The Dietary Guidelines Advisory Committee met in the Agricultural Research Service, Children’s Nutrition Research Center, 1100 Bates Street, Houston, Texas, at 9:00 a.m., Barbara Schneeman, Chair, presiding. The meeting allowed for public viewing, both in-person and by webcast.

MEMBERS PRESENT
DR. BARBARA SCHNEEMAN, PhD, Chair
DR. RONALD KLEINMAN, MD, Vice Chair
DR. JAMY ARD, MD, Member
DR. REGAN BAILEY, PhD, MPH, RD, Member
DR. LYDIA BAZZANO, MD, PhD, Member
DR. CAROL BOUSHEY, PhD, MPH, RD, Member
DR. TERESA DAVIS, PhD, Member
DR. KATHRYN DEWEY, PhD, Member
DR. SHARON DONOVAN, PhD, RD, Member
DR. STEVEN HEYMSFIELD, MD, Member
DR. HEATHER LEIDY, PhD, Member
DR. RICHARD MATTES, PhD, MPH, RD, Member
DR. ELIZABETH MAYER-DAVIS, PhD, RD, Member
DR. TIMOTHY NAIMI, MD, MPH, Member
DR. RACHEL NOVOTNY, PhD, RDN, LD, Member
DR. JOAN SABATÉ, MD, DrPH, Member
DR. LINDA SNETSELAAR, PhD, RD, Member
DR. JAMIE STANG, PhD, MPH, RD, Member
DR. ELSIE TAVERAS, MD, MPH, Member
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DR. STOODY: Okay. Good morning. My name is Eve Stoody, and I’m a Lead Nutritionist for Nutrition Guidance at USDA Center for Nutrition Policy and Promotion, and I’m also the designated federal officer for this 2020 Dietary Guidelines Advisory Committee.

It is really my pleasure to welcome you to meeting four of the 2020 Committee and also to welcome you to Texas. We are holding this meeting at the USDA Agricultural Research Service Children’s Nutrition Research Center in Houston, Texas, and thanks to Dr. Denny Bier and their team for really welcoming us and for being fantastic hosts for this event.

This is the second time that the Dietary Guidelines Advisory Committee has met outside of the Washington, D.C., and the first time was over 25 years ago.

We also want to welcome all of you who are joining us on YouTube. This meeting, like our
previous meetings, will be live-streamed. That means -- hello, I would like to see the slides.

So this meeting will be live-streamed.

There will be -- and just a note that there will be different links for the morning and afternoon; that’s the nature of YouTube. So for those of you who are joining us online, when you registered, in your registration email you should have received four links, two for today and two for tomorrow.

We’ll try to remind you to change links after lunch. So we are very happy to have 19 of our 20 members here with us today. Unfortunately, Dr. Linda Van Horn was not able to join us here in person, but she is going to join us remotely as much as she is able.

We have about 1,000 people who have registered for this meeting, with about 150 who will join us at some point in person here in Houston. And as always, I just thank you for your interest and your support of the Dietary Guidelines.

So just a little bit of background and
a reminder: The 2020 Committee was established to conduct an independent review of current research on nutrition and health to be considered by the Departments of Agriculture and Health and Human Services, and the development of the 2020-2025 Dietary Guidelines for Americans.

This Committee was selected by Secretaries Purdue and Azar from USDA and HHS from nominations received from the public, and they were selected based on their education, experience, and expertise, and they were balanced on a number of factors, including things like geographic locations.

The Committee was announced in February 2019. And just as a reminder, this is not a committee is convened to provide expert opinions or to represent a specific viewpoint, but rather they were selected as independent scientists who will work together to review current evidence on diet and health.

Since this is a federal advisory committee, the federal government is required to
outline the duties -- the missions and specific
duties of the Committee, and we have done this
through our charge to the Dietary Guidelines
Advisory Committee.

You can see this charge on our slide.

We presented it every meeting, and it’s also on our
website, and their charge is to examine the
evidence on specific topics and scientific
questions identified by the Departments, and I’ll
talk about those here more in the next few slides,
to develop a report that outlines their
science-based review and recommendations to the
Department with rationale, and then to submit the
report to the Secretaries of USDA and HHS for
consideration as the Departments develop the next

So as we’ve talked about previously,
USDA and HHS added a new step to this process to
identify the specific topics or the specific
questions that the Advisory Committee were asked
to address.

In the past, we did outline some topic
areas, but in general the Committee identified the specific questions that they would consider. For this Committee, we added the step for the Committee where the Departments identified those topics and questions and asked the Committee to address those.

We did this for a number of reasons. One, it was in part due to recommendations from the National Academies on our process to kind of have the question development occur in a separate step, and we also really felt like it permitted a more transparent, inclusive, and deliberate process.

And I do want to note that the topics and questions were not developed in isolation. The process was led by the Center for Nutrition Policy and Promotion at USDA and the Office of Disease Prevention and Health Promotion, our partners at HHS, but it did include input from a number of federal agencies, as well as consideration of thousands of public comments.

And so in that process, specifically CNPP and ODPHP developed an initial list with input from some of our federal partners, we posted a list
of topics and questions for public comment for 30
days.

We received about 6,000 public comments
on those topics and questions and refined the list,
based on that input. We did prioritize the topics
and questions using four criteria: relevance to
the Dietary Guidelines -- and I’ll talk more about
that in just a second; importance to public health;
potential impact on the federal programs and
policies that we inform; and avoiding duplication
of other federal efforts.

Now, as I think everyone in this room
knows, in the field of nutrition, there are many
possible questions of scientific and public input
that have the potential to be explored. So this
includes things on food groups, on very specific
foods, questions on nutrients, food safety, food
labeling, menu labeling, food settings, food
policies, food behaviors, medical nutrition
therapy, and more.

And we really feel like the Dietary
Guidelines have an important slice of that
nutrition conversation. The Dietary Guidelines have a specific goal and a specific time line, and that is to provide food-based dietary guidance to the general public at least every five years.

Now, we do have a number of partners who we work with kind of in this larger nutrition conversation. So for example, the National Academies developed the nutrient recommendations known as the Dietary Reference Intakes, and there are a number of federal agencies and others involved in this space, including the Department of Health and Human Services, Food and Drug Administration, who work on food safety and labeling issues, but the point being here that there’s a lot of pieces, and the hope is that we all work together to kind of speak to the bigger nutrition picture.

Now, the topics and scientific questions we’ve asked the Committee to address focus on diet and health across the lifespan, and so kind of the main emphasis for this, I would say, is about that emphasis on the lifespan.
The topics and questions we asked the Committee to address build on topics and questions examined by previous dietary guidelines -- Dietary Guidelines Advisory Committees, I should say. So we didn’t start from scratch. We had a lot to work with from our previous committees. For example, the 2015 Committee did a number of questions on dietary patterns and added sugars. The 2010 Committee had a number of specific questions on seafood and alcohol. A number of committees have addressed kind of elements of dietary fats, beverages and patterns of eating, perhaps not as -- in kind of a broader scope, but in pieces of it. Previous committees have also described current intakes of Americans, as well as status of health across the American population, which will be talked about today. And since 2005, advisory committees have conducted food pattern modeling analysis. So that’s kind of the exposure element. In terms of the outcome, the Committee was asked
to consider a range of outcomes. So many previous
advisory committees have looked at the outcomes of
body weight or obesity, cardiovascular disease,
type 2 diabetes and cancer, and we asked the
Committee to examine those in kind of that health
discussion, but also some additional outcomes, so
for example, neurocognitive health has become
really a more recent interest in nutrition science.

So we did include brain health as part
of many of the questions that the Committee were
asked to consider. Sarcopenia, in particular,
trying to think about the older adult population
and having more targeted outcomes for that
population.

Bone health, which is of course
important for the older adults, but also children
and adolescents, as well as actual all-cause
mortality. We actually haven’t had many
committees that considered that broader all-cause
mortality outcome.

Now, each committee that we’ve had also
looks at some unique topic areas, and for 2020
process, these are the birth to 24 months performance population. There has been a growing interest in us including this population.

Traditional dietary guidance has focused on two years and older. And then the 2014 Farm Bill really solidified that inclusion in this edition.

And then an expanded focus on pregnancy and lactation. Previous advisory committees hadn’t necessarily excluded pregnancy and lactation, but they hadn’t had as focused questions on pregnancy and lactation, and perhaps more specific, they hadn’t really considered outcomes related to pregnancy and lactation. And so that’s been an addition here as well, and both of those are no small additions, as you’ll hear today shortly.

So in summary, I would say that there are many similarities between the work of this Committee and previous advisory committees, but there are some new topics.

I think a lot of what we’ve been seeing
is that a lot of the questions are more expanded,
so they're kind of the similar concept areas that
kind of broader exposure to more outcomes, and
there also are, of course, the new populations.

Now, as we’ve talked about previously,
and as with all of our Dietary Guidelines Advisory
Committees, the Committee’s task is time-limited.
As we’ve discussed, USDA and HHS requested the
Committee to report by May 2020, and that is so the
Departments can meet our mandate to release the new
edition of the Dietary Guidelines within five
years, which is by December of this year, December
of 2020.

So as we move into the last phase of the
Committee’s work, which is pretty crazy to think,
it’s similar to previous committees. The 2020
Committee and federal staff have been working to
refine, streamline, and prioritize the remaining
work within the remaining time, and you’ll hear
more about that over course of the next few days.

So all meetings of the full Committee
are open to the public. As I noted, this is the
fourth meeting. If you were not able to join us for meetings one, two, and three, that information is archived on our website, including the recordings of the meetings, as well as presentations, transcripts, and minutes.

Similar to the second meeting, this meeting will include an opportunity for individuals who had registered to provide oral comments to the Committee. However, if you did not have the opportunity to travel here to provide public comments in person, the written public comment period is always open.

We opened it in March of 2019, and it will stay open into May of 2020. So this meeting will be held today and tomorrow from 9:00 a.m. until 4:30 p.m. Central. I just note that because we usually function on Eastern Time, and some of us arrived early today, thinking we were still there.

So -- but we'll be on -- the meeting will be in Central Time. The agenda is available at DietaryGuidelines.gov, and Dr. Schneeman will give an overview of the agenda in her remarks.
We do want to announce today that we will host a -- the Committee will host a meeting on its report on May 11, which is a Monday. This will be a meeting -- you know, as we just talked about, we've asked for the Committee's report in May.

Their last meeting was scheduled for March, and we wanted to provide the Committee an opportunity to come together to discuss its final recommendations and refine its report, but also for the public to be able to hear some of the discussion around the Committee's final recommendations before they submit their report to the Departments.

This is the first time that we have hosted a meeting specifically focused on the Committee's report, and we hope that it is kind of helpful in hearing firsthand about their recommendations before they submit the report.

So we'll provide more information about this. We will publish this in the Federal Register. We'll include information on our website. We'll send out listserv messages for
those of you who have signed up as we have more
information, but for now, please save the date for
Monday, May 11. This meeting will be held by
webinar only. We will not have -- there won't
travel for that meeting.

So we encourage you to follow along at
DietaryGuidelines.gov in between the meetings, as
well as here today. The Committee will talk about
a number of different questions that they are
reviewing. If you want more information about the
questions that they are talking about, you can to
go DietaryGuidelines.gov.

There is a rotating banner in the middle
of that page, the orange banner there, and if you
click on "View Protocols," it will take you to a
list -- a website with a list of questions, and if
you click on your question of interest, then it will
take you a webpage devoted to the Committee’s
review on that question. So if you have something
of interest that you really want to learn more
about, we encourage you to go to the website.

So with that, I turn it over to the Chair
of the Committee, Dr. Barbara Schneeman.

CHAIR SCHNEEMAN: Thank you, Eve, and
let me add my welcome, certainly, to the Committee
members. It's great to see you all, and a full -- a
fairly full representation from the Committee, and
also to the attendees who are in the room, but also
to all of those who are listening on the webinar.

We do appreciate the interest in the
Dietary Guideline process and the work of this
Advisory Committee. And I want to extend a special
thank you to the CNRC for hosting the Committee
here.

I see Dr. Bier sitting over here on the
side. Thank you very much for the invitation to
be here, and the staff has been fantastic in terms
of helping us and making sure that things went
smoothly.

So thank you. So I will move into the
slides. So let me start, first of all, by just
giving you an overview. Sort of following on from
what Dr. Stoody presented, I'm going to talk more
specifically about our subcommittee structure, our
approaches to examining evidence and the
information to be discussed at this meeting.

In a sense, I’m now going to talk more
about how this Committee has moved forward with the
charge that we received from USDA and HHS. So
we’ll look at the subcommittee status and the
agenda for this meeting.

So just to remind you, these are the
subcommittee structures that were set up, so that
between the -- in the time between the public
meetings, work can proceed, and we have six
subcommittees and one cross-cutting.

I know the font is small there.
Dietary Patterns, Pregnancy and Lactation, Birth
to 24 Months, Beverages and Added Sugars, Dietary
Fats and Seafood, and Frequency of Eating, and the
cross-cutting group is the Data Analysis and Food
Pattern Modeling subcommittee, so aligned with
those topics that you’ve heard.

And I’m not going to read out the names,
because as we go through the subcommittee reports,
you will be getting that information. Just to
remind you that the subcommittees review the
evidence and provide advice to the parent
Committee, so the final decisions are being made
by the full Committee, and they’re done in this
public meeting format, which we’ll be having today
and tomorrow.

So just, again, we’ve talked about that
this each meeting, how the Committee approaches the
review of the evidence, the examination of the
evidence, and we use three approaches to examine
the evidence: data analysis, food pattern
modeling, and the NESR systematic reviews.

And each of these scientific approaches
has a protocol, and the protocol is a plan for how
one of the scientific approaches will be used to
examine evidence related to one of the questions
that the Committee has been asked to address.

As they’ve been developed, each of the
protocols are available, and Dr. Stoody gave you
the web link for that. And we -- in posting the
posting the protocols, we have invited feedback
from the public, and we found that feedback to be
very helpful. People have provided additional
references for additional consideration.

So information on the approaches and
the protocols have been presented at previous
meetings, and additional information that is
available at the DietaryGuidelines.gov.

So in the next few slides, I’m going to
just go through a brief overview of the information
to be presented by the subcommittee so you see the
genral format of how each of the subcommittee’s
reports is structured.

So throughout the presentations, you
will see an analytic framework which defines the
core elements of the diet and health relationship
to be examined. So you can see that that analytic
framework includes the intervention, exposures,
and the comparators that will be used.

In some cases, we have intermediate
outcomes -- obviously we’re very interested in the
health outcomes when available for our
review -- then key factors that could impact the
relationship; confounders, covariates, moderators
are specified in the analytical framework, and also
key definitions are given.

   So each of the protocols also look at
inclusion and exclusion criteria. And so you will
hear discussion of those criteria in each of the
systematic reviews, and these criteria are
developed up front and are used to screen the
articles that will be included or excluded from a
review.

   So there are a number of standard
criteria that apply across the different reviews
that the subcommittees have used consistently
across the reviews, and so these include areas such
as the study design.

   And I’m not going to read all of the
inclusion/exclusion, because we have talked about
these at each of the public meetings, and it’s also
available -- completely available to you on the
DietaryGuidelines.gov.

   So standard inclusion criteria include
that study design, what kinds of studies are
included, what are excluded, the publication
status, peer-reviewed articles, the language of publication -- English is what we’ve included -- the country of origin or the country that studied very high or high human development so it’s comparable to the U.S. population and can be generalized to the U.S. population. And then the study participants. We’re primarily interested in studies in humans, males and females, and so exclude animal or in vitro studies.

In addition, the health status of the study participants is included in our inclusion and exclusion criteria, and generally you’ll see that, while we’re obviously interested in participants who are healthy, we do also include participants who may be at risk for chronic disease, including those with obesity, and so that concept applies.

What we’re excluding are studies in which the participants have been diagnosed with a disease or hospitalized and that -- or participants with the outcome of interest that we’re looking at, and so they’re in a treatment study, or infants who were born preterm or low birth
weight. So that sets up what we include versus what we exclude in terms of health status.

Now, some of the criteria need to be tailored to the specific review, and those kind of tailored criteria might include diet-related interventions or an exposure of interest; health outcomes; the endpoint and/or an intermediate, whether or not that data are available; the date of publication, depending on what we already have from previous versions of the Dietary Guidelines; the work of other advisory committees; the size of the study groups; study duration; and the age of the study participants. And so those will be clearly specified in the protocols that are published.

So in the NESR systematic reviews, what you will -- because we're now moving into that phase where the subcommittees have been doing their work, and they are presenting more than the protocols; they're moving into presenting their draft conclusions -- you'll see a flow chart of the literature search and screening results, a
description of the evidence that is being examined
in depth by the subcommittee, the summary of the
evidence synthesis, and some draft conclusion
statements and grades for those particular
questions.

And I do want to highlight that what
we’re going to be presenting, just in the interest
of time, are in fact summary statements. The
Committee’s review includes a much more detailed
discussion and review of the included articles,
which will be provided in the Committee’s final
report and supporting online materials.

The intent is to summarize the
information today and tomorrow for discussion
across the full Committee. And again, a lot of
what we’re doing now, because it does involve a
Committee discussion, we’re presenting things that
are in their draft format and it will only be
finalized once we submit our report.

So there will be data analysis
questions that are presented today, and they
include -- they also follow a protocol and may
include some similar elements, including the
analytical framework, the analytical plan, the
results, and then draft conclusion statements that
the Committee will be discussing today.

So for both the NESR systematic reviews
and the data analysis questions discussed today and
tomorrow, conclusion statements -- draft
collection statements will be presented, and so
that draft conclusion statement is an answer to the
question of the evidence that is being reviewed.

They have been drafted by the
subcommittees, and they’re being brought to the
full Committee for discussion at these public
meetings. And again, these are considered draft
until the Committee submits its report to the
Secretaries, so they shouldn’t be interpreted as
the Committee’s final view or recommendations. The
Committee is working toward its final decisions.

So I do want to note that after the
conclusion statements are discussed by the
Committee at the public meetings, the systematic
reviews will go through a peer-review process, and
that is being coordinated by USDA’s Agricultural
Research Service, so that these reviews will be
peer-reviewed before the Committee finalizes.

And we have, in fact, invited Dr. David
Klurfeld from ARS to provide remarks at the next
meeting, the March meeting, on the process that is
being used for the peer-review process.

And we will then post the draft
conclusion statements online after that peer
review is completed. So you’ll be learning more
about that as we move forward, and that is a new
part of the DGAC process, so we’re learning about
it as we go.

So subcommittee status. They’re -- I
just want to summarize so you understand the full
scope of the work. I can assure you that the
subcommittees have been very busy, and there’s a
lot of demand in terms of time.

I also would note that I know everyone
on this Committee is fully appreciative of the
excellent staff that has been working with us,
keeping us on schedule, keeping us on track, and
doing the tremendous of work that it takes to pull
the evidence together so that the Committee can do
its evaluation.

So if we could go back to that slide,
please? So just the draft conclusions for
approximately 30 questions will be presented at
this meeting, including both NESR’s systematic
reviews and data analysis evaluation.

And so across the subcommittees, NESR
has screened over 265,000 articles and extracted
data and assessed risk of bias for over 500
articles. And I can assure you those numbers will
only still grow as we keep moving forward.

We are utilizing nearly 50 different
types of data analysis from the NHANES What We Eat
in America. We have begun to work on the food
pattern modeling, and we’ve refined a report
outline and are beginning to prepare some of the
report content.

So the task at hand is large. There’s
a huge amount of work that has been done. We know
that there’s still a huge amount of work to be done.
So members and staff, members of the Committee and the staff have been working to refine, streamline, and prioritize the remaining work so that we can meet the timeline.

So in our meeting number four, the meeting that we’re at, we’ll describe the status and provide updates on the work of the Committee. As Dr. Stoody noted, there’s an agenda available at DietaryGuidelines.gov.

So just to make sure we -- sort of make sure we connect with our YouTube participants, we’ll be sure that the meeting begins at 9:00 a.m. Central Time, and the afternoon session will begin at 1:00 p.m. Central Time.

Breaks, however, can’t really be set at a specific time, because of the nature of the reporting that we’re doing, but we’ll take breaks as they fit within the discussion framework.

So for today’s agenda, following the opening remarks, we’ll start with the subcommittee updates, and the subcommittees we expect to hear from today are Birth to 24, Pregnancy and
Lactation, Dietary Fats and Seafood, Beverages and Added Sugar, and the Data Analysis and Food Pattern Modeling, the cross-cutting working group, and obviously, with each of those subcommittees, we anticipate there will be Committee discussion.

So for tomorrow's agenda, again, we'll start at 9:00 a.m., and the subcommittee updates that will be held tomorrow are the Dietary Pattern subcommittee, the Frequency of Eating, some Committee discussion, and then we've also scheduled public comments, which will take place in the afternoon, and we are looking forward to those public comments.

And just to note that, yes, there's been a lot of interest in the DGAC work. The Committee has received approximately 17,775 written public comments, since the work began.

If there's interest in commenting on the new protocols that are presented in today and tomorrow's public meetings, it's most useful to the Committee if those comments on the protocols are received by Friday, February 7.
And again, we’ve found the comments on the protocols to be helpful, but for the Committee to keep progressing with its work, we need them by February 7. But as noted by Dr. Stoody, the written public comment period for more general comments is open until we complete our work in May of 2020.

So with that, that concludes my comments, and I’ll just turn to the Committee members just to see if there’s anything, question or comments that any Committee members may want to make?

(No response.)

CHAIR SCHNEEMAN: So with that, I’m going to -- Dr. Kleinman, you may have some comments as well, but I’ll turn it over to you for the first subcommittee reports.

VICE CHAIR KLEINMAN: Thank you. Thanks, Barbara. That was very complete, and I have very little to add. This is our fourth meeting together, and so it’s an opportunity for us all to be here with each of the subcommittees
that’s been working on, and a great deal of work has taken place since the last meeting, so the remainder of the day today and tomorrow will be these report outs of the subcommittees.

I think we’ll go right into the first one now and then in terms of breaks, we recognize that there are some biological imperatives here, and so we will try to take a brief break, perhaps, between the first and second.

So with that, I’m going to go ahead and turn this over to Kay Dewey, and she will talk about the subcommittee for Birth to 24 Months.

MEMBER DEWEY: Thank you very much, Ron and Barbara. I am very pleased to be able to report to you today on behalf of this subcommittee. And the members of this subcommittee have been working very hard, many hours every week, to get to this point.

We have a number of questions that have been addressed, and the NESR staff have been extremely busy screening the literature, preparing the results, extracting the data, and preparing...
evidence portfolios for us to review.

And so today we will be presenting draft conclusion statements for the eight topics shown here. Although it’s eight topics, there are 66 conclusion statements we need to go through, and if I did read every single one of them in full, it would take more than an hour.

So I’m going to try to go through them as quickly as I can, while not skipping anything important. Those include three questions or topics on the relationship between human milk and infant formula and three outcome areas: micronutrient status, atopic disease, and long-term health outcomes.

Then there are five questions on complementary feeding, and five outcome areas: atopic disease, developmental milestones, growth, size, and body composition, micronutrient status and bone health.

We still have work to do for five other topics that are listed here, two additional questions related to human milk and infant formula,
and those relate to growth, size, and body composition and developmental milestones.

And then the three new questions that we have on nutrients from supplements or fortified foods, and three outcome domains: growth, size and body composition, bone health and micronutrient status.

