversity of processed food intake can be used. These foods can however be included in combination with other foods to determine overall diet diversity.	22	2	91.67%	<ol style="list-style-type: none"> 1. I would suggest separating the first sentence for clarity. Anyhow, I agree with this statement. 2. The diversity of fruits and vegetables is important to quantify and acknowledge in the overall definition of diet diversity, the diversity of types of cheese and other food groups is less so. 3. Need a better explanation.
3. Diversity of intake of foods containing specific nutrients or ingredients, for example foods containing advanced glycation end products or foods containing omega-3 fatty acids cannot be used to describe diet diversity but the terms diversity of advanced glycation end products intake or diversity of food containing omega-3 fatty acid intake can be used. These foods can however be included in combination with other foods to determine overall diet diversity. Following comments we have changed the wording to: In these instances, diversity may have a positive (fruit and vegetables) or a negative) (advanced glycation end products) connotation. This list may continue to grow as we gain more knowledge about foods/nutrients with immunomodulatory potential, for example bovine trans fatty acids which are isomers of linoleic acid.	21	3	87.5%	<ol style="list-style-type: none"> 1. We do not have any data to suggest that diversity of high AGE foods has a positive effect on dietary diversity. 2. There is a need for high level of knowledge and intellectual capacity to appropriately understand and answer this question. I do not think there is many people who can talk about diversity of advanced glycation end products.
4. For allergy (asthma, food allergy, eczema, rhinitis, allergen sensitivity), there is the opportunity to measure food microbial diversity which relates to the microbial content of foods. Following comments we have changed the wording to: There is the opportunity to measure food microbial diversity which relates to the microbial content of foods.	20	3	87%	<p>The formulation of the question is unclear as to what is the relation to "For allergy" (to determine the sensitization capacity of the food? To use the food as microbiome-modulating agent? To treat allergy with microbes on the food?)</p> <p>It is unclear what "For allergy" means here. Also, the whole question is unclear. Is it a general question what we should recommend to pursue more care. Is it a general question what we should recommend to pursue more in the future?</p> <p>I would assume that this can be extended to other food related clinical entities, not only allergies.</p>
5. For allergy (asthma, food allergy, eczema, rhinitis, allergen sensitivity), there is the opportunity to measure food biodiversity which relates to the taxonomic classification of food intake, for example classifying tomatoes as Solanum (genus) opposed to vegetables. Following comments we have changed the wording to: There is the opportunity to measure food biodiversity which relates to the taxonomic classification of food intake, for example classifying tomatoes as Solanum (genus) opposed to vegetables.	21	1	95.5%	<ol style="list-style-type: none"> 1. This is still a magic area surrounded by echo chambers even among experts. 2. Solanum—Wow—you picked a good one here. Tomatoes are a fruit, and other members of the genus include potato—vegetable and aubergine—fruit. I believe that in the US data potato (fries) and tomato (sauce) account for about 40% of fruit and vegetable intake in children. 3. why would this be only true for allergy? what would be the advantage over diversity of vegs? 4. It is unclear what "For allergy" means here. Please see my comment above.

(Continues)

TABLE 7 (Continued)

Statement	Agree	Disagree	Per cent Agreement between those who voted	Comments
6. For allergy (asthma, food allergy, eczema, rhinitis, allergen sensitivity), there is the opportunity to measure food allergen diversity which relates to the number and/or amount of food allergens introduced in a given period of time, for example the first year of life. It is important to understand that allergic food diversity may not reflect diet diversity as an emphasis on allergic food intake, may lead to an overall reduced diet diversity. Following comments we have changed the wording to: For allergy (asthma, food allergy, eczema, rhinitis, allergen sensitivity), there is the opportunity to measure allergenic food diversity which relates to the number and/or amount of food allergens introduced in a given period of time, for example the first year of life. It is important to understand that allergenic food diversity may not reflect diet diversity as an emphasis on allergenic food intake, may lead to an overall reduced diet diversity.	22	0	100%	<p>1. This point is not clear. precisely, food allergen means are h 1, cor a 9 etc what you mean is foods which a lot of children are allergic to. there is hardly a food which cannot elicit allergic reactions.</p> <p>2. Please consider also contact with food allergens other than by the oral route- this can be exposure by inhalation or via skin—so the measurement of allergen intake maybe not sufficient if control of exposure is the question.</p>
7. It is not possible at present to define the minimum amount of allergenic protein that should be consumed in order to be sufficient within the food allergen diversity measure. Following comments we have changed the wording to: It is not possible at present to define the minimum amount of allergenic protein that should be consumed in order to be sufficient within the allergenic food diversity measure. In future, there may be an index that could described the allergenic potential of foods within the context of diet diversity better.	21	2	91.3%	<p>1. Basically agreed, maybe in a short time we will be able to develop a sort of "index" specific by any specific protein, that, in combination with the food allergen diversity measure, could give a more precise idea of the allergenic potential of that protein with in a measure of an allergen diversity patterns. A sort of "diet allergen index" conceptually similar to the glyceimic index and again similarly, with two definitions as "allergenic index" and an "allergenic load". Finally (I have a dream) this could lead to a "personalized allergenic diet" based on the individually defined numbers, on the way of the personalized diet depicted by Elinav and co. at the Weizman one doubts: has someone already described this pathway? Sufficient for allergy prevention?</p> <p>2. Agree—may be a question of present/absent or the amount and frequency and timing of that.</p> <p>3. I don't understand "to be sufficient within the food allergen diversity measure."</p>
8. When possible, studies reporting data of diet diversity should include sensitivity, specificity, true and false positive/negative values to assist future meta-analyses and comparisons but should de-emphasize reporting of a cut-off value for a sample.	20	1	95.2%	<p>1. Differentiating between sensitization and allergy would be more helpful</p> <p>2. Getting this into practice will depend on cut-off values that are clinically meaningful. So, need them. Paper would though present the basic data to allow later meta-analysis.</p> <p>3. Again, not very clear to me. There are two questions within one sentence. I agree with the first part.</p> <p>4. But I do not understand clearly the second part.</p> <p>5. Not sure how this can be done in reality during a study?</p> <p>6. Sensitivity, specificity, true and false positive/negative values are values used for validation studies. it is unclear what their purpose here would be and how they can be derived.</p>

(Continues)

TABLE 7 (Continued)

Statement	Agree	Disagree	Per cent Agreement between those who voted	Comments
9. For pregnant women we recommend a minimum recall period of 2 weekdays and 1 weekend day, measured at repeated intervals throughout pregnancy. When possible, portion sizes should be collected and reported.	20	2	90.9%	<ol style="list-style-type: none"> 1. Agree with the need to measure dietary exposure at more than one interval & record portion sizes, but not necessarily the use a recall period of 2 week days and 1 weekend day—this rules out the use of FFQs and 24 h recalls, which may be more practical methods than a food diary method. 2. Any standard used to define the 2-week period? If mothers can recall up to several months for the child (as question 2 below), then they can also give recall of their own diet over several months. Is there any evidence that 2 wks is an acceptable period of time?
10. For infants, we recommend food recall periods within the first year of life. These recall periods may include the first four months of life, the first six months of life or the first year of life. Repeated measure of intake is recommended to improve the quality of data, rather than just consuming a particular food/food group once. When possible, portion sizes should be collected and reported. Following comments we have changed the wording to: For infants, we recommend food recall periods within the first year of life. These periods may include the first four months of life, the first six months of life or the first year of life. Repeated measure of intake is recommended to improve the quality of data, rather than just consuming a particular food/food group once. When possible, portion sizes should be collected and reported, and data should be recorded prospectively.	21	2	91.3%	<ol style="list-style-type: none"> 1. "Vidiotape" techniques should be developed (either at an institution or at home—parents are not reliable). 2. Again, not sure how this can be provided by the participants for practical reasons. 3. How far back can parents really remember. Certainly not a year.

11. Investigators should consider ways to ensure measuring how food is prepared/cooked in their study design, and if/when possible, this should be detailed for diet diversity. Ideally foods should be defined as raw, home-cooked, processed or ultra-processed.	22	1	95.7%	<ol style="list-style-type: none"> 1. Methodologies should be carefully defined and agreed, step by step. Caregivers (especially mothers) are extraordinarily able to mix different concentrations all these dines of preparations to get their final purpose. That infants eat somethings and/or anything whichever the source. 2. What about home-cooked foods that include some processed ingredients eg casserole containing canned chickpeas alongside other fresh vegetables. Perhaps a 4th category? 3. Water quality and milk quality are also important points to consider? 4. I agree with the first part of this statement, but not with the need for categorization. These categories are difficult as they are not mutually exclusive, a food could be processed AND home cooked (ie canned/frozen vegetables/fruit/fish used in a home cooked recipe). 5. I do not see a big difference between home cooked and processed since cooking per se is a way of processing foods.
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(Continues)

TABLE 7 (Continued)

Statement	Per cent Agreement between those who voted		Comments
	Agree	Disagree	
12. Age of introduction of solid foods, food allergens and infant formula should be clearly indicated in studies investigating the association between diet diversity and allergy outcomes. Following comments we have changed the wording to: Age of introduction of solid foods, allergenic foods and infant formula should be clearly indicated in studies investigating the association between diet diversity and allergy outcomes.	22	0	1. Difficult tasks for the same reasons explained before. Parents should not perceive these surveys as a sort of "drug-us" indication. 2. Agree but the term "food allergens" should be changed as commented before.
13. Introduction of infant formula cannot be classified on its own as diet diversity but should be included in measuring diet diversity of food and food groups. Following comments we have changed the wording to: Introduction of infant formula cannot be classified on its own as diet diversity but should be included in measuring diet diversity of food and food groups. Introduction of infant formula may in fact reduce diet diversity as breastmilk potentially exposes an infant to the diversity of the maternal diet, whereas formula is uniform.	23	0	1. This is difficult. Perhaps this statement needs a caveat? Using this definition, it would mean that an infant who is formula fed would be classified as having a more diverse diet than an infant who is exclusively breastfed? Breastmilk potentially exposes an infant to the diversity of the maternal diet, whereas formula is uniform.

^aThreshold for consensus was set at 18 votes in favour of the statement (75% of the 24 panel members).

2.4.9 | Delphi consensus

The EAACI task force agreed that the systematic review was unable to answer a number of key questions. Given the complexities and confusion/inconsistencies of the concept and terminology in the existing literature for diet diversity, the organizing members of this taskforce initiated methods to provide an expert consensus regarding multiple concepts, in order to provide a pathway forward for future research into diet diversity and allergic outcomes. In the absence of established clinical trials or observational research studies with agreed definitions and nomenclature, expert opinion obtained in this fashion becomes an acceptable alternative.^{28,122-124} The panel used the modified Delphi Method (written questionnaire was not used) technique to reach consensus on outstanding issues identified in these literature searches regarding diet diversity in relation to various allergic outcomes. A single-group, single-round method was chosen for developing consensus on all statements. Feedback was given on each statement. Questions were written by 4 committee members based on identified gaps in each subsection in this document and refined in an iterative manner among these individuals (CV, LM, RM, MG) until there was consensus, and then, the questions were formatted into an electronic survey software (RedCAP) and emailed to the group. Responses were gathered over a 2-week period of time, then tallied and discussed among the wider group. The tally was then either confirmed or revised based on panel member insights from the discussion. Consensus threshold was defined a priori as agreement on a given statement by 75% of the committee members. This threshold was chosen based on existing literature and agreed upon as an appropriate level by the experts. The questions, vote tally and final responses are summarized in Table 7, and the final recommendations noted in Table 1. Threshold for consensus was set at 18 votes in favour of the statement (75% of the 24 panel members).

3 | CONCLUSION

We have performed a systematic review particularly investigate the association between diet diversity and allergy outcomes in infancy and childhood. Currently, we suggest that diet diversity in infancy may be associated with reduced allergy outcomes, but additional studies are required to define more clearly the role of diet diversity and diet patterns, while clearly adjusting for appropriated confounders. There are no data on diet diversity in pregnancy and allergy outcomes in the offspring. Data on diet quality in pregnancy are not consistent, but usually indicate an inverse association with asthma/wheeze in the offspring. There are no data on diet quality in infancy, but studies in childhood, show a possible association with reduction in wheeze/asthma and perhaps used different definitions of diet diversity, and different instruments to measure diet diversity.