These are some of the key definitions for our reviews, which we have presented previously, but to remind you of those and the scope of the questions we’re investigating, I wanted to go through them.

Human milk refers to mother’s own milk, so our reviews did not include examinations of donor milk. And we’ve used the term human milk feeding, instead of breastfeeding, to be clear that we have examined human milk fed at the breast, as well as human milk that has been expressed and fed fresh or after refrigeration or freezing.

Infant formula refers to commercially prepared infant formulas that meet FDA or Codex Alimentarius standards. In practice, this has
been a tricky definition to apply because there are a lot of studies that examine experimental infant formulas with ingredients such as dietary nucleotides or DHA, prior to putting them on the market.

So we have included this evidence if the formulas met the FDA or Codex standards. We did this because we thought it was important to examine infant formulas with ingredients that are commercially available.

And lastly, complementary foods and beverages refers to foods and beverages other than human milk or infant formula. That includes liquids, semisolids, and solids that are provided to an infant or young child to provide nutrients and energy.

I want to thank the public for submitting comments on the work that was presented during meeting three. We carefully reviewed and discussed all of those comments, and we would very much welcome public comments on what we present today, as Dr. Schneeman mentioned, by February 7.
So to begin, I will review some draft conclusions for the relationship between duration, frequency, and volume of exclusive human milk and/or infant formula consumption and micronutrient status.

This is the analytical framework that we developed that shows the scope of this question, and we divided the duration, frequency, and volume of exclusive milk, of human milk or infant formula, into a series of four comparisons that align with the first feeding decisions that caregivers make, and those include whether or not to feed human milk; and then, for caregivers who do decide to do so, how long to feed human milk, so the duration of human milk consumption, and then how long to feed human milk exclusively.

So you’ll note that we examined exclusive human milk consumption prior to the introduction of infant formula only, and that’s to avoid overlap with another review which we will also present today that examines the timing of the introduction of complementary foods and beverages.
And then if caregivers have decided to supplement human milk with infant formula, our final comparison examines the intensity or proportion or amount of human milk that is fed to mixed-fed infants.

And then on the right, you can see that we examined iron, zinc, iodine, vitamins C and B12, and fatty acid status from birth to 24 months. This flow chart shows the literature review and screening results, and we used two different literature searches which are noted with the letters A and B in the flow chart.

Literature Search A was from the Pregnancy and Birth to 24 Months Project, which used a search date range of January 1980 to March 2016, and this literature search was very large, because it was intended to find studies for several questions related to human milk and infant formula.

Literature Search B was smaller, because it was intended to capture just the literature published in the last three years. And you can see that ultimately 23 articles were
identified that met the inclusion criteria for the question about feeding human milk and infant formula and micronutrient status outcomes.

On this slide, we want to give you a snapshot of the evidence by showing how many of those 23 studies provided evidence for each component of our analytical framework. And you can see that where there was evidence to address a topic, the number of studies was small.

Now, a small number of studies may provide sufficient evidence to determine associations, for example, if the evidence is consistent and has a low risk of bias. However, that was generally not the case in this body of evidence.

You can also see the majority of evidence addressed ever, compared with never, consuming human milk. So we’ll go one by one through those comparisons.

This is the evidence related to ever versus never consuming human milk, and these were generally studies that compare infants who were fed
human milk with infants who were fed infant formula that had a novel composition at the time of the study, such as added DHA or different levels of iron, and infants who were fed conventional infant formula.

Now, as you can imagine, the evidence would show that the formula’s composition can impact nutrient status outcomes. For example, formula with DHA can impact DHA status, and this complicates our synthesis of the evidence, because infants in the studies were fed a wide variety of infant formulas.

The 23 studies in this body of evidence generally studied healthy full-term infants who were recruited at or close to birth and who were from the U.S. and several other countries.

As I’ve already mentioned, the majority of evidence examines ever compared with never consuming human milk and the duration of human milk consumption. It’s important to note that other components of the infants’ diets varied between studies and also didn’t tend to be well reported.
For example, the exclusivity of human milk, the types and amount of formula fed in addition to human milk, the types and amount of complementary foods and beverages in addition to human milk or infant formula, and any intake of supplements.

At the bottom of the slide, you can see the outcomes that were reported by the studies for each of these nutrients. Now, there was evidence available from a small number of studies, and generally they did not show consistent associations between the comparisons that are shown in this slide.

So for ever -- compared with never consuming human milk, there were not consistent associations with anemia, hemoglobin, hematocrit and the other indicators of iron status shown here, or with zinc status.

Also, that was true for the duration of any human milk consumption among infants fed human milk, and anemia and markers of iron status, zinc status, vitamin D status and fatty acid status.
And lastly, the same was true for the duration of exclusive human milk consumption before the introduction of infant formula and fatty acid status.

The most substantial evidence that we reviewed was from seven studies that examine the relationship between ever compared with never consuming human milk and fatty acid status.

And these studies indicated that there may be an association between feeding human milk compared with infant formula and fatty acid status. And this body of evidence had an adequate number of sufficiently powered studies, with some inconsistencies that can likely be explained by methodological differences; for example, the use of formulas with different fatty acid composition.

There were several limitations that included the risk of bias, especially confounding; the study directness, because these studies are mostly designed to examine the effects of infant formula composition rather than to directly compare infants fed human milk with those fed
infant formula.

And also generalizability. For example, in two studies, there were no non-white participants, and other studies did not report race or ethnicity. Also, it’s unclear whether the experimental formulas are similar to current formulas on the market in the U.S.

But we did draft a conclusion statement regarding ever versus never consuming human milk, and this states that moderate evidence indicates that ever compared with never consuming human milk may be associated with fatty acid status. The difference in fatty acid status between infants who are fed human milk and infant formula likely depends on the fatty acid composition of the human milk and the infant formula being compared.

We found insufficient evidence available to determine the relationship between ever compared with never consuming human milk and iron and zinc status from birth to 24 months, and no evidence for the relationship between ever versus never consuming human milk and the other
micronutrient status outcomes: iodine, vitamin B12 and vitamin D status.

Continuing on with regard to duration of human milk feeding, insufficient evidence was available to examine this relationship for iron, zinc, vitamin D and fatty acid status, and there was no evidence to determine that relationship for iodine and vitamin B12 status.

In addition, regarding duration of exclusive human milk consumption, there was insufficient evidence for the relationship to fatty acid status, and no evidence for the relationship to iron, zinc, iodine, vitamin B12 and vitamin D status.

And finally, with regard to intensity, proportion or amount of human milk in mixed-fed infants, there was no evidence to examine the relationship to iron, zinc, iodine, B12, vitamin D, or fatty acid status.

So next we will review the draft conclusions for the relationship between duration of exclusive human milk or infant formula
consumption, and food allergies and atopic allergic diseases and long-term health outcomes.

Now, these questions have been answered with existing NESR systematic reviews, and our updated protocols, which are available at DietaryGuidelines.gov, describe that we will use these reviews as is, because they were completed recently and capture over 35 years of evidence. The papers from those reviews were published in the American Journal of Clinical Nutrition in 2019.

However, we would like to ask the public to please submit public comments if you know of any articles published since 2016 that meet the inclusion criteria and would also significantly affect these conclusions.

The Committee did carefully review the conclusion statements in the existing NESR systematic reviews; and we flagged those that we thought warranted an informal search to identify new evidence that has emerged since 2016, focusing on other published systematic reviews.

We did not locate any studies that would
have modified the conclusions, but again we do
appreciate any comment the public would like to
provide. So as I mentioned the Committee will be
answering these questions using the nine existing
NESR systematic reviews completed as part of the
Pregnancy and Birth to 24 Months Project by the
Infant Milk-Feeding Practices Technical Expert
Collaborative, and the link to the documentation
is provided here.

We would like to sincerely acknowledge
the work of this group of scientists who comprised
this technical expert collaborative and conducted
these reviews with NESR.

For this set of reviews, the literature
search was conducted between January 1980 and March
2016. For never versus ever feeding human milk and
atopic disease, 44 articles met the inclusion
criteria, and you can see the distribution of the
outcome that was examined. Almost all of this
evidence was from observational studies.

For duration of any human milk feeding
and atopic disease, 35 articles met the criteria,
and almost all the evidence was from observational studies.

For duration of exclusive human milk feeding prior to the introduction of infant formula, only one article met the inclusion criteria.

This summarizes what was concluded regarding the relationship between never versus ever feeding human milk and these outcomes.

Firstly, moderate evidence suggests that never, in comparison to ever being fed human milk, is associated with a higher risk of childhood asthma.

Again, just to emphasize, these statements are worded so that the risk is related to never feeding human milk. And in this case, there were 17 independent studies contributing to that conclusion statement.

For the second one, limited evidence does not suggest a relationship between never versus ever being fed human milk and atopic dermatitis in childhood.

For the other relationships, evidence
about never versus ever being fed human milk and atopic dermatitis was inconclusive, and there was insufficient evidence to examine how it related to the other outcomes that are listed here.

Again, I’m not going to read every word. All of these statements are available in the published articles.

This shows the conclusion statements for the relationship between shorter versus longer duration of any human milk feeding and these outcomes.

Moderate evidence, mostly from observational studies, suggests that among infants fed human milk, a shorter versus a longer duration of any human milk feeding is associated with a higher risk of asthma in childhood and adolescence. This included 20 independent studies.

Limited evidence does not suggest a relationship between duration of any human milk feeding and allergic rhinitis or atopic dermatitis in childhood.

Evidence about the relationship
between shorter or longer duration of human milk feeding and atopic dermatitis from birth to 24 months is inconclusive, and there’s insufficient evidence to determine the relationship with the other outcomes in this set.

In terms of the shorter or longer duration of exclusive human milk feeding before introduction of infant formula, there is insufficient evidence to examine this relationship to all of the outcomes that were examined.

Moving on, then, to the long-term outcomes, this shows the evidence that was available to examine those. First, with regard to never versus ever feeding human milk and cardiovascular disease outcomes, there were 13 articles that met the inclusion criteria, and you can see the types of outcomes that these studies examined.

For duration of any human milk feeding, there were 24 articles, and for duration of exclusive human milk feeding and cardiovascular disease outcomes, there were six articles
included.

So I’ll go through those conclusion statements as well. For never versus ever feeding human milk, limited evidence suggests that never versus ever being fed human milk is associated with higher blood pressure, within a normal range, at six to seven years of age.

The evidence about the relationship of never versus ever being fed human milk with blood lipids in childhood was inconclusive and there was insufficient evidence for the relationship to the other CVD outcomes examined.

In terms of shorter versus longer duration of any human milk feeding, moderate evidence suggests that there is no association between the duration of any human milk feeding and blood pressure in childhood.

And I wanted to call out one study here that was quite important. There was compelling evidence from the Promotion of Breastfeeding Intervention trial that is the only randomized trial in this body of evidence, and it showed no
significant relationship between duration of any human milk feeding and blood pressure at six and a half or 11-1/2 years of age.

There was also inconsistent evidence across six independent prospective cohort studies. The second bullet here, the evidence about the relationship of shorter versus longer duration of human milk with blood lipids in childhood and adulthood and with metabolic syndrome, was inconclusive, and there was insufficient evidence to determine the relationship to the other CVD outcomes.

Continuing on with shorter versus longer duration, limited evidence suggests that there is no association between the duration of exclusive human milk feeding and blood pressure in childhood or metabolic syndrome at 11.5 years of age, and most of this evidence comes from this one non-U.S. sample that was assessed using a very strong study design.

And there was insufficient evidence to determine the relationship of the duration of
exclusive human milk feeding with the other endpoint CVD outcomes.

The other long-term outcome examined was diabetes, and in this case, there were 21 articles that met the inclusion criteria for the comparison of never versus ever feeding human milk.

You can see that most of those are regard -- with regard to type 1 diabetes. For duration of any human milk feeding and diabetes, 37 articles met the criteria, and 30 were focused on type 1 diabetes.

For duration of exclusive human milk feeding, there were 18 articles that met the criteria; again, 17 about type 1 diabetes. So this summarizes what was concluded about never versus ever feeding human milk.

Limited evidence from observational studies suggests that never versus ever being fed human milk is associated with a higher risk of type 1 diabetes. There’s insufficient evidence to determine whether or not there is a relationship between never versus ever feeding human milk and
type 2 diabetes, prediabetes and the other outcomes shown here.

In terms of the duration of human milk feeding, moderate evidence from observational studies suggests that among infants fed some amount of human milk, a shorter versus a longer duration of human milk feeding is associated with a higher risk of type 1 diabetes.

Limited but consistent evidence suggests that the duration of any human milk feeding is not associated with fasting glucose or insulin resistance in childhood or during the transition from childhood into adolescence. And there’s insufficient evidence for the relationship to type 2 diabetes, prediabetes or the other outcomes shown here.

And then in terms of shorter versus longer duration of exclusive human milk feeding, limited evidence from observational studies suggests that a shorter duration is associated with a higher risk of type 1 diabetes.

Limited evidence from a single study
that used the strong design also suggests that the
duration of exclusive human milk feeding is not
associated with fasting glucose or insulin
resistance at 11.5 years of age. And there's
insufficient evidence to determine the
relationship with type 2 diabetes, prediabetes and
the other outcomes shown here.

Moving on, next we'll review the draft
conclusions for the relationship between
complementary feeding and the five outcome domains
that are listed here: micronutrient status;
growth, size and body composition; developmental
milestones, including neurocognitive development;
food allergies and atopic allergic diseases; and
bone health.

These have also been answered with
existing NESR systematic reviews, and the
protocols again are at DietaryGuidelines.gov.
And we will be using these reviews as is, again,
because they were completed recently and capture
over 35 years of evidence.

These papers were also published in the

However, as mentioned previously, we would like to ask the public to please submit public comments if you know of any articles published since 2016 that meet the inclusion criteria and would significantly affect the conclusions that I will be presenting.

So the Committee will be answering these questions using 10 existing NESR systematic reviews completed as part of the Pregnancy and Birth to 24 Months Project by the Complementary Feeding Technical Expert Collaborative, and this gives the link for the complete documentation of that work.

Again, we would like to acknowledge the work of this group of scientists who comprise the complementary feeding TEC, who conducted these reviews with NESR. This literature search spanned from January 1980 to July 2016.

For complementary foods and beverages, they were divided into two overarching types of questions: the timing of introduction of
complementary foods and beverages, and the types
of amounts.

So for this first set of outcomes, which
are micronutrient status, there were nine studies
that met the criteria for the timing of
introduction. Most of these examined iron status;
a few examined zinc, vitamin D, vitamin B12, folate
and/or fatty acid status. For the types and
amounts of complementary foods and beverages, 31
articles met the criteria. Most examined
iron-fortified cereals and meats with respect to
iron status. Several examined zinc and fatty acid
status. And very few studies examined vitamin D,
vitamin B12, and folate status.

So I’ll begin with the relationship
between the timing of introduction of
complementary foods and beverages and
micronutrient status.

Moderate evidence suggests that
introducing complementary foods and beverages at
four months of age compared to six months of age
offers no long-term advantages or disadvantages in
terms of iron status among healthy, full-term infants who are breastfed, fed iron-fortified formula, or both. And there were nine studies that met the criteria for this question.

There is not enough evidence to determine the relationship between timing of introduction and zinc, vitamin D, vitamin B12, folate, or fatty acid status.

Additional factors that need to be considered in examining the relationship between the age at which complementary foods and beverages are introduced and micronutrient status include birth weight and timing of umbilical cord clamping, both of which affect iron stores of the newborn; postnatal growth; type of feeding, at the breast or formula or mixed feeding; and intake and absorption of iron from sources other than human milk, including the types and amounts of complementary foods and beverages being consumed.

This summarizes the conclusions for the types and amounts of complementary foods and micronutrient status. Thirty-one studies met the
inclusion criteria for this review. And strong evidence suggests that consuming complementary foods and beverages that contain substantial amounts of iron, such as meats or iron-fortified cereal, helps maintain adequate iron status or prevent iron deficiency during the first year of life among infants with insufficient iron stores or breastfed infants who are not receiving adequate iron from another source.

However, the benefit of these types of complementary foods and beverages for infants with sufficient iron stores, such as those consuming iron-fortified infant formula, is less evident.

There’s not enough evidence to determine the relationship between other types and amounts of complementary foods and beverages containing lesser amounts of iron, such as fruits and vegetables and iron status.

Then in terms of the other nutrients of interest, limited evidence suggests that consuming complementary foods and beverages that contain substantial amounts of zinc, such as meats or
cereals fortified with zinc, support zinc status
during the first year of life, particularly among
breastfed infants who are not receiving adequate
zinc from another source.

However, the benefit of these types of
complementary foods for infants consuming
fortified infant formula is less evident.
Moderate evidence suggests that consuming
complementary foods and beverages with differing
fatty acid profiles, particularly long-chain
polyunsaturated fatty acids, can influence fatty
acid status.

Continuing on this theme, during the
second year of life, food sources of micronutrients
are still needed, but there’s limited evidence to
indicate which types and amounts of complementary
foods and beverages are associated with adequate
micronutrient status, and there’s not enough
evidence to determine the relationship between the
types and amounts of complementary foods and
beverages and vitamin B-12, vitamin D, or folate
status.
Now I'm going to move on to the next outcome domain, and that is food allergies and atopic allergic diseases. For the timing of introduction of complementary foods and beverages, 31 studies met the inclusion criteria, and most of them examined food allergies. For types and amounts of complementary foods and beverages, 39 met the criteria and most examined the most common allergenic foods.

This has to do with the timing of introduction of complementary foods and beverages. Moderate evidence suggests that there is no relationship between the age at which complementary feeding first begins and the risk of developing food allergy, atopic dermatitis or eczema, or asthma during childhood.

There's insufficient evidence to determine the relationship between age at which complementary foods or beverages are first introduced and risk of developing allergic rhinitis during childhood.

Now, the rest of the series of slides
focuses on the specific types of complementary foods being introduced, and so these are divided into several different slides.

I wanted to mention that the studies are mostly focused on food allergy to that particular food component. And in this case, we will be talking about peanut, tree nuts and seeds.

There is strong evidence to suggest that introducing peanut in the first year of life after four months of age may reduce the risk of food allergy to peanuts, and this evidence is strongest for introducing peanut in infants at the highest risk with severe atopic dermatitis and/or egg allergy to prevent peanut allergy, but it is also applicable to infants at lower risk. However, the evidence for tree nuts and sesame seeds is limited.

Limited evidence also suggests that there is no relationship between consumption of peanut, tree nuts or sesame seeds during the complementary feeding period and the risk of atopic dermatitis or eczema and asthma.
And there is not enough evidence to determine if there is a relationship between consuming peanut, tree nuts or seeds and allergic rhinitis.

What I want to also mention is that many of the studies included in this review exclusively enrolled or primarily enrolled subjects who were at a greater risk of allergies and/or atopic disease than the general population on the basis of family history.

However, despite this, the reviewers concluded that the results are probably generalizable to infants and toddlers who are at lower risk for atopic disease, although the magnitude of the associations may be smaller.

There were 28 studies that examined the consumption of eggs as a complementary food in relationship to the risk of developing any atopic disease, including six randomized controlled trials.

From that body of evidence, it was concluded that moderate evidence suggests that
introducing egg in the first year of life, after
four months of age, may reduce the risk of food
allergy to egg.

Limited evidence suggests that there is
no relationship between the age of introduction to
egg and the risk of atopic dermatitis or eczema and
asthma, and there’s not enough evidence to
determine the relationship between egg and
allergic rhinitis.

For fish, 24 studies examined fish as
a complementary food, including one randomized
controlled trial. From this body of evidence,
there is limited evidence that suggests that
introducing fish in the first year of life after
four months of age may reduce the risk of atopic
dermatitis and eczema, and there is not enough
evidence to determine this relationship to the risk
of allergy to fish or other foods, asthma or
allergic rhinitis, and also not enough evidence for
the relationship to the risk of food allergy,
allergic rhinitis, eczema, asthma or allergic
rhinitis.
There were 17 studies that examined the consumption of wheat or cereals and these outcomes, and all of these were observational studies. So limited evidence suggests that there is no relationship between the age of introduction or cow’s milk products such as cheese and yogurt and the risk of food allergy and atopic dermatitis and eczema.

There’s not enough evidence to determine if there’s a relationship between consuming milk products during the complementary feeding period and the risk of asthma or allergic rhinitis.

Did I skip something? I’m going to go back a second. Here we go. Sorry. There are a lot of outcomes here. So sorry. I’m going to go back to wheat and soy. And I did mention there were 17 studies that examined the consumption of wheat or cereals, and these were all observational, and there’s not enough evidence for those related to wheat to determine the relationship to risk of food allergy, atopic dermatitis and eczema, asthma or
allergic rhinitis.

For soy, there were four prospective studies that examined this relationship and that indicated that there was not enough evidence to determine if there was a relationship between soybean consumption and the risk of any of these outcomes. Okay, I think I will move on.

There were several observational studies that also examined the relationship between other types of complementary foods and beverages that are generally not considered to be major allergens; for example, fruit, vegetables, and meats, and this conclusion was that there was limited evidence from observational studies that suggest that introducing foods not commonly considered to be allergens in the first year of life after four months of age is not associated with risk of food allergy, atopic dermatitis or eczema, asthma or allergic rhinitis.

There were also several observational studies that examined dietary diversity or dietary patterns, and these were 11 prospective cohort
studies and three case control studies, but there
was not enough evidence to determine a relationship
between these aspects of the diet and any of these
outcomes.

Okay. Moving on to the next set of
outcomes, which is growth, size, and body
composition, there were 81 studies that met the
inclusion criteria for the timing of introduction
of complementary foods and beverages, and 49 that
met the criteria for types and amounts.

So in terms of timing of introduction,
moderate evidence suggests that the first
introduction of any complementary food or beverage
between four to five months, compared to
approximately six months of age, is not associated
with weight status, body composition, body
circumferences, weight, or length, among generally
healthy, full-term infants.

Limited evidence suggests that
introducing complementary foods and beverages
before four months of age may be associated with
higher odds of overweight and obesity. And
there’s not enough evidence to determine the relationship between introduction of complementary foods and beverages at seven months or later on growth, size, and body composition.

In terms of types and amounts of complementary foods, moderate evidence indicates that a higher versus lower meat intake or meat versus iron-fortified cereal intake over a shorter duration during the complementary feeding period, does not favorably or unfavorably influence growth, size and/or body composition.

And there’s insufficient evidence to determine the relationship between meat intake and prevalence or incidence of overweight or obesity. Limited evidence suggests that the type or amount of cereal given does not favorably or unfavorably affect these outcomes.

In terms of fatty acids, moderate evidence suggests that consumption of complementary foods with different fats and/or fatty acids composition does not favorably or unfavorably influence growth, size, or body
composition.

And there’s not enough evidence to
determine the relationship to the prevalence or
incidence of overweight or obesity. Limited
evidence suggests that sugar-sweetened beverage
consumption during the complementary feeding
period is associated with decreased risk of obesity
in childhood, but it is not associated with other
measures of growth, size, and body composition.

There is limited evidence that showed
a positive association between juice intake and
infant weight-for-length and child BMI z-scores.

No conclusion could be made about the
relationship about other complementary foods as
listed here and growth, size, body composition, or
overweight or obesity.

And also no conclusion could be made
about the relationship between distinct dietary
patterns during the complementary feeding period
and growth, size, body composition, or these other
outcomes.

There was a much smaller body of
evidence regarding developmental outcomes. For the timing of introduction of complementary foods and beverages, only three studies met the criteria, and for types and amounts, only eight studies met those criteria.

So not surprisingly, given that small evidence base, there was insufficient evidence to draw conclusions about the relationship between the timing of the introduction of complementary foods and beverages and developmental milestones.

One of the issues with this body of evidence is that there is the potential for reverse causation. In other words, the child might be more developed and therefore be more demanding of introduction of other foods and beverages. And so with observational studies, it’s very difficult to study this relationship.

There was also insufficient evidence to draw a conclusion about the relationships between the types and amounts of complementary foods and beverages consumed and developmental milestones.

There was also a very small evidence
base in terms of bone health. Three studies met the criteria for timing of introduction of complementary foods and beverages, and eight met the criteria for types and amounts.

So again, not surprisingly, the conclusion was that there was insufficient evidence to draw conclusions about the relationship of timing of introduction of complementary foods and beverages and bone health.

And similarly, insufficient evidence was available between the types and amounts of complementary foods and beverages and bone health.

Okay. So those are the 66 conclusion statements we had to get through today, and now I'm going to present some of the discussions that we've had related to refining and prioritizing the remaining work in front of us.

So as I mentioned, we have two questions regarding human milk and infant formula and outcome domains, including growth, size, and body composition. So for this one, it's a very, very large literature, and for this purpose, we've
decided to examine outcomes related to body composition only, which includes obesity and overweight.

Our rationale for this is that we already know that growth curves differ between infants fed human milk and those fed infant formula. In fact, the U.S. Government convened an expert panel several years ago to review such evidence, and as a result, the CDC adopted the World Health Organization growth curves from birth to age two years, which reflect the growth of breastfed children.

On the other hand, the relationship between human milk or infant formula consumption and body composition outcomes, including obesity, warrants further examination, and for that reason, we have altered the protocol for this, which is going to be available on DietaryGuidelines.gov.

We also discussed the remaining questions that examine intake of nutrients from supplements and fortified foods, and for this, we decided to prioritize for the first question
related to growth, size and body composition, to
focus only on iron and iron from supplements.

For the second one related to bone
health, we decided to focus only on vitamin D and,
again, only from supplements. And for the third
question related to nutrient status, we decided to
focus on iron and vitamin D from supplements only.

Our rationale for limiting these
reviews to the nutrients from supplements is that
the existing reviews from the previous project,
which I just reviewed with you today, examined
complementary foods and beverages and included
fortified foods.

So we feel that the real need for new
work here is on these nutrients from supplements.
Our rationale for examining iron and vitamin D only
is that we would like to review evidence about
nutrient supplements that are currently
recommended for this age group.

So that is where we will be moving
forward as we continue the work. So our next steps
are summarized here. There will be a literature
search on iron and vitamin D from supplements and nutrient status.

There will be screening of the literature also for iron and vitamin D from supplements. Oh, that will include a screening of the literature, as well as vitamin D from supplements and bone health.

We will then have to extract the data, assess risk of bias, and develop conclusions and grades for the five questions that are listed here. And again, those are human milk and infant formula consumption, growth, size and body composition, and developmental outcomes, and then the specific nutrients from supplements and the three outcome areas that I mentioned.

And then lastly, we will be drafting -- going through the peer-review process and drafting the report. And with that, I would like to again thank the members of the subcommittee very much, thank and acknowledge the very hard and extensive work by our support staff, who are listed on this slide.
Thank you very much.

(Applause.)

VICE CHAIR KLEINMAN: Thank you, Kay, for an incredibly complete summary of the work of the subcommittee. So we've already had some cross-cutting conversations between a few of the subcommittees, but this is our opportunity now as a full Committee to ask any further questions or to comment on what Kay has presented.

So I'll open it up to the Committee now for questions. Rick. And don't forget to say your name.

MEMBER MATTES: Rick Mattes. So I have five questions, but a lot of them will be really short, I think, responses. The first is, you used the term "intensity" of feeding. I'm just not clear on what intensity means, so a clarification, that would be helpful.

Your recommendation regarding fat intake and fatty acid status just referred to association, whereas all the other recommendations had directionality to them.
If it’s possible to tweak that, I think it would be more useful. If it’s not, it’s not.

In the report on never versus ever and risk of type 1 diabetes, you found an association there; this is my lack of knowledge, is there a plausibility? Is there a mechanism that would make that make sense?

With the peanut recommendation -- again I’m old school -- is there some subset of people that may actually be at risk so a general recommendation saying early introduction is okay, holds risk for some subgroup of the population, or it really is a clear bill of health for such a recommendation?

MEMBER DEWEY: Can you repeat that again?

MEMBER MATTES: So the recommendation for early exposure to peanut seemed to be just generally positive, and I’m just wondering if there is a subgroup of individuals that might be at risk? Because if people just look at that recommendation, they’d think that it’s good to go, but maybe there
are some that would be at risk. I don’t know.

And lastly, for the sugar recommendation, it says that sugar-sweetened beverage consumption during complementary feeding is associated with increase of obesity, but not associated with body composition. I’m just not clear how to juxtapose those.

MEMBER DEWEY: So those are very good questions, and I’m going to rely on others in the room to help with some of the answers.

For the intensity of breastfeeding, this is part of three different aspects of when children are fed both infant formula and human milk.

It relates to how many of the feeds are human milk versus formula or the amounts, or in some other way judging the proportion. So the intensity refers to how much of that is human milk.

It’s a guesstimate, in most cases, because they’re not measuring human milk intake. So that’s why different words are used by different researchers.
Is there any -- if anybody wants to add anything from the staff who knows these definitions by heart?

Yes, please, Darcy.

MS. GUNGOR: Just one clarification, which is that intensity, proportionate amount, be included in any evidence that was examined, either at a single point in time or over a duration of time, and that might have included another variable in the definition such as months or years, that sort of thing.

MEMBER MATTES: So in any write-up, that will be defined somewhere?

MEMBER DEWEY: In the paper that was published, that is given, yes.

And then in terms of fat composition of complementary foods, and that we said there is an association without the direction, and that was on purpose, because it really depends on what fatty acids are in those foods.

So if there is an increased amount of polyunsaturated fatty acids, for example, that
will generally show up in the fatty acid status as
a positive relationship in the child. But it's not
simple to summarize that in the conclusion
statement.

So in the paper that was published, it
goes through exactly what all those relationships
were. We can talk further about whether there is
some way to modify that, but for that question,
we're relying on the existing review that's been
published already, and that's their exact wording.

MEMBER MATTES: So it is possible for
it to be inverse in some instances, or could it just
be stated as a direct relationship?

MEMBER DEWEY: Well, I would have to
read again exactly which studies that -- there's
always theoretically the possibility that if you
increase intake of omega-6, you might reduce
omega-3 status, or vice -- I mean, so -- and that's
why I don't want to get too specific about it right
now.

MEMBER DONOVAN: Yeah. And I think in
some the ever versus never types of questions,
because the composition of breast milk fatty acids
differ from formula, because the breastfeeding
moms’ maternal diet, and formulas are added oil,
so I think in some cases they’re higher, and in some
cases, they’re lower.

   So rather than have a conclusion
statement that was three paragraphs long to go
through each of them, it was basically a general
statement of there is associations between dietary
intake and the outcome.

   MEMBER DEWEY: So I thought you were
referring to fatty acids from complementary foods
and beverages, but we’ve been also referring to the
never versus ever human milk and those questions?

   MEMBER MATTES: I’m not sure where my
brain kicked off as you were going through, but for
either of them, I’m --

   MEMBER DEWEY: Okay. So there were
two different questions where fatty acid status was
an outcome. One was from complementary foods and
beverages. That’s what I was answering.

   For human milk, ever versus never,
duration et cetera, we also shied away, as Sharon explained, from stating a direction, because it’s -- as we were talking yesterday in our subcommittee meeting, it’s complicated, because of the composition of human milk and the possibility that the mammary gland has endogenous synthesis of many of these fatty acids. And so it’s something that we will describe in more detail in the write-up.

And then you asked about never versus ever breastfeeding in type 1 diabetes and the plausibility of that argument.

Yes, there is a biological rationale for that. I’m not sure I’m ready to explain it thoroughly here, but it relates to the components that are in human milk and their relationship to development of physiological function, immune status and reaction to antigens.

If anyone else wants to go further than that, be my guest.

VICE CHAIR KLEINMAN: That’s an absolutely fair summary. But one of the things
that I think is a little bit confusing is the absence of any relationship to prediabetes, yeah type 2.

You'd expect that those same markers would be present in type 1 in advance of that disease expressing itself. So we might want to pay a little bit more attention to that, as we put this together, because you'd expect insulin resistance, glucose intolerance, A1Cs.

They don't -- you know, they rise gradually both in type 1 and in type 2.

MEMBER DEWEY: Uh-huh.

VICE CHAIR KLEINMAN: Just a point of -- for further discussion in the statement.

MEMBER DEWEY: That's a great idea. I think we will take that up.

And then your fourth question was regarding peanut exposure in the first year of life, and I think the question was, are there infants who are risk from that exposure because they are at high risk to begin with?

Now, I am, again, going to defer, I
think, to the clinicians, but my understanding is that those with a family history are usually advised to be under the sort of supervision of the health care provider when they first introduce that allergen, to be careful about that.

So do you want to go further than that?

VICE CHAIR KLEINMAN: No. I think that’s absolutely right, and the studies use test -- use individuals, infants, who have strong family history, so these are the highest-risk infants. So presumably, if they pass this test, everyone else who is at lesser risk isn’t going to be put at greater risk as a result of the introduction.

So that -- is that your question?

MEMBER MATTES: Yeah, yeah, that’s --

VICE CHAIR KLEINMAN: Yeah.

MEMBER MATTES: -- exactly my question. So the follow-up is does the recommendation need to have that caveat in it, or does that group of high-risk people sort of fall out of the definition of the healthy population
that we’re making recommendations for, and so it’s not necessary?

MEMBER DEWEY: Well, I’d like to say we’re not yet at the point of making dietary recommendations. Right now we’re only drafting conclusion statements from the evidence. How to put all this together into a recommendation is the next challenge.

Yeah. There was one more question from Rick --

VICE CHAIR KLEINMAN: Uh-huh?

MEMBER DEWEY: -- and that had to do with sugar-sweetened beverages and why were those related to overweight or obesity and not to the continuous markers of body size or composition?

I think that the strongest evidence we have is from one very large study where the outcomes were dichotomous only, and so that’s why we felt comfortable saying that. We didn’t have the same amount or strength of evidence for the direct continuous measures of weight for height or BMI or anything like that.
That’s my recollection. I’d have to go back to that paper and look at it again, but that was what I remember. Any additions to that?

MEMBER MAYER-DAVIS: So is this on? They’ll pick it up. Just start talking. I can start talking. Okay. So back to the -- so this is Beth Mayer-Davis. So, Kay, I have a question to follow up to Rick’s about type 1 diabetes. It’s not specified in the question itself, but I wonder if, in your look at infant feeding with regard to type 1 diabetes, you were looking also as the occurrence or appearance of diabetes autoimmunity?

MEMBER DEWEY: What was that?

MEMBER MAYER-DAVIS: The appearance of diabetes autoimmunity, markers of diabetes auto-antibodies, as a prelude to development of Type 1 diabetes, because that’s where some of the mechanism comes in, in answer to your question, Rick. And there is some literature on that.

MEMBER DEWEY: Oh. Darcy is quicker than me. I’m looking here.

You’re shaking your head, so those
markers were not --

MS. GUNGOR: No.

MEMBER DEWEY: Okay. So it was only
the other ones that we defined. Yeah. And again,
that was just done by the Complementary Feeding
TEC, and all of those definitions of outcomes are
in those published papers.

MEMBER SABATÉ: Yes. Joan Sabaté.
Regarding the timing and regarding the types of
foods and the outcomes that you have examined,
basically anthropometrics, biological measures of
fatty acids and minerals so on and so forth, and
also allergy, what was the outcome measured of
these studies?

I mean, within the 24 months, including
to the childhood, in adolescence or in adulthood,
or all this above?

MEMBER DEWEY: The age of outcome
assessment, if I'm correct, varied, depending on
the outcome domain. So if I remember correctly,
micronutrient status was generally the more short
term within the first two years of life.
I'm looking at Julie. She remembers.

Growth, size and body composition went up to -- was it 18 or adulthood? Development went, I think, as far as -- was available -- atopic and allergic disease went all the way to adulthood?

Is that right?

DR. OBBAGY: Yes.

MEMBER DEWEY: Up to 18. And bone health?

DR. OBBAGY: Eighteen.

MEMBER DEWEY: So most of them went pretty long term. But the evidence base or may not have been very strong out of those longer-term time points.

VICE CHAIR KLEINMAN: Are there other questions?

CHAIR SCHNEEMAN: So given the number of conclusion statements where you've had, let's say, insufficient evidence or no evidence, I'm interested to know, is the subcommittee working on the research agenda and particularly prioritizing some of the most critical needs as far as this
research?

MEMBER DEWEY: Yeah. We are trying to keep track of research recommendations as we go. From the previous reviews that have been published, they also did a good job of summarizing the research needs. So that's kind of already there.

It's going to be a huge list, as you can imagine. So prioritizing them is something that I feel we need to discuss. In the context of dietary guidelines, it might revolve around not necessarily one of the most interesting questions but which ones might have the biggest influence on what we advise people to do.

So if there's already compelling evidence from outcomes X, Y and Z for, let's say, breastfeeding, well, do we need to go further than -- and find outcomes -- you know, other outcomes to add to that or not?

Whereas for some of the other dietary recommendations for this age group, there's almost nothing, and in those cases, we may not know what to say at all, and so in that case, it might be a
higher priority.

So I'm just thinking out loud, actually, about would you prioritize in this particular situation.

VICE CHAIR KLEINMAN: All right. So I think adults need to have a little break, and we've been going for over an hour and a half, so we're going to take exactly 10 minutes. Get up and stretch or do whatever else you need to do.

And then we’ll return and hear from Sharon Donovan and the Pregnancy and Lactation Subcommittee report.

Thank you. So 10:48.

(A short recess was taken.)

DR. STOODY: Thank you. Thanks for joining us again after the break. I do want to just make a quick announcement. We are in a multistory building, and sometimes fire alarms do happen, so if you hear one, please hold tight. We are told if we are asked to evacuate, we’ll hear an announcement.

Sometimes they just evacuate the floor
that's involved and the floor above and below, so
if that is to happen, we'll hear an announcement,
and the exit is just right there at the top of the
stairs.

And thank y'all. I know several
have -- that is the preferred kind of in-and-out
for the meeting, if you can help that happen, just
to help kind of minimize some of the distraction
here at the front of the room.

So just a quick announcement, and I'll
turn it back over to the Committee.

VICE CHAIR KLEINMAN: Thank you very
much, Eve. I'm going to turn it over now to Sharon
Donovan, and she's going to summarize the work of
the Pregnancy and Lactation Subcommittee.

MEMBER DONOVAN: Okay. My
microphone's on. So my name is Sharon Donovan, and
it's my pleasure to present on behalf of the
Pregnancy and Lactation subcommittee. If I can
have the slides, please.

VICE CHAIR KLEINMAN: We need to have
the slides brought up, please.
MEMBER DONOVAN: So I’ll go ahead and start talking while that’s coming. So this shows the subcommittee members, and I’d like to thank them all for all of their hard work, on our weekly call, and on the work between the calls.

So my goal today will be to discuss the evidence synthesis creating a conclusion for eight reviews. But before we get started, I wanted to just provide just kind of an overview to remind people of the questions that were assigned to our subcommittee.

So there were three major categories. One -- the first was nutrients and supplements in fortified foods, and this could be consumed before and during pregnancy and lactation.

So we looked at up to six months prior to conception, and during pregnancy and/or lactation. We are examining six nutrients, so B-12, folate, iron, iodine, vitamin D, and omega-3s -- they should sound fairly familiar from Kay’s presentation -- and five outcomes, so human milk composition, gestational diabetes,
hypertensive disorders of pregnancy, neurocognitive development of the infant, and micronutrient status of the mother.

So that was our first set of questions. The second relates to dietary patterns during pregnancy and with five outcomes. I’m not going to read all of those, but you can see these are related to the maternal dietary patterns during pregnancy, and three outcomes during lactation, so milk composition, infant neurocognitive development, and postpartum weight loss.

We also had a third area, which was maternal diet and food allergies and atopic diseases in the infant. So as noted, the NESR staff has been working very diligently, and thus far has screened 21,500 articles and extracted the data and assessed risk of bias from 42. And obviously, additional searches and extraction are underway.

So again, just -- within each of these three areas, just to briefly remind you of where we are in the process:
So for folate, we have addressed all five questions. And so the effect of maternal folate from supplements or fortified foods on human milk composition and gestational diabetes was presented in meeting three, and that information is available on the DietaryGuidelines.gov.

Today, I will be presenting on maternal folate from supplements and fortified foods on hypertensive disorders of pregnancy, neurocognitive development of the infant, and micronutrient status of the mother.

As noted, our committee is currently in the process of refining and prioritizing the additional searches for these -- the rest of the nutrients and these outcomes. So you can just do the math to see this would have been quite a number of systematic reviews to address all of these.

So in terms of dietary patterns, today I’ll be presenting a new systematic review on the impact of dietary patterns on human milk composition, and as with the B-24 Project, there were four previous NESR systematic reviews that
were developed as part of the Pregnancy and Birth to 24 Project.

So we examined those, as Kay described. We looked at the -- we ran through each of the statements. We also looked at any papers that have been published since January 2017, which was the end of the these reviews, to see whether any primary research -- and we also looked at existing systematic reviews published since that time to see whether they caught any papers, mainly with an eye to has there anything really been published in the last two years that would impact the conclusions made in those systematic reviews?

And as with B-24, we decided to accept those existing reviews, NESR reviews, and so I’ll review those outcomes. Currently, we’re looking at the dietary patterns on gestational weight gain, postpartum weight loss, micronutrient status, and infant neurocognitive development, and the plan is to present those at meeting five. Also underway is the question on maternal diet and food allergies and atopic diseases, which will also be presented
in March.

So jumping into our first folic acid questions, what is the relationship between folic acid supplements and/or fortified foods consumed before and during pregnancy on the risk of hypertensive disorders?

So just as a reminder, the definitions that we’ve used for dietary supplements, basically from the Dietary Supplement and Health Education Act, so products other than tobacco that is intended to supplement the diet. And fortification, again, the FDA definition -- the deliberate addition of one or more essential nutrients.

So briefly, you’ve seen the layout for the analytical framework. So in terms of folic acid, our interventions and exposures were exposure to and including intake of folic acid from supplements, fortified foods or the combination, and the comparators were a different level of exposure, including no exposure from supplements, fortified foods, or a combination.
In this case, the population was the women before and during pregnancy, either healthy or at risk for chronic diseases, and in this case, hypertensive disorders of pregnancy.

We had intermediate outcomes that we examined, including blood pressure and proteinuria, and then we have the longer-term outcomes of eclampsia, preeclampsia, and gestational hypertension.

Summarized at the bottom are the key confounders, and most of those are ones that we’re including in all of our systematic reviews. We also have other factors to continue -- or consider for the hypertension -- hypertensive disorders, which include physical activity and substance abuse and gestational age.

So this search was done in combination -- oh, I’m sorry. This one. This search was -- actually, it was. I’m sorry. So this was done in combination with the search for folic acid and hypertension and gestational diabetes, and as I mentioned, gestational diabetes
was presented at the last meeting.

So we screened 622 articles and we included eight related to hypertension, and you can see on the right that the included articles were three RCTs, two non-randomized controlled trials, and three prospective cohorts.

And all of them directly asked the question of the relationship between folic acid supplements consumed during -- before and during pregnancy -- and we basically will present later, but we did not find evidence on fortified foods -- in folate in fortified foods.

So describing first the three RCTs, the sample characteristics between 123 and 450, all of these RCTs were conducted in Iran, and two were from the same study. The interventions -- so again, they were 25 in a normal pre-pregnancy BMI. The race and ethnicity and SES were not reported, but again they were all conducted in the same country.

The interventions varied by dose, so .5, 1, or five milligrams of folic acid, and they were all initiated in the first trimester and
continued through delivery. All reported preeclampsia and blood pressure, and some reported other outcomes, proteinuria, eclampsia, gestational hypertension.

So the two non-randomized controlled trials, one was conducted in Italy and one in China, range from 146 to nearly 5,000 subjects. Again, Caucasian, and race and ethnicity in China was not directly reported, nor was SES.

The group in Italy had pre -- this was in a higher-risk group, so these were women who had preeclampsia in a previous pregnancy, and so this will factor into some of our conclusions.

So this was the one thing that you can imagine, with these different studies, they have different levels of exposure, and also in this study, they had 5-methyltetrahydrofolate as a supplement, and they ranged in initiation, but they all went through delivery.

And then the three prospective cohort studies, you can see the n’s. These were in Australia, Canada, and Denmark. The women were
between 20 and 30 years of age, and you can see the race and ethnicity. They range from low to high SES within these countries.

So in these, they actually compared no supplement with a folate or folic acid alone. And they had initiation and duration of various times, so you know, by trying to look at the evidence we’re taking into account when the timing of the initiation and the duration was, and the primary outcome was preeclampsia.

So the summary of the evidence. So none of the RCTs found an association between folic acid supplementation and the incidence of hypertensive disorders of pregnancy, including gestational hypertension, preeclampsia, or eclampsia, and none of the studies compared folic acid supplementation to a control group that had no supplementation. So in these studies, the control did have a low level -- lower level of exposure.

In contrast, when we looked at the non-randomized controlled trials, both found a
significant association between folic acid supplementation from early pregnancy through delivery and reduced risk of preeclampsia and gestational hypertension, compared to controls with no folic acid supplementation.

And you can see, for preeclampsia, significant reduction, both in this case -- both for high risk and low risk, and for gestational hypertension, again, a significant reduction.

And one non-controlled RCT was among high-risk population of women who had previously been diagnosed with preeclampsia.

So when we looked at the three prospective cohort studies, the results were mixed. One found an association with folic acid in the first trimester and lower incidence of preeclampsia, but specifically for women with a higher BMI.

Another found an association -- significant association between folic acid use between 12 and 20 weeks of gestation and preeclampsia, again, in women at high risk, and
a third found no association.

So our draft conclusion statement is that limited evidence suggests that folic acid supplementation during early pregnancy may have a beneficial effect on reducing the risk of hypertensive disorders during pregnancy among women at high risk, either having a history of preeclampsia or a higher pre-pregnancy BMI, compared to no folic acid supplementation.

This conclusion was supported by three -- or two non-randomized controlled and the three prospective cohorts. The studies were all direct in terms of the question, and they were consistent for the higher-risk women.

And as with all of the studies, there were some concerns about risk of bias, precision, and generalizability, particularly for some of the studies that were not done in the U.S.

So there was moderate evidence suggesting that higher levels of folic acid supplementation during pregnancy, compared to lower levels, including no folic acid, does not
affect the risk of hypertensive disorders during pregnancy among women at low risk. So we had a separate conclusion for women at high versus low risk.

And there's no evidence available to draw a conclusion about the relationship between folic acid from fortified foods before and during pregnancy and the risk of hypertensive disorders during pregnancy.

So turning now to the relationship between folic acid supplements and/or fortified foods consumed by the mother before and during pregnancy and developmental milestones, including neurocognitive development of the infants. This is another new systematic review.