In line with the European Food Safety Authority (EFSA),¹²⁵ we endorse a " complementary approach to traditional monitoring

and surveillance programmes of dietary intake, which instead of focusing on compliance is designed to provide a solid basis for calculating population dietary exposure and assessing potential impact on public health. Harmonizing the total diet study methodology, focused on specifically allergy outcomes, will enhance the value of these programmes by improving the comparability at international level.”

CONFLICT OF INTEREST

Dr Nwaru, Dr Maslin, Remo Frei, Dr Pali-Schöll, Dr Garn, Dr Untersmayr, Dr Lunjani, Dr Sokolowska, Dr Agache, Dr Renz, Dr Feeney, Dr Stanton, Dr Smith, Dr Roberts, Dr Ben-Abdallah, Dr Muraro, Dr Agostoni, Dr Roduit and Dr Poulsen have nothing to disclose. Dr O'Mahony reports personal fees from Alimentary Health, grants from GSK, outside the submitted work. Dr Hoffmann-Sommergruber reports other from EAACI, during the conduct of the study. Dr Meyer reports grants from Danone, personal fees from Nutricia, personal fees from Mead Johnson and personal fees from Nestle, during the conduct of the study. Dr Grimshaw reports other from Nutricia Speakers fees, other from Abbott Speakers fees, other from Mead Johnson, outside the submitted work. Dr Reese reports personal fees from Nestlé Deutschland AG, personal fees from Nutricia GmbH, personal fees from Sanomega GmbH, personal fees from DAAB e.V., personal fees from Schweizer Milchproduzenten SMP, personal fees from Bofrost GmbH, personal fees from Milchwirtschaftlicher Verein BW, personal fees from Hans Karrer GmbH, personal fees from GMF GmbH, outside the submitted work. Dr Vlieg-Boerstra reports grants from Nutricia Early Life Nutrition, during the conduct of the study; personal fees from for Marfo Food Group, Lelystad, The Netherlands (Producer of commercially prepared test materials for Oral Food Challenge tests), personal fees from Nutricia, personal fees from Mead Johnson, grants from Nutricia Research, personal fees from Thermofisher, outside the submitted work;. Dr Bischoff reports personal fees from Hexal, personal fees from Nestle, personal fees from Wild Foundation, personal fees from SymbioPharm, other from Ardeypharm, personal fees from Cemet, personal fees from Pfizer, personal fees from Janssen, personal fees from ZKES, outside the submitted work. Dr Venter reports personal fees from Danone, personal fees from Nestle, personal fees from Mead Johnson, personal fees from Abbott, personal fees from DBV technologies, personal fees from Lil Mixins, outside the submitted work. Dr Akdis reports grants from Allergopharma, Idorsia, Swiss National Science Foundation, Christine Kühne-Center for Allergy Research and Education, European Commission's Horizon's 2020 Framework Programme, Cure, other from Sanofi-Aventis_Regeneron, grants from Novartis Research Institutes, Astra Zeneca, Scibase, outside the submitted work. Dr Du Toit reports grants from MRC & Asthma UK Centre, grants from UK Dept of Health through NIHR, grants from Action Medical Research, grants from National Peanut Board, personal fees from Scientific Advisory Board member Aimmune., outside the submitted work..

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AUTHOR CONTRIBUTIONS

LOM, CV, CA and MG wrote the introduction and the section; CV wrote the methods and the overview section on diet diversity (Section 1) with support of MBA; KM wrote the overview section on diet quality (Section 2); BV wrote the section on maternal diet patterns/quality in pregnancy and allergy outcomes (Section 3); RM, GdT and MF wrote the section on allergy sensitization and diet diversity (Section 3); CR and BN wrote the section on food allergy and diet diversity (Section 3); EU and IP wrote the section on atopic dermatitis and diet diversity (Section 3); MG and GR wrote the section on allergic rhinitis and asthma and diet diversity (Section 3); IR wrote the section on diet patterns/quality in infancy and childhood (Section 3); CV and CAg wrote the section on key measurement issues (Section 4); MG and CV wrote the section on Delphi consensus; and LOM, CV, CAg and RM wrote the conclusion. CV, RM, MBA, LOM and MG developed and distributed the Delphi questionnaires, and MG analysed the Dephi consensus data. MG, CAg, RM, IR, PS and LOM reviewed the first draft of the paper. All authors actively reviewed and commented on the paper and Delphi questions and edited the final version of the paper.