So the analytical framework, intervention/exposure were the same in terms of the outcomes. In this case, the population for the outcome is the infant.

So we had infants and toddlers, birth to 24 months, but we also incorporated children and adolescents from two to 18 for some of the
developmental outcomes, and you can see these are listed, because not all of the evidence is available in early childhood.

So for example, we were looking at academic performance, also attention deficit disorder, ADHD, anxiety, depression, and autism. So in addition to some of the developmental milestones, we needed to extend the search criteria.

So with key confounders, some of the aspects that we added to this one were child sex, breastfeeding practices, intensity and duration, and you can see in other factors to be considered, we also looked at -- took into account, a family history of a diagnosis of a neurocognitive disorder.

So we had a total of 1,831 articles that were screened, and six were included. There were actually four studies that produced the six articles. Two RCTs that have three articles. One prospective cohort published in two articles, and one nested case-control. And they all
addressed the question of again, folic acid
supplements consumed during pregnancy on the
neurocognitive outcomes.

So the sample characteristics are
shown, range of 39 to 130, 17 to 37 children, that
were conducted in the U.K., Germany, and then a
study that incorporated participants from three
countries in Europe.

You can see the mothers were 20 to 31,
mostly white and higher SES, and the outcomes of
children for -- were older, so the children in
these studies were between six and a half and eight
and a half.

The interventions, again, varied by
dose. They also had an intervention with or
without fish oil. The initiation was at 14 or 20
weeks gestation and through delivery. And again,
the outcomes are shown below, but we’ll go through
those.

So the one prospective cohort was done
in Norway. This was a very large study. Again,
the maternal age and high SES, and in this case,
the children were assessed at three years of age.

The dose basically was determined from a questionnaire of folic acid supplementation. And they looked at kind of two different phases. So they looked at early, which could be four weeks before conception to eight weeks of gestation, and then those mothers who are supplemented, who reported the folate between nine and 29 weeks of gestation. So we're looking at the two different, early and late.

So the outcomes were language competence and then language delay. The nested case-control was a study from Israel which, in this case, 60 percent were low SES.

They assessed the children between six and 12. The major outcome was AS -- autism spectrum disorder diagnosis, and in this case, the folic acid exposure was assessed by pharmaceutical prescriptions.

So they basically were able to look at the women who were prescribed folic acid or not, and they looked before and during pregnancy and the
duration assessed before and during pregnancy or birth.

So the summary of the evidence that generally folic acid supplementation before or during pregnancy was either not associated with or had a beneficial association with the following outcomes:

So language development. Two articles from the prospective cohort study showed a lower risk of severe language delay in three-year-olds whose mothers consumed folic acid supplements during early pregnancy.

For ASD, the one nested case-control found a significant association between folic acid supplementation before and during pregnancy and lower ASD risk in eight-to-12-year-old children.

So for cognitive development, the findings were inconsistent and no conclusions can be drawn. For social-emotional development, we included one study with concerns and no conclusion could be drawn.

When we looked at movement or physical
development, academic performance, ADD or ADHD, anxiety and depression, there was no evidence on supplementation before or during pregnancy. And developmental milestones and neurocognitive development, there's no evidence on supplementation during lactation and/or intake of folic acid from fortified foods consumed before or during pregnancy and lactation.

So the draft conclusion statement:

Limited evidence suggests folic acid supplementation during early pregnancy may be associated with lower risk of delayed language development in the child.

So that, again, the conclusions were based on two studies from one prospective cohort study. The study -- they were direct in terms of the question. We had some issues with consistency, and there were some concerns, again, regarding risk of bias, precision, and generalizability, because it was one study, one prospective cohort study.

There was limited evidence to suggest
that folic acid supplementation before and during pregnancy may be associated with lower risk of autism spectrum disorder in the child.

So again, this was based on the one nested case-control study from Israel. Consistency cannot be assigned, and there were some concerns regarding risk of bias, precision, and generalizability.

Insufficient evidence is available to determine a relationship between folic acid from supplements and fortified foods consumed before and during pregnancy on cognitive development or social-emotional development.

And there’s no evidence on supplements or fortified foods, folate, on movement or physical development of the child, academic performance of the child, and also the ADD or ADHD. So these are supplements and/or fortified foods. And so for these, these are grades not assignable.

There was also no evidence for the relationship between folate supplements and fortified foods performed during
lactation -- pregnancy and lactation on anxiety or depression. So a grade is not assignable.

So basically this was looking at lactation. So there was no evidence available to look at supplements consumed during lactation on development milestones, including neurobehavioral development, and no evidence on fortified foods consumed either during pregnancy or lactation.

So all the conclusions were based on some folate from supplements before or during pregnancy.

So the final folate question from a new systematic review is looking at the relationship between folic acid consumption and maternal micronutrient status.

Again, similar framework, basically if you look at the health outcomes, when we looked at the folate status, we looked at: plasma blood folate, B12, hemoglobin, mean corpuscular volume and red cell -- red blood cell distribution width, were the outcomes.

4,512 articles were screened, of which
there were four -- I’m sorry -- for the -- I’m sorry -- 14 for micronutrient status, and of those 14, there were nine RCTs, three prospective cohorts, one randomized cohort and one uncontrolled before-and-after study.

All of the studies addressed directly the question of supplements consumed before and during pregnancy and lactation and micronutrient status.

So to go through the nine RCTs, they range from a very small study to a study of 189. Three were conducted in Canada, two in the U.S., and one each in Iran, the U.K., Mexico, and France. The women in most studies were between the ages of 26 and 34, mostly Caucasian and high SES, but one study was in lower -- teenaged mothers with lower SES. And in one study, it was conducted in 100 percent iron-deficient anemic women.

So the interventions varied across the nine RCTs, ranging from 300 micrograms to five milligrams of folic acid. And also, one study looked at the methyltetrahydrofolate, and one
One study looked at folic acid. The initiation varied from pre-conception during pregnancy as well as postpartum, and the duration was between one and 12 months. And most included serum plasma or red blood cell folate, and other outcomes, common outcomes, were B12, hemoglobin and mean corpuscular volume.

So the three prospective cohorts -- again, we’re -- you can see the n, conducted in Ireland, Germany and Canada, again, 29- to 30-year-old women, and within these countries, race, ethnicity and SES were not reported.

They looked at folic acid supplementation via questionnaire versus none. And there was various times of initiation and duration of the study. So again, when we’re looking at the literature, we’re trying to take into account dose as well as the timing of exposure. All of these reported plasma folate, and two reported red blood cell folate, and one incidence
of folate deficiency.

    The retrospective cohort -- Sorry. I think I said that wrong before. The retrospective cohort was conducted in Turkey. They compared zero versus 400 micrograms per day of folic acid. They initiated pre-conception, but the timing of assessment varied by the participant. So they all -- they didn’t have a specific time point.

    The outcomes, again, folate, hemoglobin, and incidences of folic deficiency.

    And there was a -- one uncontrolled before-and-after study conducted -- a small study conducted in Japan, and there was a limitation of -- was -- that was not a lot in terms of the participant characteristics, other than all from the same SES.

    So these were women that they gave a supplement of one milligram per day of folic acid, and then they were each -- sort of -- their own control. They initiated this anywhere between three and 25 weeks postpartum, and the duration was four weeks.
So, all but one study found a significant association between folic acid supplementation and at least one outcome measure. So, nine of 13 found positive association between folic acid supplementation and plasma or serum folate.

Nine of the 10 found positive association between supplementation and red blood cell folate. And two of five reported positive association between folic acid supplementation and hemoglobin. And there was no association found between folic acid supplementation and these other measures that we had included.

So, based on that, we’ve drafted a conclusion that strong evidence suggests that folic acid supplementation before and during pregnancy is positively associated with folic acid status using the outcomes of serum and plasma and/or red blood cell folate.

The studies, again, were direct and precise and consistent. Some concerns regarding generalizability, but we felt that the evidence was
strong.

There was moderate evidence suggesting that folic acid supplementation during lactation is positively associated with red blood folate and may be positively associated with serum and plasma folate.

There was insufficient evidence available to determine the relationship between folic acid supplements before and during pregnancy or during lactation on hemoglobin, MCV or B12, so grade not assignable.

And no evidence to determine a relationship with folic acid supplemented during this time on red blood cell distribution width. And again, there was no data, no evidence on folic acid from fortified foods before and during pregnancy and folate status.

So that’s the summary of the three, and our final three searches on folic acid. And so now I’m going to turn to the question related to dietary patterns. So, the first is a new NESR systematic review on dietary patterns consumed during
lactation and human milk composition and quantity.

So again, a dietary pattern, as is being defined and used by all of the subcommittees: so, we’re looking at quantities, proportions, varieties, combinations, of the different foods.

So, to set up the analytical framework for dietary patterns, the intervention and exposure is consumption of and/or adherence to a dietary pattern versus consumption or adherence to a different dietary pattern or a different level of consumption.

For example, we’ll discuss studies that have different fatty acids. So, the population for milk composition, again, women during lactation, healthy or at risk of chronic disease, human milk quantity. These are exclusively or predominantly breastfeeding women who are healthy or at risk of chronic disease.

So we had a number of outcomes for human milk composition. And these -- the milk samples were all collected -- needed to be collected after 14 days postpartum, so we were looking at more
mature human milk, not colostrum.

So, we had macronutrients. We have water-soluble vitamins including choline, fat-soluble vitamins, iodine and selenium for the minerals, human milk oligosaccharides, and any bioactive -- of these bioactive proteins. And for human milk quantity, it was assessed in milk collected after 14 days.

So, the search, over 3,000 articles were screened, of which seven were included in the final summary. So, these were three RCTs that produced four articles and two cross-sectional studies.

And I just wanted to mention that, in general, cross-sectional studies are not included, or are excluded, but because oftentimes for human milk composition, that's the only type of data that's available -- there's a lot of RCTs or prospective cohorts -- so we made a decision, and this had been previously published and was open for public comment. So that is just one difference when we're looking at human milk.
So, all of them address the relationship between maternal dietary pattern during lactation and either human milk composition or quantity. So, the three RCTs, again, relatively small studies, seven to 15 mothers, conducted in the U.S. and Canada, 29 years of age, and SES and race and ethnicity not reported.

So, the initiation between six weeks and six months postpartum. The durations were four to 14 days. And they were reporting different -- varied patterns. So carbohydrate, either lower carbohydrate or higher fat versus within the acceptable macronutrient distribution range, or the AMDR. Another looked at higher fat, and a higher carbohydrate and lower fat, and another, higher fat versus consumption within the AMDR.

So, you can see the various outcomes. Most of the studies reported outcomes on fatty acids, and one for B12.

So, for the cross-sectional studies, these were conducted in the U.S. and Canada. They
were, on average, 30 -- nearly 30 -- I’m sorry -- U.S. and China. The moms in the U.S. were highly educated and in China, high-middle income, and within the U.S., the race/ethnicity, reported mostly white.

So, initiation, between 21 days postpartum and six months, and nine and a half months postpartum. So, these were ones that looked more at the overall dietary patterns. So, the study in the U.S. compared milk composition with vegan, vegetarian, and non-vegetarian mothers. And the study from China basically divided the mothers into four different dietary patterns. So, it’s mushrooms, meat, seafood; soy, nuts, dairy; fruits, vegetables; and then grains, potato, beans and eggs.

So, summary of the evidence. So, one cross-sectional study assessed the relationship between maternal dietary patterns and total fat levels in human milk and found no association.

Three RCTs assessed the relationship between maternal diet based on macronutrient
proportions and total fat level in milk. Two found a positive association — positive relationship between greater than 35 percent of energy from fat and total fat in human milk, and one study found no association between macronutrient proportions and maternal diet and total fat.

So, the draft conclusion statements for total fats is that insufficient evidence is available to determine the relationship between dietary patterns consumed during lactation and total fat in milk, and there’s limited evidence to suggest that maternal consumptions of diets higher in fat during lactation is related to higher total fat, with a grade of Limited.

The studies were consistent, but there were concerns about precision, generalizability, and consistency, and we had a long conversation yesterday, because these are also being used for other outcomes that — probably measuring fat in human milk is one of the most difficult components, because some studies were measuring during the fed state versus the fasting state, and because the
content of milk differs from fore-milk to hind-milk, so within a single feeding, if they’re just taking a single sample or not a full breast expression or sampling over 24-hour periods, all of these things can really affect the composition. So that was, you know, some of the concerns that we have about the precision.

So two cross-sectional studies and three RCTs assessed the relationship between maternal dietary patterns, including based on macronutrient proportions and levels of saturated fatty acids, MUFAs and PUFAs, and there were mixed results.

So in terms of saturated fats, MUFAs and PUFAs, there’s limited evidence to suggest the maternal dietary patterns during lactation, including diets based on macronutrient distributions, are related to the relative portions of saturated fat, MUFAs and PUFAs.

And we meant to very specifically say relative proportions, because studies also presented concentrations, and they were not
effects on concentrations, there were primarily
with the proportions of these fatty acids. So
again, some concerns about risk of bias and limited
precision and generalizability were some of the
cconcerns the committee had.

So, one RCT assessed the relationship
between maternal diet based on macronutrient
proportions and milk quantity, and there was no
association.

Also, there was one that looked
at -- one RCT -- on the relationship with total
protein levels in milk, and there was no
association. And the last, one cross-sectional
study assessed the relationship between maternal
dietary patterns and B12, and this was the study
that compared the vegan, vegetarian and
non-vegetarian, and while there is no association
with dietary patterns, we found that 56 percent of
the vegan women were taking a B12 supplement, and
so we thought that they were really kind of unable
to determine the impact of dietary patterns on B12.

So, in terms of the draft conclusion
statements, on quantity, there’s no evidence available to determine a relationship between dietary patterns and milk quantity, and insufficient evidence to determine a relationship on maternal diets differing in macronutrient distribution during lactation and milk quantity.

Again, similar -- so for total protein, no evidence for dietary patterns and no evidence for dietary patterns differing in macronutrient composition. And for B12, again, insufficient evidence is available to determine the relationship between maternal dietary patterns during lactation and vitamin B12 concentrations in human milk.

So, there were no studies found that assessed the relationship between maternal dietary patterns and human milk levels of these other nutrients that were part of our framework, so our water-soluble vitamins, fat-soluble vitamins, iodine, selenium, human milk oligosaccharides or bioactive proteins.

So, I’m not going to read all these, but
basically these are the draft conclusion
statements that there was no evidence, and so all
are grades not assignable.

So now I just -- we'll go through
summarizing the results from the existing NESR
reviews. So as was mentioned, as part of the
Pregnancy B-24 project, there were four systematic
reviews that were conducted that are pertinent to
the Pregnancy and Lactation Subcommittee.

So, the first was the relationship
between dietary patterns during pregnancy and the
risk of hypertensive disorders during pregnancy,
and the second was risk of gestational diabetes.

Then there were two systematic reviews
looking at dietary patterns during pregnancy on
infant outcomes, so gestational age at birth and
birth weight standardized by gestational age and
sex.

So we are, as I mentioned, adopting the
existing reviews. But new protocols are posted on
DietaryGuidelines.gov. So again, if you’re
interested in the complete documentation, they’re
available at DietaryGuidelines.gov.

In addition, just to acknowledge this was the member of the Pregnancy Technical Expert Collaborative, or TEC, who worked on this and drafted the conclusions. And these four systematic reviews, as with the ones that Kay mentioned, were published in the American Journal of Clinical Nutrition in 2019.

So, the two maternal outcome systematic reviews were combined in one paper, and the two infant systematic -- pregnancy outcome, birth outcomes, were in another. So you can not only review the actual results of the systematic reviews on DietaryGuidelines.gov. But, you can also refer to these manuscripts.

So just to briefly review the evidence, so for the first, what is the relationship between dietary patterns during pregnancy and the risk of hypertensive disorders?

So, this systematic review included eight studies from four cohorts and one RTC, and this was over a 37-year range of evidence. So,
I’ll just reiterate, and I mentioned before what Kay mentioned, but we also did then look to see what was published after January 2017 in order to make our final decision on whether we would go ahead and accept the existing reviews.

So for these questions related to dietary patterns and risk of hypertensive disorders, the limited evidence in healthy Caucasian women with access to health care suggest the dietary patterns before and during pregnancy higher in vegetables, fruits, whole grains, nuts, legumes, fish and vegetable oils and lower in meats and refined grains are associated with reduced risk of hypertensive disorders during pregnancy, including preeclampsia and gestational hypertension.

Not all components of the assessed dietary patterns were associated with all hypertensive disorders. So limited -- the grade was limited.

Evidence is insufficient to estimate the association between dietary patterns before
and during pregnancy and the risk of hypertensive disorders in minority women and those of lower socioeconomic status. So, grade not assignable.

So, the relationship between dietary patterns during pregnancy and gestational diabetes. This was -- included 10 prospective cohorts and one pilot RCT, again, collected between publication January 1980 and January 2017.

So, this systematic review concluded there was limited but consistent evidence suggesting certain dietary patterns before pregnancy are associated with a reduced risk of gestational diabetes.

These protective dietary patterns are higher in fruits, vegetables, whole grains, nuts, legumes and fish, and lower in bread and processed meats. Most of the research was conducted in healthy Caucasian women with access to health care.

Evidence is insufficient to estimate the association between dietary patterns during pregnancy and the risk of gestational diabetes. So, again a conclusion on diet before pregnancy,
but not actually during pregnancy, so grade not assignable.

So, in turning now to the infant outcomes. The relationship between dietary patterns in pregnancy and gestational age at birth. There were 10 prospective cohorts and one RCT, again, over the same time range.

So, limited but consistent evidence suggests that certain dietary patterns during pregnancy are associated with lower risk of preterm birth and spontaneous preterm birth. Protective dietary patterns are higher in vegetables, fruits, whole grains, nuts, legumes and seeds, and seafood, for preterm birth only; and lower in red meat, processed meats, and fried foods. Again, noting a limitation, most of the research was conducted in healthy Caucasian women with access to health care.

And this is kind of the opposite, that the evidence was insufficient to estimate the association on dietary patterns before pregnancy and gestational age at birth, as well as preterm
birth.

So, the last relationship between dietary patterns during pregnancy and birth weights, standardized by gestational age and sex, there were 18 prospective cohorts, one retrospective cohort, and two randomized controlled trials.

So, the conclusion is that no conclusion can be drawn on the association between dietary patterns during pregnancy and birth weight outcomes. Although research is available, the ability to draw conclusions is restricted by inconsistency of study findings, inadequate adjustment of birth weight for gestational age and sex, and variation in study design, dietary assessment methodology, and adjustment for key confounding factors.

And insufficient evidence exists to estimate the association between dietary patterns before pregnancy and birth weight outcomes. In this case, there were not enough studies available to answer the question.
So, our ongoing work is -- I mentioned that we are refining and prioritizing work on dietary patterns during pregnancy and micronutrient status; dietary patterns during lactation and developmental milestones of the child, including neurocognitive development; and dietary supplements and fortified foods for all the other nutrients besides folate.

So as noted, we’ll review the evidence, grade, and draft conclusion statements for these following questions: dietary patterns in pregnancy and gestational weight gain; patterns during lactation and postpartum weight loss; the maternal diet during pregnancy and lactation on the risk of child food allergies and atopic diseases. And the plan is then to present these at the meeting in March.

So again, thanking the subcommittee members, as well as our support staff, which we would not be able to get through all of this work without all their hard behind-the-work scenes.

So, I will be happy to take questions.
(Applause.)

VICE CHAIR KLEINMAN: That was a great summary. So, any questions from the Committee? Rick?

MEMBER MATTES: Only one this time. Rick Mattes. So, what’s known about the validity of self-reported supplement use during pregnancy and lactation? Is it different from the general population? Can we believe this data more or less than general studies about diet and outcomes?

And in any of these trials, was there objective verification of compliance with a prescribed dose?

MEMBER BAILEY: So, you can get a compliance sometimes in a clinical trial -- this is Regan answering with Sharon, not for Sharon. So, there are ways to look at the supplements by putting PABA in and getting recovery from urine, so that’s one way to test it.

I can’t speak to whether or not that was done in your studies, but I just wanted to make that comment.
MEMBER DONOVAN: I think it’s a great question, and I don’t know if anyone else is aware of studies where they’ve looked at self-reported compliance of pregnant versus non-pregnant women. Obviously, women during pregnancy may be taking supplements more often and may be more motivated, but I don’t think there’s the evidence and -- just thinking offhand -- and if anyone can speak to that in terms of the studies that we reported; I’m not sure that anyone actually confirmed intake of the folate supplements.

MEMBER STANG: Jamie Stang. Yeah. I was on the Pregnancy TEC, and studies for the most part did not report compliance. I know from unpublished work that the compliance rates start out high, and as you go through pregnancy, they drop off, but in terms of actual published documentation of what that compliance would be, I’m not aware of any.

MEMBER MATTES: So just -- this is a comment that may be a good point to add into the discussion of this section.
MEMBER DONOVAN: That’s a great point, and also research needs -- to have better collection of that type of data.

(Off-mic comments.)

MEMBER DONOVAN: Perfect.

MEMBER BOUSHEY: So, this is Carol Boushey. And I’m looking at the analytical framework for the folic acid with supplements. And you don’t have to look at it; you have it memorized. So -- but folic acid and supplements and the fortified foods, before and during pregnancy and lactation.

And one of the studies, I think, had 11 people or 15 or something like that, and you had said it was a small sample size, and in the frequency-of-eating group, we actually did -- went through the process of figuring out sample size so that we could screen out some of the smaller studies.

And I wonder if you might consider doing that for some of your studies, so that you can determine if those studies actually didn’t meet
sample size.

So it wasn’t that you, you know, went crazy over it anyway, but still it might be nice to have that documentation.

MEMBER DONOVAN: Yeah. No. That’s a great point. I remember we had this conversation at the last meeting, and I think -- we also talked about for the RCTs, that, because they’re more controlled, that the n was not as -- necessarily as much of a consideration, but I think -- I don’t remember if this study with 11 was an RCT or -- so I can’t speak to that directly, but I think it’s an excellent point.

And as you could see, the studies varied from 11 to 45,000, so it’s quite a mixed literature that we’re trying to assess and draw conclusions from.

CHAIR SCHNEEMAN: I had a quick -- this is Barbara Schneeman. I had a question, again, going back to the supplementation.

I know that in your protocol, you allowed for multivitamin supplements, and I’m just
wondering then, as you went through the data and
the evidence where you're trying to then look at
the impact of one nutrient, folic acid, how did you
deal with the multivitamin side of it?

DR. DONOVAN: So, I think when we
looked at that then the control group would have
had that exposure without the folic acid. So, we
didn't just look at folate within a multivitamin
supplement alone.

So, if they needed, they could have
other vitamins without folic acid or those vitamins
with the folic acid.

VICE CHAIR KLEINMAN: Any other
comments or questions? Everybody must be very
hungry.

(No response.)

VICE CHAIR KLEINMAN: All right, then.
Any concluding remarks?

CHAIR SCHNEEMAN: My only concluding
remarks would be to thank the subcommittees, you've
covered a lot of information in a -- in actually
a relatively short period of time, and also thank
you to the staff for the work that’s done to help pull this together.

So, I think -- you know, our next subcommittee is doing to be Dietary Fats and Seafood Subcommittee, but I think we’re best to start that after the lunch break, because I know they have a lot to report back on as well. Correct?