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REFERENCES

- Ruel MT. Is dietary diversity an indicator of food security of dietary quality? A review of measurement issues and research needs. *Int Food Policy Res Inst.* 2003;1:58. Discussion paper. No. 140.
- Castro-Quezada I, Roman-Vinas B, Serra-Majem L. The Mediterranean diet and nutritional adequacy: a review. *Nutrients.* 2014;6:231-248.
- Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. *Curr Opin Lipidol.* 2002;13:3-9.
- Complementary feeding; 2018. http://www.who.int/nutrition/topics/complementary_feeding/en/
- Boyce JA, Assa'a A, Burks AW, et al. Guidelines for the diagnosis and management of food allergy in the United States: summary of the NIAID-Sponsored Expert Panel Report. *Nutrition.* 2011;27:253-267.
- Dietary Assessment primer. Food Frequency Questionnaire at a Glance; 2018. <https://dietassessmentprimer.cancer.gov/profiles/questionnaire/>
- Boucher B, Cotterchio M, Kreiger N, Nadalin V, Block T, Block G. Validity and reliability of the Block98 food-frequency questionnaire in a sample of Canadian women. *Public Health Nutr.* 2006;9:84-93.
- Institute NC. Assessment Primer: 24 hour recall at a glance; 2018.
- Gabriel da Silva LB, Rosado EL, de Carvalho PP, et al. Food intake of women with gestational diabetes mellitus, in accordance with two methods of dietary guidance: a randomised controlled clinical trial. *Br J Nutr.* 2018; 121:1-11.
- Bisgaard H, Stokholm J, Chawes BL, et al. Fish oil-derived fatty acids in pregnancy and wheeze and asthma in offspring. *N Engl J Med.* 2016;375:2530-2539.
- Baraldi E, Galderisi A. Fish oil in pregnancy and asthma in offspring. *N Engl J Med.* 2017;376:1191.
- Garcia-Larsen V, Del Giacco SR, Moreira A, et al. Asthma and dietary intake: an overview of systematic reviews. *Allergy.* 2016;71:433-442.
- Ahluwalia N, Andreeva VA, Kesse-Guyot E, Hercberg S. Dietary patterns, inflammation and the metabolic syndrome. *Diabet Metabol.* 2013;39:99-110.
- Asarat M, Apostolopoulos V, Vasiljevic T, Donkor O. Short-chain fatty acids produced by synbiotic mixtures in skim milk differentially regulate proliferation and cytokine production in peripheral blood mononuclear cells. *Int J Food Sci Nutr.* 2015;66:755-765.
- Wirth MD, Hebert JR, Shivappa N, et al. Anti-inflammatory Dietary Inflammatory Index scores are associated with healthier scores on other dietary indices. *Nut Res.* 2016;36:214-219.
- Roduit C, Frei R, Depner M, et al. Increased food diversity in the first year of life is inversely associated with allergic diseases. *J Allergy Clin Immunol.* 2014;133:1056-1064.
- Claesson MJ, Jeffery IB, Conde S, et al. Gut microbiota composition correlates with diet and health in the elderly. *Nature.* 2012;488:178-184.
- Savage JH, Lee-Sarwar KA, Sordillo JE, et al. Diet during Pregnancy and Infancy and the Infant Intestinal Microbiome. *J Pediatr.* 2018; 203: 47-54.
- Bisgaard H, Li N, Bonnelykke K, et al. Reduced diversity of the intestinal microbiota during infancy is associated with increased risk of allergic disease at school age. *J Allergy Clin Immunol.* 2011;128:646-652.
- Simonyte Sjodin K, Hammarstrom ML, Ryden P, et al. Temporal and long-term gut microbiota variation in allergic disease: A prospective study from infancy to school age. *Allergy.* 2018; 74:176-185.
- Venter C, Brown KR, Maslin K, Palmer DJ. Maternal dietary intake in pregnancy and lactation and allergic disease outcomes in offspring. *Pediatr Allergy Immunol.* 2016; 28:135-143.
- Garcia-Larsen V, Ierodiakonou D, Jarrold K, et al. Diet during pregnancy and infancy and risk of allergic or autoimmune disease: A systematic review and meta-analysis. *PLoS Med.* 2018;15:e1002507.
- Matricardi PM. 99th Dahlem conference on infection, inflammation and chronic inflammatory disorders: controversial aspects of the 'hygiene hypothesis'. *Clin Exp Immunol.* 2010;160:98-105.
- Du Toit G, Katz Y, Sasieni P, et al. Early consumption of peanuts in infancy is associated with a low prevalence of peanut allergy. *J Allergy Clin Immunol.* 2008;122:984-991.
- Perkin MR, Logan K, Marris T, et al. Enquiring About Tolerance (EAT) study: Feasibility of an early allergenic food introduction regimen. *Journal of Allergy & Clinical Immunology* 2016;137:1477-1486.
- Lundell AC, Hesselmar B, Nordstrom I, Adlerberth I, Wold AE, Rudin A. Higher B-cell activating factor levels at birth are positively associated with maternal dairy farm exposure and negatively related to allergy development. *J Allergy Clin Immunol.* 2015;136:1074-1082.
- Du Toit G, Roberts G, Sayre PH, et al. Randomized trial of peanut consumption in infants at risk for peanut allergy. *N Engl J Med.* 2015;372:803-813.
- Caporali R, Carletto A, Conti F, et al. Using a modified Delphi process to establish clinical consensus for the diagnosis, risk assessment and abatacept treatment in patients with aggressive rheumatoid arthritis. *Clin Exp Rheumatol.* 2017;35:772-776.
- Kant AK, Block G, Schatzkin A, Ziegler RG, Nestle M. Dietary diversity in the US population, NHANES II, 1976-1980. *J Am Diet Assoc.* 1991;91:1526-1531.
- Hatloy A, Torheim LE, Oshaug A. Food variety—a good indicator of nutritional adequacy of the diet? A case study from an urban area in Mali, West Africa. *Eur J Clin Nutr.* 1998;52:891-898.
- Roche ML, Creed-Kanashiro HM, Tuesta I, Kuhnlein HV. Traditional food diversity predicts dietary quality for the Awajun in the Peruvian Amazon. *Public Health Nutr.* 2008;11: 457-465.
- Motbainor A, Worku A, Kumie A. Stunting is associated with food diversity while wasting with food insecurity among underfive children in east and west Gojjam zones of Amhara Region, Ethiopia. *PLoS ONE.* 2015;10:e0133542.
- Hatloy A, Hallund J, Diarra MM, Oshaug A. Food variety, socio-economic status and nutritional status in urban and rural areas in Koutiala (Mali). *Public Health Nutr.* 2000;3:57-65.
- Chandrasekhar S, Aguayo VM, Krishna V, Nair R. Household food insecurity and children's dietary diversity and nutrition in India. Evidence from the comprehensive nutrition survey in Maharashtra. *Maternal Child Nutr.* 2017;13:1-8.
- Wright MJ, Bentley ME, Mendez MA, Adair LS. The interactive association of dietary diversity scores and breast-feeding status with weight and length in Filipino infants aged 6–24 months. *Public Health Nutr* 2015;18:1762-1773.
- Ey Chua EY, Zalilah MS, Ys Chin YS, Norhasmah S. Dietary diversity is associated with nutritional status of Orang Asli children in Krau Wildlife Reserve, Pahang. *Malaysian J Nutr.* 2012;18:1-13.
- Onyango A, Koski KG, Tucker KL. Food diversity versus breast-feeding choice in determining anthropometric status in rural Kenyan toddlers. *Int J Epidemiol.* 1998;27:484-489.
- Shamim AA, Mashreky SR, Ferdous T, et al. Pregnant women diet quality and its sociodemographic determinants in Southwestern Bangladesh. *Food Nutr Bull.* 2016;37:14-26.