So, I’ll just open it up to the Committee. Do you have any general comments at this point before we break, particularly if you start to see things where you’re seeing threads across the different subcommittee work or things that sort of tie these areas together?

(No response.)

VICE CHAIR KLEINMAN: Hunger wins.

CHAIR SCHNEEMAN: Okay. So, we’ll adjourn for now, and then reconvene at one o’clock, and it is important that we start at one o’clock, because that’s for the webinar folks. That’s when we’ll start the YouTube again.

So have a good lunch.

(A lunch recess was taken.)
CHAIR SCHNEEMAN: It’s time to get started, and I think they have the YouTube set up. Just a couple of reminders to the Committee: A couple of people pointed out that they couldn’t hear as well, so when you’re using the microphone, please make sure it’s in front of you when you use the microphone, just to make sure people can hear the questions.

And I just want to repeat something that I said in my opening remarks, that what you’re hearing are summary statements, draft conclusions, and they’re being presented here for the full Committee consideration in their decision-making process.

And the final decisions are what will be in the report. So I just want to, once again, highlight that what you’re hearing about our draft conclusions, summaries of statements.

The committee themselves are looking in much more detail at all of the publications that are being presented. So with that, we’re ready to go to our next subcommittee report, and that’s the
Dietary Fats and Seafood Subcommittee.

Dr. Linda Snetselaar is going to give the report.

MEMBER SNETSELAAR: I want to acknowledge my committee, Dr. Regan Bailey, Joan Sabaté, and Linda Van Horn, who is here by phone, and also our Advisory Chair, Barbara Schneeman.

The NESR, or NESR staff, is implementing protocols for the first two dietary questions that you see on this particular slide, and the topics will be addressed at a future Advisory Committee meeting.

We will be presenting a summary of the evidence, draft conclusion statements, and grades on the three seafood questions today. They are in red. And the remaining questions focus on dietary fats and neurocognitive outcomes, along with dietary fats and cancer.

As a reminder, we are defining seafood in the following manner. It is marine animals that live in the sea and in freshwater lakes and rivers, and seafood here includes fish and shellfish.
And this particular slide is designed to sort of orient you to the three questions that we will be focusing on today during my presentation, and we’re doing this because the first two questions have a lot of similarities and some subtle differences.

The first question is seafood intake during pregnancy or lactation and neurocognitive development of the child. And the second question is seafood intake during childhood and adolescence and neurocognitive outcomes.

As you will see in the upcoming slides, there are many neurocognitive outcomes, and it’s easy to get confused between these two questions and the various outcomes on which we are reporting.

Because the neurocognitive outcomes are varied and most studies did not examine all components of the outcomes, we decided to develop separate conclusion statements for each component.

And then the third question, the seafood question here, is looking at seafood intake during childhood and adolescence and
cardiovascular disease outcomes.

So the first question we addressed was, what is the relationship between seafood consumption during pregnancy and lactation and neurocognitive development of the infant?

And we used NESR systematic review to answer this particular question. As a refresher, here is the analytic framework we used to approach this question. And we did review this framework in detail during the July Advisory Committee meeting.

And in this question, the exposure was assessed in pregnant and lactating women, and the outcome was measured in children, birth to 18 years. This is a reminder of the specific intervention exposure and comparators that we focused on.

The criteria apply to all of our seafood protocols. And the particular item here to note is that studies must measure seafood consumption. So biomarkers of seafood intake, which might include fish oil or omega-3 polyunsaturated fatty
acid supplement studies, or studies that evaluated infant formula with added DHA or EPA were not included.

This flowchart illustrates the literature search and screening results for two systematic review questions related to seafood consumption and neurocognitive outcomes. One question addresses seafood intake during pregnancy and lactation, and the second question, as I noted before, addresses seafood intake during childhood.

There were 25 studies that were included in this review of seafood consumption during pregnancy and lactation and neurocognitive development of the infant, and that’s highlighted in red there.

As a reminder, we decided to develop separate conclusion statements for each neurocognitive outcome. The outcomes shown in blue here are the ones we presented draft conclusion statements for during the October public meeting.

And I’m going to briefly review these
draft conclusion statements that have been previously presented. So as a review from the October public meeting, our subcommittee found insufficient evidence was available to determine the relationship between seafood intake during pregnancy and attention deficit disorder, ADD; also attention deficit hyperactivity disorder, ADHD; and autism spectrum disorder-like traits or behaviors or an ASD diagnosis in a child.

And due to there being no included studies examining the bottom three outcomes, no evidence was available to determine the relationship between seafood intake during pregnancy and academic performance, anxiety, and depression.

The grade was not assignable for all of these outcomes, and that then concludes our review of the statements presented at the last public meeting.

I will now present draft conclusion statements for the developmental domain outcomes for the very same question: What is the
relationship between seafood consumption during pregnancy and lactation and neurocognitive development of the infant?

Our subcommittee reviewed evidence pertaining to four developmental domains, and they are shown here on this slide, and we then drafted conclusion statements for each. This evidence was reported during the last public meeting, but it does bear repeating.

No studies that met inclusion criteria assessed the relationship between maternal seafood intake during lactation and neurocognitive outcomes, including developmental domains in the child.

Twenty-four articles from 18 prospective cohort studies assessed seafood intake during pregnancy and developmental domains outcomes. These studies were primarily conducted in the U.S. and also in Europe.

Maternal seafood exposure was primarily measured using food frequency questionnaires, though the timing, the type, and
the amounts of seafood intake were varied. The
categorization of seafood intake also varied
across studies, so that one study might look at
quintiles, and another study might look at servings
per week. There was a variety of assessment tools
used within each outcome domain. Now I
will focus on the first domain, developmental
domain, cognitive development. There were 20
articles from 15 prospective cohorts which met
inclusionary criteria.

The majority of the studies detected
positive or null associations between seafood
intake during pregnancy and cognitive development
in children five months to 11 years. And then
looking at IQ, or composite intelligence measures,
that was done in children four to 11 years.

Few studies accounted for all of the key
confounders, and there was heterogeneity across
the studies in seafood intake categories used in
analyses and cognitive assessment methods. This
tended to limit the specificity of the conclusion.

So our draft conclusion statements are
moderate evidence suggests that seafood intake during pregnancy is associated with improvements in cognitive development in the child.

The grade here is moderate for specifically pregnancy. No evidence is available to determine the relationship between seafood intake during lactation and cognitive development in the child, and the grade here is not assignable, specifically for lactation.

Next, we looked at the second developmental domain, language and communication development. There were 14 articles from 12 prospective cohorts which met inclusionary criteria. The majority of studies detected a beneficial or null association between seafood intake during pregnancy and language development or verbal IQ in children six months to 11 years of age.

Few of these studies accounted for key confounders, and there was heterogeneity in maternal seafood intake, such as the timing, during pregnancy, the type and the amount of seafood
intake.

Seafood categorization and analysis was varied. Outcome assessment tools and measurements were varied, and the ages of children at assessment was also varied.

So our conclusion draft statements are moderate evidence suggests that seafood intake during pregnancy is associated with improvements in language and communication development in the child. The grade here is moderate, specifically for pregnancy.

No evidence is available to determine the relationship between seafood intake during lactation and language and communication development in the child. So the grade here is not assignable, specifically for lactation.

Then our third developmental domain involved movement and physical development. There were 13 articles from nine prospective cohorts which met the inclusion criteria.

The majority of the studies found either null or beneficial associations between
seafood intake during pregnancy and movement and physical development in the child. Few of the studies accounted for key confounders, and there was heterogeneity in, again, maternal seafood intake, timing, type and amount, and types of movement and physical development examined were varied.

The outcome assessment tools were varied, and the ages of children at follow-up was also varied. Our draft conclusion statement is insufficient evidence is available to determine the relationship between seafood intake during pregnancy and movement and physical development in the child.

No evidence is available to determine the relationship between seafood intake during lactation and movement and physical development in the child, so the grade here is not unassignable for pregnancy and lactation.

Now, for the fourth developmental domain, social, emotional and behavioral development, there were nine articles from six
prospective cohorts which met the inclusion criteria.

There were no apparent trends across studies, since there were mostly non-significant associations. There was a concern for risk of bias, which we cared greatly about, and as we’re working on these questions, we do focus on this, and this risk of bias was due to few studies accounting for all key confounders, differences in measurement of exposure and outcomes, heavy reliance on parental report for most of the outcomes. And it was difficult to determine the relationship due to heterogeneity. And again, maternal seafood intake, the timing, type and amount, the ages of children at follow-up, six months to 13 years, so variable there, and outcome assessment tools varied. The dimension of social, emotional and behavioral development was also varied in these studies.

Our draft conclusion statement is insufficient evidence is available to determine the relationship between seafood intake during
pregnancy and social, emotional and behavioral
development in the child.

No evidence is available to determine
the relationship between seafood intake during
lactation and social, emotional and behavioral
development in the child. And the grade here is
not assignable for pregnancy and lactation.

Moving on now to question two, this
particular question is, what is the relationship
between seafood consumption during childhood and
adolescence and neurocognitive development? And
we did again using NESR’s systematic review to
answer this particular question.

This is the analytic framework we used
to approach this question. This was reviewed in
detail during the July Advisory Committee meeting,
and in that particular meeting, we discussed the
exposure, childhood and adolescence, through 18
years of age, and the outcome was measured in
individuals two years and older.

This flowchart highlights studies
which met the inclusion criteria. Thirteen
studies were included in this review of seafood consumption during childhood and adolescence and neurocognitive development.

Thirteen studies from both randomized controlled trials, RCTs, and prospective cohort studies met inclusion criteria for this review. There were six articles from three randomized controlled trials.

Two randomized controlled trials evaluated fish intake in children between the ages of four and six, and the intervention for both the RCTs consisted of fatty fish meals compared to meat meals three times a week for 16 weeks.

The third RCT, the FINS-TEEN study, was conducted with adolescents 14 to 15 years, and participants in this particular study consumed fish meals compared to meat meals three times a week for 12 weeks.

Outcomes were assessed before and after the trial, and assessment tools tended to vary. There were seven articles from six prospective cohort studies which evaluated seafood intake.
during childhood and neurocognitive development.

These studies were done in the U.K. and Sweden and China and Canada. Seafood intake was reported as oily fish or just fish intake, and the majority of studies assessed fish intake using a food frequency questionnaire.

Outcomes were assessed in children three to 18 years of age, and there were a variety of assessment tools used. For this particular question, no prospective cohort study accounted for all key confounders.

Now I will focus on the evidence which evaluated the developmental domains. The four developmental domains are shown here, along with how many articles. We evaluated outcomes from these specific domains. And the majority of studies were conducted in Northern Europe, particularly in Scandinavian countries.

For the first developmental domain, cognitive development, there were seven articles included in our review. Of these seven, four articles were from three randomized controlled
trials and three articles were from prospective cohort studies.

The four articles from three randomized controlled trials found predominantly null or beneficial effects of seafood, compared to meat meals in children four to six years, and 14- to 15-year-old adolescents.

There were three articles from three prospective cohorts. Beneficial associations were found between child seafood intake at nine to 15 years and cognitive development in children 12 to 18 years of age.

No association was found between child seafood intake and cognitive development at 3.5 years. Our draft conclusion statement then is insufficient evidence is available to determine whether seafood intake during childhood and adolescence is associated with improvements in cognitive development in children and adolescents.

Grade not assignable here for a specific improvement.

Then moderate evidence suggests that
seafood intake during childhood and adolescence does not have detrimental impact on cognitive development in children and adolescents. And here, the grade is moderate relative to no detrimental impact.

For the second domain, language and communication development, five articles were included, three from two RCTs and two from prospective cohort studies.

Evidence from the two RCTs found no effect of fish compared to meat meals on language and communication development at four to six years in primary analysis. The two prospective cohort studies found a positive association between seafood intake during childhood and adolescence and language and communication development and verbal IQ in children 12 to 18 years of age.

Heterogeneity was found in child seafood intake, looking at timing, type, amount and duration, and the age of children at assessment was variable, and outcome assessment tools were also variable in these studies.
So our draft conclusion statement here is insufficient evidence is available to determine whether seafood intake during childhood and adolescence is associated with improvements in language and communication development in those children and adolescents. And the grade here is not unassignable, specifically focusing on improvement.

Moderate evidence suggests that seafood intake during childhood and adolescence does not have detrimental impacts on language and communication development in children and adolescents. The grade is moderate for no detrimental impact.

For the third domain, movement and physical development, there were two randomized controlled trials included in our review. Both randomized controlled trials used the nine-hole peg test as the assessment tool.

In children four to six years of age, intake of fatty fish meals compared to meat meals have predominantly null effects on manual
dexterity and fine motor coordination.

One study found that fish meals had a beneficial effects on fine manual dexterity, and the fine motor coordination only applied in the non-dominant hand.

Due to limited amounts of studies, our draft conclusion statement is insufficient evidence is available to determine the relationship between seafood intake during childhood and movement and physical development in children. The grade here is not assignable.

For the fourth domain then, social, emotional and behavioral development, three studies were included in the review, two randomized controlled trials. One was conducted in four- to six-year-olds, and one in 14- to 15-year-olds, and they did not find a significant effect of fish meals compared to meat meals on change in behavioral symptoms in primary analysis.

In the one article from the one prospective cohort study, there was a null association between seafood intake at three years
and social, emotional and behavioral development in children at four to 13 years of age.

All of these studies used strength -- a strength and difficulties questionnaire, and there was heterogeneity in the ages of the children at intervention, exposure and outcome assessment. And child seafood intake varied in terms of timing, type, amount and duration.

Our draft conclusion statement is insufficient evidence is available to determine the relationship between seafood intake during childhood and adolescence and social, emotional and behavioral development in children and adolescents, and the grade here is not assignable.

So moving on to attention deficit disorder, ADD; and attention deficit hyperactivity disorder, ADHD-like behavior for seafood intake during childhood and adolescence, there were two randomized controlled trials included in our review, and these studies found fish meals three times a week compared to meat meals had a null effect on ADD, ADHD-like behavior at four to six
years, and 14 to 15 years.

It was difficult to determine a relationship due to the inadequate number of studies, and outcome assessment relied mostly on parental report.

So our draft conclusion statement is insufficient evidence is available to determine the relationship between seafood consumption during childhood and adolescence and attention deficit disorder or attention deficit hyperactivity disorder-like traits or behaviors. And the grade here then is not assignable.

No studies included examined autism spectrum disorder as an outcome. Therefore, our draft conclusion statement is no evidence is available to determine the relationship between seafood intake during childhood and adolescence and autism spectrum disorder-like traits or behaviors or autism spectrum disorder diagnosis, and here the grade is not assignable.

Moving onto academic performance for seafood intake during childhood and adolescence,
there was one prospective cohort study included in our review, and this study found a significant positive association between frequency of consumption of meals containing fish at 15 years and higher total school grade at 16 years.

However, it’s important to keep in mind that it’s difficult to determine a conclusion here due to an inadequate number of studies and concern for risk of bias from measurement of exposure and outcome.

So our draft conclusion statement is insufficient evidence is available to determine the relationship between seafood consumption during adolescence and academic performance in those adolescents. And the grade here is not assignable.

For the outcomes of anxiety and depression for seafood intake during childhood and adolescence, there were two prospective cohort studies included in our review.

One prospective cohort study found a significant positive association between greater
fish intake at 10 to 11 years and lower odds of the
diagnosis of internalizing disorder; that included
anxiety or depression at 10 to 14 years.

And then one prospective study did not
find an association between fish intake at 14.5
years and depressive symptoms at 17.5 years. It’s
difficult to determine a relationship here due to
an inadequate number of studies, inconsistent
results, and little information describing
exposure.

So our draft conclusion statement is
insufficient evidence is available to determine
the relationship between seafood consumption
during childhood and adolescence and anxiety and
depression in children and adolescents. The grade
here is not assignable.

No included studies examined
neurocognitive health in adulthood as an outcome,
and therefore our draft conclusion statement is no
evidence is available to determine the
relationship between seafood intake during
childhood and adolescence and neurocognitive

health, which includes cognitive decline, anxiety, and depression in adulthood, and the grade here is not assignable.

That concludes our review of the second seafood question.

And the third seafood question we reviewed was, what is the relationship between seafood consumption during childhood and adolescence and risk of cardiovascular disease, so we’re moving away from the neurocognitive area.

We used NESR’s systematic review to answer this particular question. This is the analytic framework we used to approach this particular question.

This was reviewed in detail during the July Advisory Committee meeting, and in this question, the seafood exposure was assessed in childhood and adolescence through age 18 years of age, and intermediate outcomes were measured in children and adults, while endpoint outcomes were only measured in adults.

This is a flowchart for the literature...
search and screening results for the third seafood question addressing seafood intake during childhood and adolescence and risk of cardiovascular disease.

And there were four studies included. Of the four studies included, two were randomized controlled trials. In both randomized controlled trials, children were 10 to 12 years of age.

The first study provided children with school meals, and that included either 100 grams of oily fish or a cheese salad sandwich five times per week for 12 weeks. The second randomized controlled trial provided schoolchildren with six, seven or eight grams of tuna fish. And the intervention frequency, duration and control conditions in this particular study were not recorded. These studies measured blood pressure and blood lipids.

The other two studies included in the review were prospective cohort studies, and one study assessed fish intake of 10 years using a seven-day food record at baseline, three and six
months, and outcomes were assessed looking at blood pressure and blood lipids.

The other study assessed fish and oily fish intake at 7.5 years. This was done in the late 1930s, and it included a household inventory. And those outcomes looked at were stroke mortality and coronary heart disease mortality, and these particular outcomes were measured during 60 years of follow-up.

Results from the few available studies were not consistent. It’s difficult to determine a relationship due to an inadequate number of studies and serious methodological limitations in some of the studies.

So our draft conclusion statement here is insufficient evidence is currently available to accurately determine the relationship between seafood consumption during childhood and adolescence and risk of developing cardiovascular disease. The grade here is not assignable.

We have completed the systematic review of the three seafood questions, and these now will
undergo peer review, and we will begin drafting this section of the report.

Our subcommittee will now move to examining dietary fats with a series of questions related to that topic, and we will be starting with the relationship between dietary fat and risk of cardiovascular disease.

I want to thank the subcommittee members, and additionally thank the staff for the huge amount of work that goes into doing these systematic reviews, and thank you all for being here today to listen to what we have synthesized relative to this particular topic.

Thank you.

(Applause.)

CHAIR SCHNEEMAN: So if we could have comments or questions from the Committee?

MEMBER DEWEY: Thank you very much. That was very clear and nicely laid out. The question has mainly to do with the seafood intake during childhood and adolescence and cognitive development outcomes.
As I recall, there were three randomized controlled trials, but the duration of those was 12 or 16 weeks, and so the question is whether you think it’s plausible that that’s long enough to create the kind of tissue changes that one might think would be the link between seafood and something in the brain?

And if it’s not long enough, you know, what is the feasibility of addressing that question with randomized controlled trials of sufficient duration? And should we then look more carefully at the prospective cohort studies?

In that situation, I think you said there were three, and that two showed a relationship, and one with the younger age group does not.

So in this situation, the value of prospective cohort studies might be pretty high, and I would like to know what your group felt were the key limitations that led you, I think, to the conclusion of insufficient evidence, despite those positive relationships?
MEMBER SNETSELAAR: Yes. I think we did look at type of study. You’re right. I do think that in many instances -- and we are coming up with some future direction kinds of things, that it would be great to certainly include more prospective studies possibly.

But in addition to that, we as a committee have looked at what are some of the concerns that went into looking at prospective studies and, in addition, randomized controlled trials?

And do we need additional studies that would focus on more consistency among the assessment, the timing, those things, the duration of the study, those kinds of things. So I think everything you’re bringing up is 100 percent correct.

We looked at this and came to a conclusion. I think that one of the things following a list of our conclusions is to work closely with your committee as well and come up with some final conclusions that would incorporate both
ideas from your committee, and the work your
committee has done as well, along with our
committee, has been helpful.

MEMBER DEWEY: If I could just follow
up with that? I don’t think you mentioned it, I
did know that you didn’t find any studies for
exposure from birth to 24 months and where the
outcome was assessed after 24 months of age.

So we actually don’t have anything to
say about seafood consumption in the first two
years of life and developmental outcomes.

MEMBER SNETSELAAR: So that may be a
future direction.

MEMBER DEWEY: Yeah.

VICE CHAIR KLEINMAN: Linda, that was
just great. My question is about, again,
neurocognitive outcomes and the positive results
of seafood consumption during pregnancy. And I
wonder, is there a dose-response in those studies
or was that examined, I guess?

And then a second question was, you
noted that none of those studies adequately
controlled for confounders. And I'm wondering whether the effect diminished significantly when confounders were considered?

MEMBER SNETSELAAR: Yes. I think -- I remember beginning to talk about this question, and then working very carefully on what should our confounders be? Because the more confounders you have, the more likely you are to end up with no results.

And so you know, I think that's an important question. It was just something that came up again and again, but particularly the prospective cohort studies. And then what was your first question again? I'm sorry.

VICE CHAIR KLEINMAN: Whether there is a dosed response in consumption of seafood and the outcome. So, as the exposure increased -- did any of the studies look at increasing exposure and the consequence of that, to lend more credibility to the intervention?

MEMBER SNETSELAAR: Yeah. I think we need more studies on, certainly, increased
exposure, more specifics on the studies we looked at, I'd look to our NESR team to answer that question.

But certainly, exposure is incredibly important. And when you look at the amount of seafood in some of the studies, it was incredibly small.

CHAIR SCHNEEMAN: Dr. Sabaté.

MEMBER SABATÉ: In some of the studies, and I don’t remember exactly which on the slide, as far as answering your question on the dose-response, I mean in some studies it was flat.

I mean, for some studies there was a dose response and others had a U-shape. So the intermediate, I mean, had some relationship at the highest amount -- I mean, lower back to no exposure, so it had a U-shape in some of the studies.

MEMBER BAILEY: There was a lot of variability. Some studies reported fish intake in grams, others reported it in servings, and so it was kind of hard to synthesize how much actually
was the exposure.

MEMBER NAIMI: Tim Naimi, Boston University. Linda, that was a really nice presentation, and I guess my question is similar to Ron’s and related also to the dose-response, but more along the lines of -- for those ones in which you had exclusively observational studies and none of them had all of the key confounders, and you know the confounding is likely to bias in the direction you found, can you talk about giving it a moderate evidence grade, as opposed to a limited one? I guess that’s where I feel a little bit uncomfortable.

VICE CHAIR KLEINMAN: I didn’t push that far, but that’s where I was going as well.

MEMBER SNETSELAAR: I think that’s a very good point, and certainly, as we look at these conclusion statements, again, these are not carved in stone, as Barbara has been mentioning several times.

And I think as we look at these statements and maybe work with some of the other
subcommittees, we may make some changes. So very
good point.

MEMBER BAILEY: For most of the studies
there was a beneficial association or a null
association, and so we really -- I think there was
one study in one subgroup that there was a
detrimental association.

So the vast majority of the literature
was either beneficial or null, which is why we went
with a moderate, because of all of the
consistency -- and the inconsistency.

MEMBER SNETSELAAR: But those
decisions were hard-fought and we spent a great
deal of time thinking about it.

MEMBER SCHNEEMAN: But certainly part
of the point here is from the discussion for the
subcommittee to take the information and consider
the points being raised, also to look where we need
cross-talk between the subcommittees.