39. Christian AK, Marquis GS, Colecraft EK, et al. Caregivers' nutrition knowledge and attitudes are associated with household food diversity and children's animal source food intake across different agro-ecological zones in Ghana. *Br J Nutr.* 2016;115:351-360.
40. Vadiveloo M, Dixon LB, Mijanovich T, Elbel B, Parekh N. Development and evaluation of the US Healthy Food Diversity index. *Br J Nutr.* 2014;112:1562-1574.
41. Mok E, Vanstone CA, Gallo S, Li P, Constantin E, Weiler HA. Diet diversity, growth and adiposity in healthy breastfed infants fed homemade complementary foods. *Int J Obes.* 2017;41:776-782.
42. Bezerra IN, Sichieri R. Household food diversity and nutritional status among adults in Brazil. *Int J Behav Nutr Phys Act.* 2011;8:22.
43. Krebs-Smith SM, Smiciklas-Wright H, Guthrie HA, Krebs-Smith J. The effects of variety in food choices on dietary quality. *J Am Diet Assoc.* 1987;87:897-903.
44. Lachat C, Raneri JE, Smith KW, et al. Dietary species richness as a measure of food biodiversity and nutritional quality of diets. *Proc Nat Acad Sci USA.* 2018;115:127-132.
45. Remans R, Flynn DF, DeClerck F, et al. Assessing nutritional diversity of cropping systems in African villages. *PLoS ONE.* 2011;6:e21235.
46. Ntwenya JE, Kinabo J, Msuya J, et al. Rich food biodiversity amid low consumption of food items in Kilosa District, Tanzania. *Food Nutr Bull.* 2017;38:501-511.
47. Jones AD. On-farm crop species richness is associated with household diet diversity and quality in subsistence- and market-oriented farming households in Malawi. *J Nutr.* 2017;147:86-96.
48. Chomat AM, Solomons NW, Koski KG, Wren HM, Vossenaar M, Scott ME. Quantitative methodologies reveal a diversity of nutrition, infection/illness, and psychosocial stressors during pregnancy and lactation in rural mam-mayan mother-infant dyads from the western highlands of Guatemala. *Food Nutr Bull.* 2015;36:415-440.
49. Rukundo PM, Andreassen BA, Kikafunda J, Rukooko B, Oshaug A, Iversen PO. Household food insecurity and diet diversity after the major 2010 landslide disaster in Eastern Uganda: a cross-sectional survey. *Br J Nutr.* 2016;115:718-729.
50. Woo JG, Herbers PM, McMahon RJ, et al. Longitudinal development of infant complementary diet diversity in 3 international cohorts. *J Pediatr.* 2015;167:969-974.
51. Agize A, Jara D, Dejenu G. Level of knowledge and practice of mothers on minimum dietary diversity practices and associated factors for 6-23-month-old children in Adea Woreda, Oromia, Ethiopia. *BioMed Res Int.* 2017;2017:7204562.
52. Gewa CA, Murphy SP, Weiss RE, Neumann CG. Determining minimum food intake amounts for diet diversity scores to maximize associations with nutrient adequacy: an analysis of schoolchildren's diets in rural Kenya. *Public Health Nutr.* 2014;17:2667-2673.
53. Leroy JL, Razak AA, Habicht JP. Only children of the head of household benefit from increased household food diversity in northern Ghana. *J Nutr.* 2008;138:2258-2263.
54. Msaki MM, Hendriks SL. Do food quality and food quantity talk the same? Lesson from household food security study in Embo, South Africa. *J Am College Nutr.* 2013;32:165-176.
55. Conklin AI, Monsivais P, Khaw KT, Wareham NJ, Forouhi NG. Dietary Diversity, Diet Cost, and Incidence of Type 2 Diabetes in the United Kingdom: A Prospective Cohort Study. [Erratum appears in PLoS Med. 2016 Aug;13(8):e1002123; PMID: 27541996]. *PLoS Med.* 2016;13:e1002085.
56. Isa F, Xie LP, Hu Z, et al. Dietary consumption and diet diversity and risk of developing bladder cancer: results from the South and East China case-control study. *Cancer Cause Control.* 2013;24:885-895.
57. Torheim LE, Barikmo I, Parr CL, Hatloy A, Ouattara F, Oshaug A. Validation of food variety as an indicator of diet quality assessed with a food frequency questionnaire for Western Mali. *Eur J Clin Nutr.* 2003;57:1283-1291.
58. Haines PS, Siega-Riz AM, Popkin BM. The Diet Quality Index revised: a measurement instrument for populations. *J Am Diet Assoc.* 1999;99:697-704.
59. Arvaniti F, Panagiotakos DB. Healthy indexes in public health practice and research: a review. *Crit Rev Food Sci Nutr.* 2008;48:317-327.
60. Kennedy ET, Ohls J, Carlson S, Fleming K. The healthy eating index: design and applications. *J Am Diet Assoc.* 1995;95:1103-1108.
61. Trichopoulou A, Kouris-Blazos A, Wahlqvist ML, et al. Diet and overall survival in elderly people. *BMJ.* 1995;311:1457-1460.
62. Monteiro HMC, de Mendonca DC, Sousa MSB, Amancio-Dos-Santos A. Physical exercise counteracts the increase in velocity of propagation of cortical spreading depression imposed by early over-nutrition in rats. *Nut Neurosci.* 2018;1-9. <https://doi.org/10.1080/1028415X.2018.1481180>
63. Puchau B, Zulet MA, de Echavarri AG, Hermsdorff HH, Martinez JA. Dietary total antioxidant capacity: a novel indicator of diet quality in healthy young adults. *J Am Coll Nutr.* 2009;28:648-656.
64. Cavicchia PP, Steck SE, Hurley TG, et al. A new dietary inflammatory index predicts interval changes in serum high-sensitivity C-reactive protein. *J Nutr.* 2009;139:2365-2372.
65. Kant AK. Indexes of overall diet quality: a review. *J Am Diet Assoc.* 1996;96:785-791.
66. Castro-Rodriguez JA, Ramirez-Hernandez M, Padilla O, Pacheco-Gonzalez RM, Perez-Fernandez V, Garcia-Marcos L. Effect of foods and Mediterranean diet during pregnancy and first years of life on wheezing, rhinitis and dermatitis in preschoolers. *Allergol Immunopathol.* 2016;44:400-409.
67. Chatzi L, Garcia R, Roumeliotaki T, et al. Mediterranean diet adherence during pregnancy and risk of wheeze and eczema in the first year of life: INMA (Spain) and RHEA (Greece) mother-child cohort studies. *Br J Nutr.* 2013;110:2058-2068.
68. Chatzi L, Torrent M, Romieu I, et al. Mediterranean diet in pregnancy is protective for wheeze and atopy in childhood. *Thorax.* 2008;63:507-513.
69. de Batlle J, Garcia-Aymerich J, Barraza-Villarreal A, Anto JM, Romieu I. Mediterranean diet is associated with reduced asthma and rhinitis in Mexican children. *Allergy.* 2008;63:1310-1316.
70. Ballmer-Weber BK, Fernandez-Rivas M, Beyer K, et al. How much is too much? Threshold dose distributions for 5 food allergens. *J Allergy Clin Immunol.* 2015;135:964-971.
71. Lange NE, Rifas-Shiman SL, Camargo CA Jr, Gold DR, Gillman MW, Litonjua AA. Maternal dietary pattern during pregnancy is not associated with recurrent wheeze in children. *J Allergy Clin Immunol.* 2010;126:250-255.
72. Moonesinghe H, Patil VK, Dean T, et al. Association between healthy eating in pregnancy and allergic status of the offspring in childhood. *Ann Allergy Asthma Immunol.* 2016;116:163-165.
73. Loo E, Ong L, Goh A, et al. Effect of maternal dietary patterns during pregnancy on self-reported allergic diseases in the first 3 years of life: results from the GUSTO study. *Int Archive Allergy Immunol.* 2017;173:105-113.
74. Miyake Y, Okubo H, Sasaki S, Tanaka K, Hirota Y. Maternal dietary patterns during pregnancy and risk of wheeze and eczema in Japanese infants aged 16-24 months: the Osaka Maternal and Child Health Study. *Pediatr Allergy Immunol.* 2011;22:734-741.
75. Shaheen SO, Northstone K, Newson RB, Emmett PM, Sherriff A, Henderson AJ. Dietary patterns in pregnancy and respiratory and atopic outcomes in childhood. *Thorax.* 2009;64:411-417.
76. Rifas-Shiman SL, Rich-Edwards JW, Kleinman KP, Oken E, Gillman MW. Dietary quality during pregnancy varies by maternal characteristics in Project Viva: a US cohort. *J Am Diet Assoc.* 2009;109:1004-1011.
77. Sausenthaler S, Koletzko S, Schaaf B, et al. Maternal diet during pregnancy in relation to eczema and allergic sensitization in the offspring at 2 y of age. *Am J Clin Nutr.* 2007;85:530-537.