MEMBER MATTES: Rick Mattes. Two
questions that are bit more global. So your group,
I think singularly reports effects of positive and
negative, and in this case, there was no
significant effect, which could also be said
there’s no association, which is the way all the
rest of our recommendations seem to read, and I
think we should be consistent.

Either we’re going to say that there’s
effects this way or that way, or we’re just going
to say there’s no association, and that difference
is between the groups now. We --

MEMBER SNETSELAAR: Yes. And --
MEMBER MATTES: -- probably can report
that --
MEMBER SNETSELAAR: Yeah. I think
your 100 percent correct, that we do need to be very
consistent across the subcommittees. And I think
that process is being thought about, and will be
in the works soon.

MEMBER MATTES: One other, in terms of
consistency. So in at least one other subgroup,
where you have -- like your question one you have
all prospective cohort studies. Did you downgrade
trials that only had a single estimate of intake
at baseline and then track for 10 years and look at an outcome, as opposed to trials that repeated, say, a food frequency question or whatever, so you have some sense that that level of exposure was maintained during that 10-year interval or that the response on that question there was reliable.

We held, in another group, a higher standard, and I’m just wondering how you use that?

MEMBER SNETSELAAR: Good question. Can I defer to Joanne?

MS. SPAHN: What was the question?

Specifically address seafood intake during pregnancy.

(Off-mic comments.)

MS. SPAHN: So when we extracted the data, there were maybe a third and maybe a little less than a third of the studies that measured seafood intake more than once during pregnancy, and then during childhood, the tables will indicate whether or not there were repeat measures.

Certainly, the RCTs, you know, were a definitive measure of time. I don’t recall the
childhood having a lot of repeat measures.

MEMBER MATTES: Yeah. I would just
suggest that when you assign the strength, that
that would be a factor that you put into the
consideration.

MEMBER SABATÉ: Joan Sabaté. I think
this is a good point. I recall in the discussions,
and I think there was no studies which had repeated
measures in childhood that I remember.

MEMBER DEWEY: Kay Dewey. I want to
follow up on the comment about -- I think you
mentioned, Rick, the statement that said that
moderate evidence suggests that seafood intake
during childhood and adolescence does not have
detrimental impact, and that’s been one that you
thought you’d just say there’s no association?

Is that the one you were --

MEMBER MATTES: Well, there are --

MEMBER DEWEY: -- referring to?

MEMBER MATTES: -- a couple where there
is a report of whatever the evidence is pro and
whatever the evidence is negative, in no case was
there a significant association in either direction.

In some cases, there was insufficient evidence, but if there’s evidence of no detrimental, is that different from no evidence of association?

MEMBER DEWEY: Right. And so I just want to clarify that we’re talking about the same conclusion statement. And I’m not sure if this is explained, but my understanding is that this was driven in part by the concern about mercury exposure, and that there is the concern about detrimental effects.

MEMBER SNETSELAAR: Uh-huh.

MEMBER DEWEY: Now -- so in -- on one hand, I think having a statement about no harm is useful, but on the other hand, the way you approach that question is different than when you’re trying to show a relationship in the sense of it being a safety kind of study analysis.

And so I don’t know if the studies looked at it the right way in terms of ruling out
harm, which is different from the way you approach it when you are saying that there is a benefit.

So that might be something to look at again. If they did it the appropriate way --

MEMBER SNETSELAAR: Uh-huh.

MEMBER DEWEY: -- I think it's important to say that --

MEMBER SNETSELAAR: Uh-huh.

MEMBER DEWEY: -- and which -- how many did, because if the statement is possible about no harm, that would be extremely useful.

MEMBER SNETSELAAR: Good point.

Thank you.

MEMBER SABATÉ: Again, Joan Sabaté.

The no harm relates to the cognition, not to any other factors. You know we had a series of studies -- maybe 14 or 15, I don’t remember of which only two or three seems to have some relationship that was significant, one with a U-shape included, but many had basically flat, no relationship.

So we can do the conclusion that seems there was none that has a detrimental effect as far
as cognition. And we can do this conclusion. The
no harm relates only to cognition, not to any other
parameter. As far as the studies, especially the
prospective studies, not all, as a matter of fact,
if I remember correctly, many of them were not
originally designed to test the fish effect; it was
mainly to design the harmful effects of mercury.

And by the way, in the second
publication, there was something as far as the
consumption of fish and cognition. So we have some
of the studies of the prospective studies that were
not originally designed for, you know, fish intake
and cognition.

MEMBER ARD: Jamy Ard. So just to
continue on in terms of how we’re describing
certain effects, my initial impression if, after
seeing the words around -- associated with
"improvements in," et cetera, makes me feel like
that’s a treatment effect or that something started
from a deficit, and I don’t know if that is shared
by others, but I just wanted to share that, in terms
of it’s not the same to me as something that might
say it had a beneficial effect or -- I don’t know exactly the other ways that you -- these things are quantified in terms of cognition or language and communication and those types of things.

But that may be something to think about.

MEMBER SNETSELAAR: Good point.

MEMBER NOVOTNY: Rachel Novotny.

This is a little bit out of place, but related to this conversation about language and how we’re reporting -- and it makes me wonder with most of our questions whether we don’t want to consider both protective effects.

At any rate, to consider whether there’s another pass at our questions for some of these things, I’m thinking specifically about -- which I’ve been talking to Sharon about -- the upper limit for folic acid, which was not specifically our question, but it feels like we should say something about that.

So we may need to make another pass at our studies as well to see if there’s something we
could legitimately say about that. I think it's a general question for us as to whether we're considered sort of both ends of the spectrum for many of our questions.

CHAIR SCHNEEMAN: Other comments or questions?

MEMBER BAILEY: Linda Van Horn is on the line, I don't know if she - if we want to give her an opportunity to comment, or if she has anything to say.

CHAIR SCHNEEMAN: Is she on the line? She is mainly listening, so she'll let us know.

(Laughter.)

CHAIR SCHNEEMAN: I think these were all useful comments for the subcommittee to take back and look at.

MEMBER SNETSELAAR: Uh-huh.

CHAIR SCHNEEMAN: And also working with the staff to make sure we do have consistency across the subcommittee. So I think then we'll move to our next subcommittee report, which is the Beverages and Added Sugars subcommittee, and Dr.
MEMBER MAYER-DAVIS: Thank you very much. So first I do want to recognize the great work of the Committee, Drs. Leidy, Mattes, Naimi and Novotny, and Schneeman, to say nothing of, of course, the NESR staff that just continues to amaze us every day.

So let’s see. I’m clicking the clicker, but see myself instead of a slide. That’s not really that I want to see, actually. So let’s see. What do I need to do here? Oh, that’s way farther than I need to be.

Let’s see.

(Pause.)

MEMBER MAYER-DAVIS: All right. There we go. So let me just overview what we will go over in this session today. This is just a brief summary of the questions that we addressing in this particular committee, questions related to non-alcoholic beverages, added sugars and alcohol.

So we have completed our work towards our draft conclusions for birth weight
standardized for gestational age and sex as an outcome related to non-alcohol beverages underway; a set of questions related to various non-alcoholic beverages in relation growth, size, body composition, and risk of overweight and obesity, for which there’s been a screening of some 17,000 articles and 214 articles identified to be included for that set of questions, with 70 articles currently under review for a subset of questions that are focused on milk.

I won’t be presenting details on that particular piece today because that is -- we are in the midst of that effort. Also underway are questions related to added sugars and risk of cardiovascular disease, screening is underway relatively early on for that with 5,000 articles screened.

Coming up next are questions related to added sugars and risk of type 2 diabetes and also outcomes of growth, size, body composition, and risk of overweight and obesity.

We are also working on questions
related to alcohol and all-cause mortality, and I will be presenting some more information about that today, as well as presenting information about non-alcoholic beverages and birth weight.

So that’s the overview. So now we will focus on this particular question that you see here on the screen. What is the relationship between beverage consumption during pregnancy and birth weight standardized for gestational age and sex? And this is approached via the NESR systematic review process.

This is our analytic framework, and I do want to take a moment on this, because there are actually quite a large number of decisions embedded in this analytic framework that took a fair amount of time to sort through and that follow for much of our work for a range of questions.

So you’ll see in terms of intervention and exposure, you know, we set out these various sub-types of beverages, and you can see the list here. We’ve shown this before. That’s the same list that we’ve had.
The comparator is something that I want
to highlight, because that does impact on the
studies that we review. So for our comparator,
we’re looking at differences in amount of the same
beverage consumed, which could include milk
consumption, of a particular beverage, or versions
of the beverage diluted with water.

We also consider as a comparator a given
beverage versus a solid form of that same food,
broadly speaking, a given beverage versus water.
And then specifically we are looking at
sugar-sweetened beverages compared to low- or
no-calorie sweetened beverages, and we’re looking
at dairy milk with different amounts of fat.

So this provides the scope, really, of
what we’re doing. Otherwise, you know, if you just
have beverages with no clarity with regard to
comparator, you would not be able to go through this
in any kind of coherent manner.

So then for outcomes for this
particular question, we’re looking at birth weight
that could be presented in a continuous fashion or
in categories, small for gestational age or large for gestational age, or birth weight for length.

The population then for exposure would be women either before or during pregnancy, and then the outcome is infants at birth. You’ll see key confounders here: child sex and gestational age, maternal age, race/ethnicity, SES, and a variety of additional confounders listed there.

Other factors that are considered are total energy intake -- that definitely becomes important in a good amount of this work -- and then a variety of other variables related to other components of diet, as well as parity, medications, and supplement use.

So for the question here related to beverages during pregnancy and birth weight, these are the numbers of studies. We started out with some 7600, and that got pared down through screening of titles, abstracts and then full text, relative to our criteria, and the articles that emerged then for complete review are 19 in number, to be included in our systematic review.
So this is a table that we’ve shown before, just showing how we’re sort of categorizing beverages so that we, you know, can go through this work systematically. What you see highlighted are the types of beverages for which there was a literature available for us to look at.

So milk, low- and no-calorie sweetened beverages, sugar-sweetened beverages, coffee and tea, and plain water are the relevant categories. And we’ll start here with sugar-sweetened beverages and low- or no-calorie sweetened beverages.

Now, for this particular segment of the presentation, our subcommittee opted to provide more detail here than we will subsequently, and the reason that we’re doing that is that we wanted to make sure that it was clear to all of you, you know, really what is the way in which we’re proceeding with this work? How are we looking at the data? What does this really look like?

So we’re giving a little bit more
specifics here, just for that purpose, of providing that kind of an example.

So starting again here, beverages during pregnancy and birth weight, for these exposures there were seven studies, all of which are prospective cohort studies.

And in terms of the exposures across those studies, three of them examined sugar-sweetened beverages independently. Two of the studies examined low- or no-calorie sweetened beverages, independently. And then two of them had a combined category of sugar-sweetened beverages and low- and no-calorie sweetened beverages intake.

The outcomes included continuous birth weight and categorical small for gestational age and large for gestational age. So this is an example of three studies -- and one of the various types of summary tables that we look at, where you can see, for each study, the sample size, the country where the study was conducted, the exposure and the comparator.
For the first study, sugar-sweetened beverage estimated intake in servings per week assessed in the second or third trimester by validated food frequency, representing current intake.

And you can see, glancing through here, there is variability across studies in terms of how the exposure was measured, the timing of the measurement as well. And then the outcomes and whether or not the outcome of birth weight was adjusted for gestational age and/or sex or not.

I need to take a moment for a glass of water here. Excuse me. Sorry about that.

And for these studies, TEI, we’re looking for adjustment for total energy intake. You can see the first couple did not address this at all, but the last adjusted, albeit a step-wise process.

And then you see participant characteristics here, just to give you sort of a glimpse at, you know, what’s considered here. I’m not walking through all this detail. Don’t
worry. That would be not good.

But this just shows sort of a -- some --
a couple of reminder of comments about this study,
and then the actual results for continuous birth
weight and the categorical birth weight, with some
color-coding to identify where statistically
significant findings were available.

Does anyone have a cough drop? That
was an actual question.

FEMALE VOICE: We have one.

(Pause.)

MEMBER MAYER-DAVIS: All right. Back
to pregnancy and birth weight, looking at
sugar-sweetened beverages only, those three
studies. So here we found mixed findings, so very
mixed findings.

So in one study, it was found a greater
intake of sugar-sweetened beverages was related to
higher birth weight. Another study found the
opposite, and then the third study, the
relationship was not statistically significant.

And none of these particular studies
used the same categorical outcome, so there wasn’t a way to compare across. This is a view of risk of bias. Many of the presentations have mentioned an evaluation of risk of bias, and there is this specific tool that’s used that considers confounding.

Confounding is based on the specific key confounders listed in the analytic framework in this case, as I showed at the beginning, and those -- the risk of bias is then classified as low, moderate, serious or critical, and then as well, selection of participants, classification of exposures, deviation from intended exposures, missing data, outcome measurement, selection of the reported result from the paper as a whole, and the most common risk for bias in this particular set, inadequate adjustment for key confounders and inadequate description or definition of the exposures.

So then moving towards the literature that focused on low- and no-calorie sweetened beverages or the combination of those, plus
sugar-sweetened beverage, two studies examined the low- and no-calorie sweetened beverages independently.

One of those studies reported greater intake was related to lower birth weight, and another study relationship was found to be not statistically significant, and neither of those studies examined the categorical birth weight outcomes.

There were two studies that combined those categories of beverages, sugar-sweetened and low- and no-calorie sweetened beverages, and for those, one study reported a greater combined intake in relation to lower birth weight.

Another study reported greater intake related to higher risk of small for gestational age, and then the third study reporting a relationship between combined intake and small for gestational age that was not statistically significant.

So our conclusion then for this particular question is here: insufficient
evidence is available to determine the relationship between consumption of sugar-sweetened beverages or low- and no-calorie sweetened beverages during pregnancy and birth weight outcomes, so the grade is not assignable.

Moving then to the question of beverages during pregnancy and birth weight, focusing now on dairy milk, there were six studies that assessed dairy milk intake: one RCT and then five prospective cohort studies. And the exposure was commercially available dairy milk of varying fat and sweetener content.

The outcomes here were five studies that assessed continuous birth weight and three studies that assessed categorical birth weight outcomes.

In terms of findings, four studies found greater milk intake related to higher birth weight. One study found lower milk intake related to higher birth weight. With the outcome of SGA, one study found greater dairy milk intake related to lower risk of SGA. Another study found the
relationship to be not statistically significant.

A study that looked at large for gestational age did not find a statistically significant association. And then a study that looked at low birth weight, greater milk intake was related to lower risk.

The conclusion statement here was that there was insufficient evidence is available to determine the relationship between consumption of dairy milk during pregnancy and birth weight outcomes, with the grade not assignable.

And I’ll just fill in here a little bit. And I’m not sure on all the details of those studies reviewed, but the risk of bias was considerable across studies, primarily due to concerns with adjustment for key confounders, particularly total energy intake, and also very little evidence or no evidence, and certainly not consistent evidence with regard to dose response. So that’s a little bit of, you know, some background that led us to this particular conclusion.

Turning then to tea, as the beverage of
interest, there were eight studies that assessed tea intake. All of these were prospective cohort studies.

Most of the studies combined tea into a single exposure variable, although some looked at some specific types of tea, oolong, black, green tea, and three of the studies specifically looked at caffeinated tea versus tea that is without caffeine.

Six of the studies assessed birth weight in a continuous fashion. Eight studies looked at categories of birth weight outcomes. And in terms of findings, three of the studies reported greater intake of tea related to lower birth weight. Three studies showed a relationship with birth weight that was not statistically significant.

In terms of SGA, three studies showed a relationship to be not statistically significant, while two studies showed greater tea intake in relation to higher risk of SGA.

In terms of low birth weight as an
outcome, three studies showed a relationship that was not statistically significant. And for large for gestational age, one study showed highest intake level was related to higher risk for LGA.

And so the conclusion here was also insufficient evidence is available to determine the relationship between consumption of tea during pregnancy and birth weight outcomes, grade not assignable.

Moving then to coffee. There were seven studies that assessed coffee intake in relation to birth weight, and again, these were all prospective cohort studies. The exposure generally was average coffee intake.

Three out of those studies examined caffeinated coffees specifically. Five studies assessed continuous birth weight. Six assessed categorical birth weight outcomes.

From three of those studies, greater coffee intake was related to lower birth weight. In two studies, the relationship with birth weight was not significant. For SGA, in two studies,
greater coffee intake was associated with higher risk, and in two studies, the relationship was not significant.

Low birth weight, similarly, two studies, relationship was not significant. One study, greater coffee intake was associated with higher risk.

In terms of our conclusion statement, insufficient evidence is available to determine the relationship between consumption of coffee during pregnancy and birth weight outcomes, grade not assignable for coffee.

And again, in terms of risk of bias, there were considerable concerns with regard to adjustment for key confounders, particularly energy intake, and also the difficulty of this issue of, you know, coffee versus caffeine. Now, that may or may not be important, so we just -- there was overall, again, insufficient evidence, grade not assignable.

Plain water. Again this is intake during pregnancy with respect to the outcome of
birth weight. So there were two studies that
assessed plain water intake, both of which were
prospective cohort studies. The studies looked at
tap and bottled water, and did not include
flavored, carbonated or fortified water by way of
how the exposure was specified.

Outcomes: again, two studies assessed
continuous birth weight and two assessed
categorical birth weight outcomes. In terms of
findings, for two studies, the relationship with
birth weight was not significant, and two studies
found the relationship to not be significant for
the outcomes, categorical of small for gestational
age or low birth weight.

So again, the conclusion: insufficient evidence is available to determine
the relationship between consumption of plain
water during pregnancy and birth weight outcomes,
grade not assignable.

So I’ve mentioned some of these, but
just in summary, across this literature, with
regard to beverage consumption during pregnancy
and birth weight, there were quite a few of these studies where the attrition was greater than 25 percent, which provides a risk of bias in terms of selection.

Total energy intake was considered in some studies, but in many studies was not considered. Some of the samples had poor generalizability with respect to lower socioeconomic status and minority populations.

In terms of concerns regarding exposure, variation in fat or sweetener levels in these different beverages, that information was generally not available. I mentioned a moment ago that about half of the tea and coffee evidence examined only caffeinated versions or the difference wasn’t clear, between the caffeinated and uncaffeinated.

There were a lot of issues regarding exposure definitions and assessment methods and timing of the assessment of intake of those beverages during pregnancy.

Birth weight -- and I hadn’t mentioned
this, and should have highlighted this probably a little bit more -- definitely contributed to some of these conclusions. Birth weight was inconsistently adjusted for gestational age and sex, and actually, our original question specified birth weight for gestational age and sex.

Many studies adjusted for one but not the other. Some adjusted for neither. Again, inconsistency in the outcomes assessed and definitions used. And for some of the studies where there was a statistically significant result, the effect size, in some cases, was quite small, with the practical and clinical significance of that was unclear.

So moving now to alcohol as an exposure. We are working on the question of what is the relationship between alcohol consumption and all-cause mortality? And again, this is via the NESR systematic review process.

We do have a new protocol, compared to what we've discussed about before that's posted on DietaryGuidelines.gov. And we'll talk about it
here, but it is posted as well.

So first, in terms of definition, for this question of all-cause mortality in relation to alcohol consumption, all-cause mortality is defined as the total number of deaths from any and all causes during a specified time period, and this does not include, then, studies of cause-specific mortality; in other words, total number of deaths from a specific cause, CVD, cancer or otherwise. So that’s the outcome all-cause mortality.

So this is the analytic framework, and this -- you know, the alcohol field is one that has a number of unique characteristics that we really thought through carefully with regard to establishing our analytic framework, and so I wanted to highlight a number of things here.

In terms of our intervention and exposure, the primary exposure is average consumption of alcoholic beverages, as well as the pattern of consumption of alcoholic beverages, meaning, for example, number of drinks per drinking day or drinks per drinking occasion.
In terms of comparator, the comparator would be different average alcohol consumption or different pattern of alcohol consumption among current drinkers as a primary comparator.

A secondary comparator would be intake compared to never drinkers. And you’ll notice that former drinkers are not shown here, because there are a whole variety of reasons that people are former drinkers such that that group as a comparator would not be appropriate. So primary and secondary comparators here are important to note.

The population, we’re focusing primarily on adults 21 years and older, which means that if there are studies that, you know, are primarily adults but happen to include some individuals younger than the drinking age, that’s fine, but we’re not looking at studies that would be specifically focused on underage drinking.

And then outcome -- you know, all-cause mortality, I already discussed, and so this again, primarily, adults, 21 years and older. Now, in
terms of key confounders, we’re looking at sex, age, race, ethnicity; some markers of SES we consider to be quite important in this work, as well as consideration of eating pattern or diet quality, physical activity, and smoking. These are our key confounders.

In addition, as a key confounder for average consumption exposure, pattern of consumption would be considered important. And then on the flip side of that, for pattern of consumption as the exposure, average consumption would be important to consider.

In addition, other factors to be considered: total energy intake, ideally without alcohol; and then age distribution of the study sample, anthropometric measurements, hypertension, blood pressure, diabetes, glucose, lipids, medications, as well as family history of chronic disease, and beverage type.

So we started with the standard criteria for study design, publication status, language, country, and health status of
participants, as we discussed earlier, and a little bit more detail here, again, because of this particular topic of alcohol. I already mentioned the exposure, and so it’s important in terms of inclusion criteria that the exposure is that which we’ve defined.

Information on type of beverage will be collected if available, but we don’t exclude a study if that’s not available; it’s just good if we can have it.

And again, in terms of exclusion criteria, data on non-drinker groups, where never and former are combined, say, in an observational study, would actually be excluded, just because of the potential problems and bias with combining and including the former drinker group along with the never drinker group.

So the never drinker group is a secondary comparator, but a study would be excluded if the study includes former drinkers. And the comparator, I’ve already focused on that, so don’t need to repeat that.
And again, in terms of exclusion criteria with regard to the comparator, again, the former drinker issue needs to be considered, and that would be excluded if there was a study where the comparison with never and former drinkers was combined.

In terms of the dates here, we include studies from January 2000 to now, January 2020, and exclude articles published prior to January 2000, and again, I already mentioned about the age of participants.

So we saw from a couple of the earlier examples for this particular subcommittee, the numbers of studies is very high, and so we, you know, have had to really think about prioritizing our remaining work, and what we’re doing is, with regard to added sugars and health outcomes for cardiovascular disease, we are approaching that, building on the 2015 NESR systematic review.

In terms of type 2 diabetes, we’ll be building on the 2015 Advisory Committee report, as well as for growth, size, body composition and risk
of overweight and obesity, and building on the 2015.

For alcohol and health outcomes, we are prioritizing all-cause mortality as the first outcome to be examined. I just discussed about that work underway, and as time allows, we will move then to address as outcomes CVD, cancer, neurocognitive health, as well as growth, size, and body composition.

So that's where we are. Again, thanking the members of the subcommittee, as well as the support staff, doing wonderful work.

Okay. So questions?

CHAIR SCHNEEMAN: Thank you.

(Applause.)

CHAIR SCHNEEMAN: So questions for the subcommittee? Ron?

VICE CHAIR KLEINMAN: That was a great presentation. This is a minor comment, and you alluded to it. It has to do with the outcome of birth weight, and I think you mentioned the small effect size and whether these are biologically
significant so I wonder if it’s worth just noting that when you say higher birth weight studies, either in parentheses or something, within normal reference weight values or something to indicate, these are still normal children.

(Off-mic comments.)

MEMBER MAYER-DAVIS: Correct, and that is the case. So yes, that’s a good comment. So in the report, it will be important to provide that framework. Yeah. Thank you for that. Yeah.

MEMBER DEWEY: Kay Dewey. With regard to the dairy milk and birth weight analyses, one of them was, I think, one of the five studies for birth weight showed a positive relationship, and one didn’t.

I was wondering what the sample sizes were for all those studies, and I know that you gave the conclusion statement as insufficient evidence. I’m just curious about the choice between limited and insufficient, and knowing in our subcommittee when most of the studies are going in a certain direction, we might have chosen limited. And this
one seems to be a case where that might be the
situation. I know you mentioned some important
limitations in -- but one that you mentioned was
adjusting for total intake.

And I feel that that’s one that is one
of those gray areas, because it could be on the
causal pathway. So it’s a little bit different
than a regular comparator. And so I wondered if
you could speak to that and the differing

conclusion.

MEMBER MAYER-DAVIS: Yeah, yeah. So
that -- first of all, it was the case that for those
four studies, there were concerns in terms of risk
of bias and accounting for key confounders was one
of the primary concern.

Total energy intake was a concern, and
you know, the role of total energy in this kind of
situation is always a question. Whether you
consider that to be part of a causal pathway or not
could be debated probably for hours.

So I appreciate that concern. One of
the problems with that literature also had to do
with dose response. So for example, I’m recalling now, although the NESR people will recall better, but I recall now, at least one of the studies where there was a significant effect -- there was no evidence for dose response.

So there were quartiles, so any quartile compared to the first, once you got to the second quartile, that was it; flat thereafter. So that’s just an example of one of the several problems across that literature.

But I appreciate that, and one of the reasons that I could answer the question with that level of detail in terms of that one study is because I looked at that myself, and again, I said, now, let me make sure I remember why we made that decision.

So that was a great comment. And I’m looking over here at Brittany, and I don’t know if you want to add to that. That would be helpful, too.

MS. KINGSHIPP: Sure. Brittany Kingshipp. So I was also just glancing at the milk
literature, the sample size question, and it ranges from the mid 100s up to about 3,000 depending on what cohort they were looking at.

And so there were concerns about the things Beth has noted. Also, as was noted, kind across this body of evidence. That body of evidence in particular had multiple studies with very attrition rates, and so that, combined with inconsistency in whether birth weight was adjusted for gestational age and sex or not, half the studies did, half did not.

The same was true for total energy intake. So what we did is look at total energy intake kind of beyond the scale of a regular confounder, so that we were interested, if they did adjust for it, that’s answering one question. If they did not, that’s answering another question, both of which we are interested in.

And so it wasn’t necessarily that they got penalized if they did not. They just got treated differently in interpreting findings, and so because that was also done inconsistently in
that body of evidence, all of those inconsistencies
kind of snowballed to the point that no clear, even
limited, conclusion can be drawn.

MEMBER MAYER-DAVIS: Any other
questions?

(No response.)

CHAIR SCHNEEMAN: Okay. Seeing none,
I suggest we take a break right now.

(A short recess was taken.)

CHAIR SCHNEEMAN: So we’re now ready
for our last subcommittee report of the day. Is
it -- no, it’s working now. So -- and that will
be the Data Analysis and Food Pattern Modeling, the
Cross-Cutting Working Group.

And so Dr. Regan Bailey will be giving
that subcommittee report.

MEMBER BAILEY: It’s my great pleasure
to do so and represent the people on the committee
and Jamy Ard, Jamie Stang, Tim Naimi, and Teresa
Davis, and supported by Dr. TusaRebecca Pannucci.

Wow, I look tired. It’s a very strange
thing to see your face that big. So today we will
be presenting very summary types of statements, 
draft conclusions of summaries of so much data.

So in your Committees, I’m hearing a constant theme of we have insufficient evidence. Subcommittee seven has nothing but evidence. We have so much data. And we can’t -- we will share it all with you in the report, but what I’d like to attempt to do is to show you some of the highlights, the top-level kind of findings today, and where we’re thinking.

So you’ll see those. And then the remaining work we have to do is, we have to work within our committee with the B-24, as well as the Pregnancy and Lactation committee, to refine some of those questions as they relate to food and nutrient intakes and nutrients of public health concern.

And then the last part, of course, our subcommittee is responsible for the food pattern modeling that will be informed by the evidence that we all have from your committees.

So today, we will focus primarily on
Americans two years and older. So infants and toddlers, the B-24, and pregnant and lactating women aren’t going to be the focus of the data I’m presenting today.

So all of the data that we will be talking about, we have as age groups, by sex, by race/ethnicity, and socioeconomic status, and again, I can’t show you all those, but I’m going to give you some high-level takeaways.

And just a reminder of the analytic framework. So we’re using for the dietary intakes, the NHANES What We Eat in America. At this point, the data I’m presenting today are just from foods and beverages, so the nutrient intake data are not inclusive of dietary supplements at this point.

We’re presenting data on chronic diseases from these sources, and again, this is all posted online, and we’ve gone over it, but just to have it fresh in your mind.

So the first question that we will be presenting evidence on is to describe and evaluate
current intakes of food groups and nutrients. And so we’ll go through these at a pretty high level.

So for fruit, the top contributors to fruit are whole fruit, 100 percent fruit juice, and sweetened beverages. And then in bold, I have the intakes, the mean or average intakes for Americans two and older, so about one cup-equivalent per day of fruit.

For vegetables, vegetables are primarily being consumed as part of burgers and sandwiches and mixed dishes. So less than 50 percent of the vegetables that are being consumed are discrete vegetables.

And if there’s one thing you’re going to hear me say today, again, over and over, is burgers and sandwiches. Okay? So that’s something that really will come through in this data, and that is kind of reflective of the American dietary pattern.

Dairy: So most of dairy intake, about one and a half cups per day on average coming from fluid milk and cheese. Fluid milk intake
decreases with age, and over time, since 2007-08, total dairy intake has decreased in the United States.

Whole grains is coming primarily through breakfast cereals and bars. So we have seen increases in whole grain intake across time, but only 2 percent of Americans are currently meeting whole grain recommendations.

And then protein foods, primarily coming from animal-based sources. In general, it’s adequate for most Americans, except for females ages 12 to 19 and 70 years and older, with about 5.8 ounce-equivalents per day.

The majority of the American population for all groups examined are exceeding recommended energy intake from solid fats and added sugars. The main source of solid fats is burgers and sandwiches, desserts and sweet snacks.

And then in children less than 11 years of age, high-fat dairy is also a significant source of solid fat. So the main source of added sugar is sweetened beverages, desserts and sweets and
snacks, and coffee and tea.

And so I use the ampersand to keep food
groups together. I don’t generally like the
ampersand, but just for clarity. So burgers and
sandwiches together, desserts and sweet snacks
together, coffee and tea together.

So our draft conclusion statement is
that for Americans ages two and older, intakes of
fruits, vegetables, dairy and whole grains are
generally below recommended amounts and have not
changed over time. Intake of total grains and
total protein generally meet recommended amounts.
Okay.

For ages one and older -- because the
food group, we’re looking at compliance with
previous dietary guidelines. When we’re looking
at nutrients, we’re looking at one and older,
because the dietary reference intake age groupings
are one to three. So sometimes you’ll see
two-plus, one-plus, so just for some clarity there.

So 9 percent of children and 58 percent
of males, 67 percent of females have carbohydrate
intakes within the AMDR. Across all age groups, protein intake is within the AMDR.

Protein also has an EAR, and I mentioned that older adults and teenaged females have intakes that are below the EAR. So the proportion of the population with fat within the AMDR is about 60 percent for children and 50 percent for adults, and for all ages, sodium, saturated fats and added sugars are overconsumed.

In terms of nutrients that are underconsumed, we have several, including vitamins A, C, D, E, K, calcium, magnesium, fiber, choline, and potassium. In addition to those, other population groups have nutrients or food components that are underconsumed.

And so we’re going to focus on these a little bit more when we do the last question, question 5, on nutrients of public health concerns. So keep these in your mind. We’re going to come back to them.

And then in young children, retinol, zinc, copper and selenium are overconsumed,
relative to the upper level.

So moving on to dietary patterns and beverage consumption, just a reminder of the analytic framework.

At this point, we have the average HEI, total and component scores, but we are awaiting the distribution of those scores. We’ll be looking at that, as well as food category contributions to total energy intake.

And so where we’re talking about dietary patterns, just a reminder that we don’t have self-reported patterns of intake. So we’re looking at reported intakes relative to the HEI, not necessarily able to categorize patterns as vegetarian or Mediterranean. Okay?

So for children and adults, we will look at beverage intake data in the following ways, and we’ve talked about this, by the population groups, mean intakes, and the percent of energy and nutrients coming from beverages, as well as calories.

Just a reminder of the definitions of
the beverage categories that we’ll be talking about today. We’ve seen these before. Okay. We’ve seen this slide before. Out of 100 points, the American diet is currently at a score of 59.

And what’s encouraging is that it has increased slightly over the last decade, from 56 to 59. And we do see age differences. So young children, two to five, and adults over the age of 65 tend to have higher scores than all other age groups.

So this might look complicated, but let’s walk through it. So all of the ways that the 100 points are divided are around the edge of this spider web here. So if you start with total fruits and start going clockwise, you get higher points for more compliance, whereas if you went counterclockwise from total fruits, lower intakes are associated with a higher point score.

So in an ideal spider web -- I don’t know what these are actually called, but -- and what is called?

MALE VOICE: Radar plot.
MEMBER BAILEY: Radar plot. Okay.

In an ideal radar plot, you would have the whole -- if you had a score of 100, it would be all around the outside. So in this slide, we can see some differences among race/ethnic groups.

So non-Hispanic Asians have the highest HEI score, and those are represented with the color red on the radar plot. And you’ll see differences and -- within certain food categories. For example, look at greens and beans, how more compliant that race/ethnic group is with the recommendations. Non-Hispanic blacks have the lowest HEI score relative to the other groups.

So our draft conclusion one from some of that data is that, while average diet quality has slightly improved, scores are not necessarily consistent with the current recommendations, and we do see differences with sex, age, race, ethnicity and income, but the differences are generally small.

This is where the food categories that are contributing to energy. And the -- I’m just
going to take a second to walk through these,
because I know we’ve looked at them before, but just
to remind you.

So the first bar is for all Americans
two and older. All the different colors represent
the top 10 food categories. And one thing that is
surprisingly consistent is those food categories
do not change. What changes is the proportion by
age group.

So these are food category sources.
You can see that for children two to five, there
is less proportion from burgers and sandwiches
than, say, in adulthood.

And we’ll come back and look at some of
those when we look at how patterns track across
life, but just to give you a sense of food category
source of energy for this context right now.

So our second draft conclusion is that
foods and beverages consumed via mixed dishes, such
as sandwiches, casseroles and pizza, sweets and
snacks, and beverages, contribute about 50 to 60
percent of total energy intake.
Food subcategory source contributions to energy vary by all of the population demographics that we’ve talked about, but for the total population, about five subcategories make up most of the energy, and that’s burgers and sandwiches, desserts and sweet snacks, rice, pasta and grain-based mixed dishes, sweetened beverages, and chips, crackers and savory snacks.

So this is looking at where beverages are contributing to energy intakes. So in general, about 15 percent of energy comes from beverages, and this is specifically among two- to 19-year-olds.

So beverages contribute about 40 percent or more of added sugar in two- to 19-year-olds. The percent of added sugar from beverages significantly increases with age, and so when milk is decreasing, it tends to be replaced with sweetened beverages.

But all is not lost in the beverage category, for 40 percent of vitamin C and D and more than 20 percent of carbohydrates, calcium,
potassium, and magnesium are coming from beverages, mainly milk and 100 percent fruit juice. And not surprisingly, more than 80 percent of caffeine comes from beverages. This is looking at adults. So this is a pie chart showing, of all the beverages’ calories, what specific foods they are coming from.

And the three top sources are sweetened beverages, alcohol, and coffee and tea. And what’s interesting here is there are sex differences. So males have more energy intake in terms of beverage calories from alcohol, whereas women are more likely to have coffee and tea calories, which brings me to conclusion number three.

Calories from total beverage account to 15 to 18 percent of total energy for Americans. Fluid milk as a beverage decreases, starting in early childhood, and intake of sweetened beverages increases.

And beverages account for 40 to 50 percent of added sugars in the diet, and
alcoholic beverages contribute 21 among females
and 31 percent among males of total beverage
calories.

We are probably only about 20 percent
of the way through my slides. So I just wanted to
give you, like, some context. Just take a deep
breath. We’re going to do this.

Okay. So this question asks about how
patterns track across life stages, and ideally, to
answer this type of question we’d have longitudinal
data.

What we have is cross-sectional data,
so we can look at different age groups and try to
deret some trends and some patterns, but we can’t
necessarily say how they track within an individual
or within populations or subgroups.

So we will use this analytic framework,
again looking at food category source, means,
beverage contributions, as well as HEI scores,
across different life stages for two and above.

This radar plot shows you how diet
quality and different components of the HEI changes
by age. So remember, I said the youngest children and older adults have the highest diet quality.

When we put the two- and 19-year-olds together, in blue, that kind of changes the story a little bit, but nevertheless, you can see that older adults have a higher HEI score in what foods are represented in the diets of older adults: things like total vegetables, greens, beans, seafood and plant proteins, as well as fruit, refined grains, and lower added sugars, as we talked about before.

So once children begin to age, their milk intake goes down, and so do scores in the dairy component. You can see that clearly from this plot.

So this is going to be the start of a marathon of slides that look exactly like this, but the title is going to change. And the colors are not always consistent. So if you want me to stop and you want to look at them in a little bit more detail, our safe word in our subcommittee is tangerine.
So if you want me to stop, just say, tangerine, and I’ll know it’s time to stop. Okay.

So this is looking at energy. You’ve already seen this one, so we won’t spend too much time here.

But next, looking at vegetables, and how those change with different age groups. So the green is represented by vegetables, including beans and peas that are not starchy. As I mentioned earlier, less than 50 percent of vegetables are consumed as a vegetable alone.

Chips, crackers, and savory snacks and pizza are a larger source of vegetables for children than for adults or older adults. And mean vegetable intakes tend to increase with age.

Looking at fruit, you can see, as I told you earlier, that primarily coming from, you know, whole fruit, but it does decrease after the age of five, and then pretty much levels off and stays about the same after the age of five.

And 100 percent fruit juice decreases after adolescence.

This is looking at whole grain intake,
and we talked about the mean intake earlier, but chips, crackers, and savory snacks as a source of whole grains decreases, and yeast bread and tortillas increases as a source of whole grains among individuals in older age groups.

So you can just see some of the patterns. The number-one contributors generally stay the same, but the proportions change with different age groups.

So going on to dairy, there is a food category source shift from higher fat among young children to burgers and sandwiches among young and middle-aged adults, and older adults, desserts and sweets and snacks are really a large contributing source to dairy.

This is looking at protein foods. So mean total protein is generally within recommended ranges. We talked about those groups that it’s not. For older children and younger adults, burgers and sandwiches is the main category source, and mixed dishes contribute a smaller proportion of protein to the intakes of older adults.
Looking at added sugars, mean added sugar intake is highest in adolescence and early adulthood. The food category sources here change across the life course. So desserts and sweet snacks are a large contributor for both young children and older adults, whereas in between those age groups, it's really sweetened beverages, so from six to 50.

And for adults, coffee and tea are also a source of added sugar. These are -- coffee and tea are not naturally contained in this, so this inclusive of the additions. I should have made that point earlier.

This is looking at calcium. The slide is set up in just the same way. So high-fat milk and yogurt is the largest contributor among young children, and it shifts to burgers and sandwiches for adolescents and adults. And water makes up a large contributor among adults to calcium intakes.

This is looking at potassium, so milk and yogurt is a large contributor for young children, and that shifts to --
VOICE: burgers and sandwiches.

MEMBER BAILEY: -- burgers and sandwiches. Thank you. Somebody’s awake. All right. And then coffee and tea and vegetables in adulthood, as a large contributor.

This is looking at sodium, and as we mentioned earlier, it’s overconsumed across all life stages, and this is primarily coming from burgers and sandwiches, and that’s pretty consistent across most age groups.

This is looking at vitamin D. And remember, I’m only showing you the highlight reel. Okay? So you can imagine how much data we’ve been looking at.

So vitamin D is underconsumed across all life stages. Again, children are getting vitamin D, similarly to calcium, from high-fat, low-fat dairy and milk, and in adults, it’s burgers and sandwiches.

So this is our draft conclusion statement. There is general consistencies in diet quality seen across life stages. Diet quality is
better among young children and older adults, but
even so, it still does not align with existing
guidance.

Food category sources of food groups
and nutrients differ across life stages. In
particular, in the case of milk and yogurt, after
eyear childhood decrease, and intakes of added
sugar from beverages increase.

Fruit and vegetable intake declines
through adolescence and adulthood, and then
increases among older adults. Intakes of burgers
and sandwiches contribute to most food groups,
nutrients and food components which fall outside
of recommended ranges.

So burgers and sandwiches help
contribute to underconsumed nutrients, because
they’re so ubiquitously consumed, but they also,
at the same time, contribute to those nutrients and
food components that we want to limit, such as
sodium and saturated fat.

Deep breath. Okay. So for the
prevalence of nutrition-related chronic
conditions, we have, as I mentioned earlier, several data sources. What I’m going to do here is a word that I learned yesterday called bookend.

So I’m going to tell you the conclusion, and then I’m going to show you the data, and then we’ll revisit the conclusion as a group to get some input, because there is a lot of information I’m going to give to you, and distilling it into a couple of sentences is very complicated. So we’d really love to hear the Committee’s feedback on what you think are the most salient points to include in this section.

So we are looking at this with a life stage approach, and the colors are simply there to show you that there are certain things, like body composition, that we will looking at in most age groups, cardiovascular endpoints. So we’ll start with young children.

We only have two outcomes in young children. We have body composition. So more U.S. children under the age of 24 months are overweight, about 9 percent, than underweight, based on weight
for recumbent length.

The prevalence of low birth weight and very low birth weight are 8.3 and 1.4 percent respectively. Non-Hispanic black mothers have the highest prevalence of low birth weight babies, and this has increased over time.

We have a different age group for allergies. So based on proxy report, the prevalence of food allergy is 6.6 percent. So this is not clinically confirmed data. It’s -- I felt strange using the word self-report, because it was birth to four.

And I’m just imagining like a little baby trying to tell you -- anyway. So proxy report. So you know, there’s obvious limitations with that kind of data; it’s not clinically confirmed.

So looking at the data that we have available in children in the following categories, the prevalence of overweight is about 17 percent, obesity, 18.5 percent, and underweight, 3 percent.

Prevalence of underweight is higher in
boths than girls and increases with age. However, the prevalence of underweight has decreased over time. The prevalence of obesity is higher in boys than girls. It increases with age, and has increased since 2007 and 2008.

Among girls, the race/ethnic group with the highest prevalence of obesity is non-Hispanic black, whereas among boys, highest prevalence is in Hispanic and Mexican-Americans.

The prevalence of obesity is lowest among children whose head of household has a college degree or higher.

So our draft conclusions for CVD intermediate outcomes: the prevalence of hypertension is 4 percent, and it's higher in males, non-Hispanic blacks, 18- to 19-year-olds, and those with obesity relative to their peer counterparts.

Prevalence of high LDL is 5 percent, and prevalence of low HDL is 15.5 percent. The prevalence of high LDL is higher in non-Hispanic whites, and Hispanic and Mexican-Americans -- you
can see the percentages there -- when compared with non-Hispanic black and Asian youth.

The prevalence of low HDL cholesterol is higher in males, non-Hispanic whites and youths with obesity. So for each chronic health condition we've examined in children, the highest prevalence is among those with obesity.

We have one cancer outcome, and that is leukemia, and you can see the incidence and mortality rate, both of which are higher among boys than girls, and so this is inclusive of birth to 19 years of age, from the SEER data.

In terms of diabetes and prediabetes, we have data on 12- to 19-year-olds, and the prevalence of those combined is 23 percent. This is coming from NHANES data.

Dental caries: So first, looking at two- to 19-year-olds, the prevalence is about 46 percent, and then untreated dental caries is about 13 percent, so this tends to be associated with age.

Again, this is cross-sectional data, so
we can’t say the prevalence of caries increases with age, but the age groups and the prevalence track in the same way.

Hispanic youths have the highest prevalence of dental caries, but non-Hispanic blacks have the highest prevalence of untreated dental caries. The prevalence of both caries and untreated caries is lower among families with a higher income. And there has been a slight downward trend over time for the prevalence of total and untreated dental caries.

Moving on to adults, the overall prevalence of underweight among adults is 1.5 percent. The prevalence of overweight and obesity and severe obesity are highlighted there in parentheses. The prevalence of overweight has decreased, while the prevalence of obesity and severe obesity has increased.

And the prevalence of obesity and severe obesity is higher in women than it is in men. Mean body weight, weight circumference, and BMI have increased over time.
Adults 40 to 59 have the highest prevalence of obesity. Hispanic and Mexican-Americans have the highest prevalence among men, and among women, it’s non-Hispanic black women.

Looking at the data, the overall prevalence of dental caries among adults age 20 to 64 is 90 percent, and 96 percent among adults ages 65 and older.

Women have a slightly higher prevalence than men among 20- to 64-year-olds, but the prevalence converges after the age of 65. Non-Hispanic blacks have the highest prevalence of untreated dental caries, and the overall prevalence of complete tooth loss is 2 percent in 20 to 64, but increases to 17 percent among those age 65 and older.

So looking at cardiovascular, intermediate and outcomes, high cholesterol among adults is 12 percent. Low HDL, 18 percent. Hypertension, 29 percent. Coronary heart disease, 6 percent, and stroke, around 3 percent.
So when we talk about adults in general, we’re talking about 19 and older, but some of the data come from different surveys, so that is why we have 18 and 19 for some of these age groups here.

The prevalence of high cholesterol and low HDL has decreased since 2007-08. Women have a higher prevalence of high cholesterol. Men have a higher prevalence of hypertension, low HDL, CHD and stroke.

Adults 40 to 59 have the highest prevalence of total cholesterol and low HDL. However, adults ages 65 and older have the highest prevalence of hypertension, CHD and stroke.

So some more key findings here. Non-Hispanic whites have the highest prevalence of high cholesterol among women. However, Hispanics have the highest prevalence of high total cholesterol among men.

Hispanics have the highest prevalence of low HDL for both men and women. Non-Hispanic blacks have the highest prevalence of hypertension and stroke. American Indian and Alaska Natives
have the highest prevalence of coronary heart
disease.

The prevalence of hypertension, coronary heart disease and stroke are lower among those with higher education levels, and those with a college degree tend to have the lowest prevalence of these cardiovascular outcomes.

In terms of diabetes and metabolic syndrome, diabetes is prevalent in about 14 percent of U.S. adults; prediabetes, 34 percent; and metabolic syndrome, almost 35 percent.

So men have a higher prevalence of diabetes and prediabetes, but there’s no differences for metabolic syndrome. So the prevalence of diabetes and metabolic syndrome is higher among older age groups.

In fact, 52 percent of older adults have metabolic syndrome. The prevalence of diabetes is higher among those with higher BMIs, and Hispanics and Mexican-Americans have the highest prevalence of diabetes and metabolic syndrome.