78. Markevych I, Standl M, Lehmann I, von Berg A, Heinrich J. Food diversity during the first year of life and allergic diseases until 15 years. *J Allergy Clin Immunol*. 2017;140:1751-1754.
79. Nwaru BI, Takkinen HM, Niemela O, et al. Introduction of complementary foods in infancy and atopic sensitization at the age of 5 years: timing and food diversity in a Finnish birth cohort. *Allergy*. 2013;68:507-516.
80. Sausenthaler S, Heinrich J, Koletzko S, Ginipius G. Early diet and the risk of allergy: what can we learn from the prospective birth cohort studies GINIplus and LISAplus? *Am J Clin Nutr*. 2011;94:2012S-2017.
81. Zutavern A, Brockow I, Schaaf B, et al. Timing of solid food introduction in relation to atopic dermatitis and atopic sensitization: results from a prospective birth cohort study. *Pediatrics*. 2006;117:401-411.
82. Zutavern A, Brockow I, Schaaf B, et al. Timing of solid food introduction in relation to eczema, asthma, allergic rhinitis, and food and inhalant sensitization at the age of 6 years: results from the prospective birth cohort study LISA. *Pediatrics*. 2008;121:e44-52.
83. Fergusson DM, Horwood LJ, Beautrais AL, Shannon FT, Taylor B. Eczema and infant diet. *Clin Allergy*. 1981;11:325-331.
84. Fergusson DM, Horwood LJ, Shannon FT. Risk factors in childhood eczema. *J Epidemiol Community Health*. 1982;36:118-122.
85. Fergusson DM, Horwood LJ, Shannon FT. Early solid feeding and recurrent childhood eczema: a 10-year longitudinal study. *Pediatrics*. 1990;86:541-546.
86. Fergusson DM, Horwood LJ. Early solid food diet and eczema in childhood: a 10-year longitudinal study. *Pediatr Allergy Immunol*. 1994;5:44-47.
87. Nwaru BI, Takkinen HM, Kaila M, et al. Food diversity in infancy and the risk of childhood asthma and allergies. *J Allergy Clin Immunol*. 2014;133:1084-1091.
88. Roduit C, Frei R, Loss G, et al. Development of atopic dermatitis according to age of onset and association with early-life exposures. *J Allergy Clin Immunol*. 2012;130:130-136.
89. Turati F, Bertuccio P, Galeone C, et al. Early weaning is beneficial to prevent atopic dermatitis occurrence in young children. *Allergy*. 2016;71:878-888.
90. Alphanonogeorgos G, Panagiotakos DB, Grigoropoulou D, et al. Investigating the associations between Mediterranean diet, physical activity and living environment with childhood asthma using path analysis. *Endocr Metab Immune Disord Drug Targets*. 2014;14:226-233.
91. Arvaniti F, Priftis KN, Papadimitriou A, et al. Adherence to the Mediterranean type of diet is associated with lower prevalence of asthma symptoms, among 10-12 years old children: the PANACEA study. *Pediatr Allergy Immunol*. 2011;22:283-289.
92. Arvaniti F, Priftis KN, Papadimitriou A, et al. Salty-snack eating, television or video-game viewing, and asthma symptoms among 10- to 12-year-old children: the PANACEA study. *J Am Diet Assoc*. 2011;111:251-257.
93. Chatzi L, Apostolaki G, Bibakis I, et al. Protective effect of fruits, vegetables and the Mediterranean diet on asthma and allergies among children in Crete. *Thorax*. 2007;62:677-683.
94. Chatzi L, Torrent M, Romieu I, et al. Diet, wheeze, and atopy in school children in Menorca. *Spain. Pediatr Allergy Immunol*. 2007;18:480-485.
95. Grigoropoulou D, Priftis KN, Yannakoulia M, et al. Urban environment adherence to the Mediterranean diet and prevalence of asthma symptoms among 10- to 12-year-old children: The Physical Activity, Nutrition, and Allergies in Children Examined in Athens study. *Allergy Asthma Proc*. 2011;32:351-358.
96. Castro-Rodriguez JA, Garcia-Marcos L. What are the effects of a mediterranean diet on allergies and asthma in children? *Front Pediatr*. 2017;5:72.
97. Suarez-Varela MM, Alvarez LG, Kogan MD, et al. Diet and prevalence of atopic eczema in 6 to 7-year-old schoolchildren in Spain: ISAAC phase III. *J Investig Allergol Clin Immunol*. 2010;20:469-475.
98. Tamay Z, Akcay A, Ergin A, Guler N. Dietary habits and prevalence of allergic rhinitis in 6 to 7-year-old schoolchildren in Turkey. *Allergol Int*. 2014;63:553-562.
99. Akcay A, Tamay Z, Hocaoglu AB, Ergin A, Guler N. Risk factors affecting asthma prevalence in adolescents living in Istanbul, Turkey. *Allergol Immunopathol (Madr)*. 2014;42:449-458.
100. Nagel G, Weinmayr G, Kleiner A, Garcia-Marcos L, Strachan DP. Effect of diet on asthma and allergic sensitisation in the International Study on Allergies and Asthma in Childhood (ISAAC) Phase Two. *Thorax*. 2010;65:516-522.
101. Garcia-Marcos L, Robertson CF, Ross Anderson H, et al. Does migration affect asthma, rhinoconjunctivitis and eczema prevalence? Global findings from the international study of asthma and allergies in childhood. *Int J Epidemiol*. 2014;43:1846-1854.
102. Nurmatov U, Devereux G, Sheikh A. Nutrients and foods for the primary prevention of asthma and allergy: systematic review and meta-analysis. *J Allergy Clin Immunol*. 2011;127:724-733.
103. Papamichael MM, Itsiopoulos C, Susanto NH, Erbas B. Does adherence to the Mediterranean dietary pattern reduce asthma symptoms in children? A systematic review of observational studies. *Public Health Nutr*. 2017;20:2722-2734.
104. Lv N, Xiao L, Ma J. Dietary pattern and asthma: a systematic review and meta-analysis. *J Asthma Allergy*. 2014;7:105-121.
105. Garcia-Marcos L, Canflanca IM, Garrido JB, et al. Relationship of asthma and rhinoconjunctivitis with obesity, exercise and Mediterranean diet in Spanish schoolchildren. *Thorax*. 2007;62:503-508.
106. Gonzalez Barcala FJ, Pertega S, Bamonde L, et al. Mediterranean diet and asthma in Spanish schoolchildren. *Pediatr Allergy Immunol*. 2010;21:1021-1027.
107. Calatayud-Saez FM, Calatayud Moscoso Del Prado B, Gallego Fernandez-Pacheco JG, Gonzalez-Martin C, Alguacil Merino LF. Mediterranean diet and childhood asthma. *Allergol Immunopathol (Madr)*. 2016;44:99-105.
108. Romieu I, Barraza-Villarreal A, Escamilla-Nunez C, et al. Dietary intake, lung function and airway inflammation in Mexico City school children exposed to air pollutants. *Resp Res*. 2009;10:122.
109. Silveira DH, Zhang L, Prietsch SO, Vecchi AA, Susin LR. Association between dietary habits and asthma severity in children. *Indian Pediatr*. 2015;52:25-30.
110. Rice JL, Romero KM, Galvez Davila RM, et al. Association between adherence to the mediterranean diet and asthma in peruvian children. *Lung*. 2015;193:893-899.
111. Trichopoulou A, Martinez-Gonzalez MA, Tong TY, et al. Definitions and potential health benefits of the Mediterranean diet: views from experts around the world. *BMC Med*. 2014;12:112.
112. Tromp II, Kiefte-de Jong JC, de Vries JH, et al. Dietary patterns and respiratory symptoms in pre-school children: the Generation R Study. *Eur Respir J*. 2012;40:681-689.
113. Jonsson K, Green M, Barman M, et al. Diet in 1-year-old farm and control children and allergy development: results from the FARMFLORA birth cohort. *Food Nutr Res*. 2016;60:32721.
114. Grimshaw KE, Bryant T, Oliver EM, et al. Incidence and risk factors for food hypersensitivity in UK infants: results from a birth cohort study. *Clin Transl Allergy*. 2015;6:1.
115. Loo E, Sim J, Toh JY, et al. Relation of infant dietary patterns to allergic outcomes in early childhood. *Pediatr Allergy Immunol*. 2017;28:490-495.
116. Lee SC, Yang YH, Chuang SY, Liu SC, Yang HC, Pan WH. Risk of asthma associated with energy-dense but nutrient-poor dietary pattern in Taiwanese children. *Asia Pac J Clin Nutr*. 2012;21:73-81.
117. de Cassia Ribeiro Silva R, Assis AM, Cruz AA, et al. Dietary patterns and wheezing in the midst of nutritional transition: a study in Brazil. *Pediatr Allergy Immunol Pulmonol*. 2013;26:18-24.

118. Grimshaw KE, Maskell J, Oliver EM, et al. Diet and food allergy development during infancy: birth cohort study findings using prospective food diary data. *J Allergy Clin Immunol*. 2014;133:511-519.
119. Grimshaw KE, Maskell J, Oliver EM, et al. Introduction of complementary foods and the relationship to food allergy. *Pediatrics*. 2013;132:e1529-e1538.
120. Agostoni C, Silano M, Fattore G. Health implications of dietary habits in transition countries—a life course perspective. *Pediatr Res*. 2018;83:754-756.
121. Silano M, Agostoni C, Fattore G. Italy's unsolved childhood obesity crisis. *Arch Dis Child*. 2018, 104:202-203.
122. Ninane V, Corhay JL, Germonpre P, et al. Inhaled treatment of COPD: a Delphi consensus statement. *Int J Chron Obstruct Pulmon Dis*. 2017;12:793-801.
123. Sudore RL, Lum HD, You JJ, et al. Defining advance care planning for adults: a consensus definition from a multidisciplinary delphi panel. *J Pain Symptom Manage*. 2017;53:821-832.
124. Baumann MH, Strange C, Heffner JE, et al. Management of spontaneous pneumothorax: an American College of Chest Physicians Delphi consensus statement. *Chest*. 2001;119:590-602.
125. European Food Safety Authority FaAOotUN, World Health Organization. Towards a harmonised Total Diet Study approach: a guidance document. *EFSA J*. 2011;119:2450.
126. 2016 FaF.Dietary Diversity for Women: A Guide to Measurement. Nutrition and Consumer Protection Division, Food and Agriculture Organization of the United Nations and USAID's Food and Nutrition Technical Assistance III Project (FANTA), Managed by FHI 360; 2016.
127. Ogle BM, Hung PH, Tuyet HT. Significance of wild vegetables in micronutrient intakes of women in Vietnam: an analysis of food variety. *Asia Pac J Clin Nutr*. 2001;10:21-30.
128. Franceschi S, Favero A, La Vecchia C, et al. Influence of food groups and food diversity on breast cancer risk in Italy. *Int J Cancer*. 1995;63:785-789.
129. Elbert NJ, Duijts L, den Dekker HT, et al. Maternal psychiatric symptoms during pregnancy and risk of childhood atopic diseases. *Clin Exp Allergy*. 2017;47:509-519.
130. Perkin MR, Strachan DP. Which aspects of the farming lifestyle explain the inverse association with childhood allergy? *J Allergy Clin Immunol*. 2006;117:1374-1381.
131. Nicklaus S, Divaret-Chauveau A, Chardon ML, et al. The protective effect of cheese consumption at 18 months on allergic diseases in the first 6 years. *Allergy*. 2018, 74:788-798.
132. Smith PK, Masilamani M, Li X, Sampson HA. "The False Alarm" hypothesis: Food allergy is associated with high dietary advanced glycation end products and pro-glycating dietary sugars. *J Allergy Clin Immunol*. 2016. <https://doi.org/10.1016/j.jaci.2016.05.040>
133. DeChristopher LR, Uribarri J, Tucker KL. The link between soda intake and asthma: science points to the high-fructose corn syrup, not the preservatives: a commentary. *Nutr Diabetes*. 2016;6:1-4.
134. DeChristopher LR, Uribarri J, Tucker KL. Intakes of apple juice, fruit drinks and soda are associated with prevalent asthma in US children aged 2–9 years. *Public Health Nutr*. 2016;19:123-130.
135. Li Z, Rava M, Bedard A, et al. Cured meat intake is associated with worsening asthma symptoms. *Thorax*. 2017;72:206-212.
136. Ellwood P, Asher MI, Garcia-Marcos L, et al. Do fast foods cause asthma, rhinoconjunctivitis and eczema? Global findings from the International Study of Asthma and Allergies in Childhood (ISAAC) phase three. *Thorax*. 2013;68:351-360.
137. Willers SM, Wijga AH, Brunekreef B, et al. Maternal food consumption during pregnancy and the longitudinal development of childhood asthma. *Am J Respir Crit Care Med*. 2008;178:124-131.
138. Miyake Y, Sasaki S, Tanaka K, Hirota Y. Dairy food, calcium and vitamin D intake in pregnancy, and wheeze and eczema in infants. *Eur Respir J*. 2010;35:1228-1234.
139. Willers SM, Devereux G, Craig LC, et al. Maternal food consumption during pregnancy and asthma, respiratory and atopic symptoms in 5-year-old children. *Thorax*. 2007;62:773-779.
140. Nwaru BI, Erkkola M, Ahonen S, et al. Maternal diet during lactation and allergic sensitization in the offspring at age of 5. *Pediatr Allergy Immunol*. 2011;22:334-341.
141. Jaudszus A, Jahreis G, Schlormann W, et al. Vaccenic acid-mediated reduction in cytokine production is independent of c9, t11-CLA in human peripheral blood mononuclear cells. *Biochim Biophys Acta*. 2012;1821:1316-1322.
142. Kennedy G, Ballard T, Dop M. In: *Guidelines for measuring household and individual dietary diversity*. Nutrition and Consumer Protection Division, Food and Agriculture Organization of the United Nations; 2011: 1-60. ISBN 978-92-5-106749-9.
143. Guthrie HA, Scheer JC. Validity of a dietary score for assessing nutrient adequacy. *J Am Diet Assoc*. 1981;78:240-245.
144. Brownie C, Habicht JP, Cogill B. Comparing indicators of health or nutritional status. *Am J Epidemiol*. 1986;124:1031-1044.
145. Drewnowski A, Henderson SA, Shore AB, Fischler C, Preziosi P, Herberg S. Diet quality and dietary diversity in France: implications for the French paradox. *J Am Diet Assoc*. 1996;96:663-669.
146. Steyn NP, Nel JH, Nantel G, Kennedy G, Labadarios D. Food variety and dietary diversity scores in children: are they good indicators of dietary adequacy? *Public Health Nutr*. 2006;9:644-650.
147. Arimond M, Wiesmann D, Becquey E, et al. Simple food group diversity indicators predict micronutrient adequacy of women's diets in 5 diverse, resource-poor settings. *J Nutr*. 2010;140:2059S-S2069.
148. Kennedy GL, Pedro MR, Seghieri C, Nantel G, Brouwer I. Dietary diversity score is a useful indicator of micronutrient intake in non-breast-feeding Filipino children. *J Nutr*. 2007;137:472-477.
149. Lang JM, Eisen JA, Zivkovic AM. The microbes we eat: abundance and taxonomy of microbes consumed in a day's worth of meals for three diet types. *PeerJ*. 2014;2:e659. <https://doi.org/10.7717/peerj.659>
150. Maslin K, Venter C. Nutritional aspects of commercially prepared infant foods in developed countries: a narrative review. *Nutr Res Rev*. 2017;30:138-148.
151. Venter C, Maslin K. The future of infant and young children's food: food supply/manufacturing and human health challenges in the 21st century. *Nestle Nutr Inst Workshop Ser*. 2016;85:19-27.
152. Venter C, Pereira B, Voigt K, et al. Factors associated with maternal dietary intake, feeding and weaning practices, and the development of food hypersensitivity in the infant. *Pediatr Allergy Immunol*. 2009;20:320-327.
153. Katz Y, Nowak-Wegrzyn A, Grimshaw KE, et al. Is it the true incidence of IgE-cow's milk allergy (CMA) or CMA or IgE-CMA in some countries and CMA in others. *Allergy*. 2015;70:1509-1510; reply 10.
154. Global atlas of Allergy. 2014. <http://www.eaaci.org/globalatlas/GlobalAtlasAllergy.pdf>
155. Filipiak B, Zutavern A, Koletzko S, et al. Solid food introduction in relation to eczema: results from a four-year prospective birth cohort study. *J Pediatr*. 2007;151:352-358.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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