In terms of chronic liver disease, we
have two different measures. First is
self-report, which is about 2 percent, but then
looking at ALT and AST, ALT is elevated in about
10 percent, and AST in 16 percent of U.S. adults.

Hispanics have a higher prevalence of
liver disease, high ALT and high AST, when compared
with non-Hispanics. American Indian and Alaska
Natives have the highest prevalence of liver
disease.

Men and women have the same prevalence,
and mortality rates differ. So men have a higher
mortality rate than women. And mortality rates
have increased over time, particularly in men.
And then men age 55 to 64 have the highest mortality
rate from chronic liver disease.

So these are a different data source.

So this is from the National Vital Statistics
System. These are the age-adjusted prevalence
rates for chronic liver disease and cirrhosis, and
you can see that they have increased since 2006 to
2016 in every age group, except for males 45 to 54.
And men 55 to 64 have the highest mortality rate
from chronic liver disease, and the lowest rate is among females 25 to 34.

Moving on to cancer, so the cancer with the highest incidence rate among females is breast cancer, followed by male prostate cancer.

Age group and cancer type with the highest incidence rate is prostate cancer for men among ages 65 and older. Mortality rate is highest for lung and bronchus cancer, and the age group is the highest mortality rate from that cancer is among older adults, 65 and older.

Men have a higher incidence and mortality rates than women across all shared cancer types, and the incidence and mortality rates for every cancer type are highest among individuals 65 and older.

So we’ll talk last in this section about pregnant women. We’re just going to talk about gestational diabetes today, because the 2018 pregnancy-induced hypertension data is just coming into our emails now, so we’ll hold off on that until next time, and just focus on gestational diabetes
right now.

Total prevalence, about 6 percent. It is higher in women who are older than 40. There’s some race/ethnic differences. So non-Hispanic Asians, 11 percent have gestational diabetes. Also very high in American Indian and Alaska Natives and Native Hawaiians and Pacific Islanders.

The prevalence remains relatively stable across educational status, but among those with obesity, particularly Class III obesity, the prevalence is 14 percent.

So older adults, we have two outcomes here, muscle strength and osteoporosis and bone health. So 19 percent of older adults have reduced muscle strength. This is data coming from NHANES.

And there’s really an increase with age. So 48.6 percent of adults over the age of 80 have reduced muscle strength. So the age-adjusted prevalence is not different between men and women. It’s about 19 percent, and similarly, women who are older than 80, it’s slightly higher in women than
men, but not substantially different, so 49 versus 47.

Non-Hispanic Asians have the highest age-adjusted prevalence rate, followed by Hispanics, and then Non-Hispanic blacks have a prevalence that is about 19 percent, and non-Hispanic whites, about 18 percent.

In terms of osteoporosis, it’s estimated that about 11 percent of adults over the age of 50 have osteoporosis, and about 45 percent have low bone mass, and as we know, women are disproportionately affected by both osteoporosis and low bone mass, and that’s amplified here in the last bullet point.

So again, given all of the complexity of the data that we showed you, it was very hard to come to a conclusion statement. So this is a work in progress, and we really want it to be informed by you.

But we kind of started with this large umbrella, to try to be inclusive of all the things that we found, but we’d like to drill down and have
some more specific conclusions. Okay.

We are on the last question. Evaluate nutrients of public health concern. So we’ve talked a lot about intakes of food groups, short/long nutrients, and this will be our last question.

So with nutrients of public health concern, we have tried to use the terminology food component, because there’s things that we’re talking about that aren’t essentially nutrients.

So if you are confused, that is why.

The question was written for nutrients, but we are trying to use food components. So we developed this flowchart ahead of time. That’s where we looked at the data to make decisions.

So sometimes we had dietary data available.

Sometimes we have biomarkers. Sometimes we have clinical outcomes.

So we had a decision tree in place before we looked at the data to try to be as transparent as possible. And I don’t expect you to read that, because it’s very small. It will be
in the report.

So we first started by casting a wide net. We defined underconsumed or overconsumed when a food component was not within the range of 5 percent or higher relative to a dietary reference intake or a quantitative authoritative recommendation, such as a previous dietary guideline recommendation for saturated fat.

Similarly, for overconsumed. Then those are elevated to a nutrient or food component of potential public health concern when supporting data through biomarkers, functional indicators, that these low intakes or high intakes are directly related to a health condition.

Then we are proposing this category called nutrient or food component that poses special challenges. This is a term that was used by the 2005 Committee to identify food components for which dietary guidelines to meet recommendations was challenging.

But we’ve extended this to also include nutrients or food components that pose special
challenges in identifying at-risk groups. And I’ll show you what I mean on the next couple of slides.

So casting our wide net of 5 percent for underconsumed nutrients, there were a number of nutrients that were either in the population or in specific subgroups, noted with an asterisk, that were not well aligned with recommendation, either the EAR or the AI.

So when we next evaluated whether there was a biomarker or clinical endpoint that we could tie low intakes to, we were able to eliminate several nutrients, and those that are listed in bold have previously been linked to a health outcome or biomarker, whereas we still have a few that are listed there in red that are special challenges.

In terms of overconsumption, we’ve already talked about sodium, saturated fat and added sugars, but compared to the UL, young children are exceeding the UL for retinol, zinc, selenium and copper, and you can see those
prevalence estimates in the parenthesis there, ranging from about 6 percent for copper to 50 percent for zinc and selenium.

So then we come to these food components that pose special challenges, and this is where we can also use some of the Committee feedback and guidance. So I mentioned that protein was underconsumed in adolescent and older females.

Vitamin B12 might be a concern in older adults, both dietary data and biomarker. Choline intakes are low relative to the AI for most age/sex groups, after young children. Phosphorus intake is low in 9 to 14-year-olds, as well as magnesium.

So magnesium is low, relative to the EAR, across most age groups. So our analytic summary so far is that nutrient intakes have not changed considerably since the evaluation. Nutrient intake distributions taken into consideration with biological endpoints and clinical outcomes suggest that vitamin D, calcium, fiber and potassium are underconsumed.

Sodium, saturated fat and added sugars
are overconsumed for all Americans ages one and older. We’re still talking, and that’s why we brought this to you today, in terms of the distinction of what is a nutrient of public health concern for some of those remaining nutrients?

In addition to those for all age groups, we’ve looked at this as a life stage kind of approach, and so iron is of particular concern among adolescent and premenopausal females. That’s both dietary and biomarker data.

Older adults seem to be at risk for low intakes of protein, and I showed you the data on the muscle strength, as well as vitamin B12. Adolescents, there was -- this is what we’re calling a constellation of dietary risk.

So this age group has the highest prevalence of not meeting recommendations across most nutrients, and particularly adolescent girls. So protein, folate, B6, phosphorous, magnesium and choline.

And then young children, as I showed you earlier, one to three, have high intakes of
retinol, zinc, selenium and copper, relative to the UL.

So our remaining work I talked about a little bit earlier is what is the role of added sugar in meeting food group recommendations, frequency of eating, looking at beverages, and meeting food group and nutrient recommendations, as well as dietary patterns?

We already mentioned that we're going to be working more with the B to 24 and Pregnancy and Lactation to identify nutrients of public health concern in those populations.

And then finally, we'll end with a few pattern modeling questions. Thank you very much for your time and attention. And I definitely will answer questions, but we also really want to hear from you guys.

(Applause.)

CHAIR SCHNEEMAN: A long presentation. It was very quick, though. So you've heard some questions from the subcommittee, but I think also you have questions or comments for the
member donovan: so thanks, regan. i have two questions, and one is related to the kids in the -- of early ages that are in the upper limits. do we know what food groups are contributing to the high intakes of the zinc and selenium?

member bailey: yeah. we haven't really looked at it that way. i could guess at what i think those food sources are, but i think that's premature.

member donovan: okay.

member bailey: and then, you know, there's a lot of discussion around are those uls the right number? so it might be that the diets are okay, and the uls are often set based on extrapolated data down for children.

so that's why we are calling it, you know -- maybe this is a concern. we certainly didn't want to make a statement without talking to you all, but we are really unsure about to do, and this is just from food alone.
So when we will look at supplements for nutrients, those prevalences are going to increase.

MEMBER DONOVAN: So my second question was related to some of the -- primarily the cardiovascular outcomes, and is there any factoring in of medications that are used to manage hypertension or cholesterol?

MEMBER BAILEY: The way the data are collected is -- so particularly, some of the biomarkers -- no. I mean, we know that a lot of people are on statins. These are the prevalence estimates for a national representative sample of adults.

So there are people who taking medications for hypertension. There are people who taking medications for various things, but they’re in the survey.

CHAIR SCHNEEMAN: So Kay, and then Richard --

MEMBER MATTES: Sure.

CHAIR SCHNEEMAN: -- and then -- and
Steve. So Kay?

MEMBER DEWEY: Thank you very much.

Kay Dewey. So first, I want to just comment that the UL percent is very, very lightly -- it's too low, because we see this problem across the board, you know, in many countries, and so that's just one comment.

So I have three questions. The first is when you're looking at inadequate nutrient intakes, we've talked about the fact that people, at least for adults, tend to over -- underreport their energy intake, and so that might make it look like their nutrient intake is too low.

And I think you've talked about this, but if you'd answer again whether you're attempting any correction for that, or at least a sensitivity analysis that would let you judge, you know, is it really low, or it's just they're underreporting energy?

MEMBER BAILEY: Yeah. We know that there is underreporting of energy, but for nutrients, it's really not well known how
differential that is. We only have recovery biomarkers for a few food components, and so we really can’t make estimates about what other nutrients are low as a result of energy underreporting.

We haven’t really talked about sensitivity analysis. I know there’s been some work done with the survey before, so we might want to look at -- especially the nutrients that we do agree are a public health concern, maybe we could do a sensitivity analysis, trying to exclude energy underreporters, and see what those prevalence estimates would look like.

That’s a really good idea.

MEMBER DEWEY: Well, you know, I was thinking more along the lines of if we assume that underreporting is not the assumption -- not necessarily a the assumption, but for a sensitivity analysis, if you’re assuming that, you can then apply it as a correction factor across the board, just to see which ones would still emerge as being under the EAR, for example.
MEMBER BAILEY: Yeah, but people differentially underreport specific food components, like alcohol or sugars, and those aren’t things that really would be good nutrient sources anyway, so I don’t know that we could have a correction factor.

MEMBER DEWEY: Okay. Well, anyway, something to think about. The second the question is regarding the birth weight outcome you reported, and just in terms of low birth weights, I remember -- and maybe I don’t remember. But do we have estimates for SGA, LGA, and preterm?

MEMBER BAILEY: Yeah. We don’t have preterm right now that I have seen, but we do have all the anthropometrics. We can give you guys all that data, probably be a good conversation to have together.

MEMBER DEWEY: Yeah. And one of the issues to take into account is the multiple births and trends in those, because those drive a lot of those numbers. And then lastly, there was a slide where you had a bunch of nutrients, and then you
crossed them out, and I think it was based on
whether there was a biomarker or some other --

MEMBER BAILEY: Not whether there was
a biomarker --

MEMBER DEWEY: Or --

MEMBER BAILEY: -- but whether that was
linked to low intake. So for example, vitamin E,
there is a very prevalence of vitamin E inadequacy
if you look at the diet, but when you look at the
biomarker, it's less than 1 percent.

MEMBER DEWEY: So when it was crossed
out, it meant that --

MEMBER BAILEY: That it wasn't
confirmed with a biomarker or a clinical outcome
endpoint.

MEMBER DEWEY: But what if there is no
biomarker? Was it crossed out?

MEMBER BAILEY: No, I don't think so.
Like what are you thinking of? We try to be,
like -- all the ones that we were -- special case
that we wanted to talk about, which if we could pull
up that last slide, that might be helpful.
But for a lot of those, they do have biomarkers or no clinical endpoint. Like we have vitamin C from the blood, we don’t see a lot of scurvy. You know, so that’s why that could be crossed out.

CHAIR SCHNEEMAN: So can you go backwards --

MEMBER BAILEY: I put it at the end, so I was anticipating this. Okay. Perfect.

CHAIR SCHNEEMAN: I think she was looking at the table where you listed everything that was --

MEMBER BAILEY: That might take a minute.

MEMBER DEWEY: Okay. So here’s the list, and then if you could explain what were the reasons for crossing the ones that are crossed out? I just didn’t -- you went fast.

MEMBER BAILEY: Okay. So probably not a nutrient by nutrient, but there was not a biomarker that could confirm low dietary intakes were a problem, and there was not -- it was not
related to any clinical or health outcome.

MEMBER DEWEY: Okay. So in some cases, there is a biomarker, but it didn’t show a problem --

MEMBER BAILEY: Right.

MEMBER DEWEY: -- but in other cases, there is no biomarker, so we don’t know.

MEMBER BAILEY: So what are you thinking there’s no biomarker?

MEMBER DEWEY: Well, I’m just -- well, I --

MEMBER BAILEY: Can you go back to the flowchart? There we go. Okay. So we would start with -- there are -- for most things, we have dietary data available. So is that available? Yes.

Are the prevalence estimates within the threshold, is it more than 5 percent of the population or any population subgroup that might have a problem? Yes. Then is there a biomarker available? Yes.

Is there suggested evidence of a risk
supported by a biological or clinical indicator?
No. Like, you know, every path on -- you put the	nutrient through -- each nutrient through this
kind of pathway to see what was available.

MEMBER DEWEY: Okay. So the specific
question I have is, the arrow that goes from "Are
biomarker data available?" And it says, no.

MEMBER BAILEY: Uh-huh.

MEMBER DEWEY: And it goes to the left
and it says "Lack of evidence to be considered a
nutrient or food component of public health
concern." Are there any nutrients where there is
no biomarker?

MEMBER BAILEY: Fiber. And it is a
nutrient of public health concern, because it’s
linked to a clinical outcome.

MEMBER DEWEY: Okay. So there’s
only -- yeah. So basically none of them have been
excluded on that basis. Okay. Thank you.

MEMBER BAILEY: That was a long way
around. Sorry. I didn’t get that question.

CHAIR SCHNEEMAN: No, but --
MEMBER BAILEY: But I spent so much
time on this, so I just really have to --

CHAIR SCHNEEMAN: I know. It’s burned
into your mind, so you’ve got to bring us all along.

MEMBER BAILEY: And it’s the color.

CHAIR SCHNEEMAN: So I can -- did you
have anything --

MEMBER DEWEY: No.

CHAIR SCHNEEMAN: Okay. So Rick?

MEMBER MATTES: Rick Mattes. I’m not
trying to add anything to your plate, but when you
look at beverages, will you be looking at them when
consumed alone versus with meals? It’s a question
that comes up quite often, and it’s kind of
relevant.

I mean, beverages serve functions.
When they’re consumed with a meal, they help you
swallow, and it doesn’t mean that one has to use
a sweetened beverage to accomplish that, but
weighing costs and benefits and hydration or not,
it is a more complicated question of knowing to what
degree beverages alone are contributing energy and
nutrients.

MEMBER BAILEY: Yeah. We don’t have that built into the analytic framework right now, but I think that can be something that we incorporate into the report as a research recommendation --

MEMBER MATTES: Yeah.

MEMBER BAILEY: -- something that could be looked at.

CHAIR SCHNEEMAN: Yeah. Is it available?

MEMBER BAILEY: Every eating occasion is recorded as a separate eating occasion. So you could do it that way. So if someone just reports a beverage, you could separate that out with the NHANES data.

CHAIR SCHNEEMAN: Dr. Heymsfield?

MEMBER HEYMSFIELD: How was strength measured?

MEMBER BAILEY: With hand grip.

MEMBER HEYMSFIELD: Hand grip?

MEMBER BAILEY: Yeah.
MEMBER HEYMSFIELD: Because what caught my eye was the Asians have highest prevalence of low strength, and you use that measure for a sarcopenia diagnosis, and strength is related to body size. I wondered if adjustments were made for body size?

MEMBER BAILEY: No. These -- what we presented today are just prevalence estimates. At some point, they were age-adjusted when I specified that for things that -- like cancer, but we haven’t done it like that for the muscle data, but that’s a good idea.

So that does bring up the point about protein, and it’s low in older adults. There’s a rather high prevalence of low muscle strength. How do you feel about that in terms of would that rise to the level of something you would consider to be a public health concern?

I’m not putting you on the spot --

MEMBER HEYMSFIELD: Yeah.

MEMBER BAILEY: -- specifically, but I mean, that --
MEMBER HEYMSFIELD: I’m not sure I know the answer.

MEMBER BAILEY: You can just say, tangerine. You know, that’s an option for you.

MEMBER HEYMSFIELD: Yeah. Just work with NHANES data a lot and am very interested in sarcopenia, and I think the body size is a very important covariant in that analysis, so I think before you make any conclusions about Asians being -- lacking strength, you need to really adjust for body size in some way.

I’m not sure how, but --

MEMBER TAVERAS: In the same vein of not wanting to add anything to this long list, but there are two things that I was going to ask about. One was, you talked about obesity in the adults but not in children, and I think there are really good definitions and NHANES, I think, now is reporting on severe obesity in children.

So I would ask if that is available and going to be included?

MEMBER BAILEY: So Jamy did some
follow-up on body composition data. Do you recall if those numbers --

    MEMBER ARD: I am sure they’re probably there, severe obesity for children. So I think that should be included. Yes.

    MEMBER TAVERAS: And along those same lines with an increase in severe obesity, we’re starting to see nonalcoholic liver disease in children, and it’s not one of the outcomes, so --

    MEMBER BAILEY: That wasn’t measured.

    MEMBER TAVERAS: It wasn’t?

    MEMBER BAILEY: Children -- but we talked a lot of about that. We really know that that’s an issue, and we wish we had more data to address that.

    MEMBER NAIMI: Yeah. If I -- that’s -- some of the elevated ALTs and ASTs in adults are accounted for by fatty liver, by alcohol as well as by hepatitis C, but there’s no way to tease those apart. And the AST/ALT for kids B-

    (Off-mic comments.)
MEMBER MAYER-DAVIS: Yeah. So again, speaking of adding things, so you mentioned that HEI was available for the cycle of NHANES data that you are using, but not other indices related to dietary patterns, and not wanting to, you know, steal any thunder from my colleague here to my left, you know, a variety of dietary patterns, you know, are -- you know demonstrate some really interesting findings.

So I don’t know how impossible is it to look at other types of indices related to dietary patterns beyond the HEI?

MEMBER BAILEY: Yeah. I agree with you. I think that there are a lot of different dietary patterns, but when we really looked at this data, it boiled down to five food groups that were contributing almost half or more energy.

So I think what we’re looking at is an American pattern. I think there’s variations in there. But from the 2015 to 2020 extensive work on dietary patterns, they recommended only indexes and scores be applied to characterize dietary
patterns because, after a cluster analysis, were
subject to too many decisions, and couldn’t be
reproduced across cohorts.

MEMBER MAYER-DAVIS: Right.

MEMBER BAILEY: And so --

MEMBER MAYER-DAVIS: So I was thinking
more specifically about Mediterranean diet, for
example, or DASH as another example.

MEMBER BAILEY: Well --

MEMBER MAYER-DAVIS: And I completely
agree with you --

MEMBER BAILEY: So the National Cancer
Institute has a dietary patterns methods
project --

MEMBER MAYER-DAVIS: Yes.

MEMBER BAILEY: -- and they use all the
different scores, and there’s a very high
congruency between the HEI and the Mediterranean
score and the DASH index. I’m not saying there’s
perfect agreement, but they’re pretty robust.

MEMBER BOUSHEY: Want me to back you up
on that?
MEMBER BAILEY: Sure do.

MEMBER BOUSHEY: Yeah. That’s what I was going to say. I mean -- and the other issue is creating those dietary patterns, you know, for individual food items is actually a little -- it is more challenging, not that it wouldn’t be a wonderful thing to do.

It’s just that it would be a large investment of time on your part, and we do know with the adult data from the dietary patterns methods project, but they are -- they all come out very similarly.

MEMBER BAILEY: But I really do hear your point, Beth --

MEMBER BOUSHEY: Yeah.

MEMBER BAILEY: -- and you know, we know what they’re not doing. They’re not doing this, but we don’t know what they are doing, and I think it could be a research recommendation that future committees walk in the door with knowing what the existing patterns are that are different than just HEI. That would be very helpful.
MEMBER MAYER-DAVIS: Yeah. Because there are established approaches to these scores, some of which are more common in the literature, but some are, you know, fairly obscure.

MEMBER BAILEY: Uh-huh.

MEMBER MAYER-DAVIS: So -- yeah.

MEMBER BOUSHEY: I wanted to give you a shout-out, a team shout-out. Your screener that you developed addressed one of the very comments that have come from the National Academy of Sciences report, so you have a lot to be proud of. That really answered a big question, that you have a method now of looking at these nutrients of concern. So I really have to give you a shout-out.

MEMBER BAILEY: We really took that report to heart when we were developing this, but what we realized is that system works nutrient by nutrient, but it failed us when we came across the adolescent females, because we were like this is how we’re going to say something is a food component of concern.

And then we were like wait a minute. We
have this high-risk -- what we consider to be a high-risk group, but -- so nothing is perfect, but we thank you very much.

CHAIR SCHNEEMAN: Actually, related to that topic -- and first of all, let me remind the Committee that the subcommittee has also asked for your input on presenting the conclusions around the chronic health conditions.

And if you’d recall -- and you can look back at your slides -- but there’s a general statement, and then Regan went through a lot of very specific data. And so part of the question that is being asked is, how do we represent -- well, how do we find a balance in representing that? Do we do just a general statement and then each one separately? Or do we need an overall conclusion statement?

And then the other that I think we have gotten some discussion is looking at these nutrients of public health concern.

And I’m going to ask you about potassium, because I know that potassium was below
the AI across the food groups, but we also have the new DRI report which did not give us a chronic disease reference value for potassium.

And so maybe just -- some of you in the subcommittee are thinking about potassium as a nutrient of public health concern.

MEMBER BAILEY: Yeah. As you know, the DRI was recently updated, and we talked about this. Jamy, if you want to -- can you summarize what we said in our small group meeting yesterday about potassium from -- Jamy was on the committee.

MEMBER ARD: Sure. So the issues with potassium, from a clinical standpoint -- yes, there are no issues with people coming into primary care and emergency rooms with rampant hypokalemia. So that’s not an issue.

I think the main potential chronic disease risk related to lower potassium intake is related to cardiovascular disease, and in particular, hypertension, and some of the sequelae of that.

So you know that there’s a relationship
between higher potassium intake and lower prevalence of high blood pressure. We know that there are differences in subpopulations in terms of potassium intake and some of those differences maybe explain some disparities in outcomes and health outcomes.

So for minority populations, African-Americans in particular, you see higher prevalence of hypertension, lower intake of potassium in that group. And we also know that potassium has a blunting effect in terms of the hypertensive effect of sodium.

So in populations where potassium intake is higher, even for a higher sodium intake, you see a less robust response in terms of blood pressure.

So I think part of the discussion we had yesterday was there could be an argument made that potassium intake is part of the public health concern group, even though we don’t have some of the, you know, sort of classical direct links; you have more indirect ones, per se.
But there is a body of evidence that supports the idea that higher potassium intakes may actually have an impact at the population level. So I think that, you know, summarizes the points in the discussion.

MEMBER BAZZANO: Lydia Bazzano. So I would second what Jamy just said, Dr. Ard, and also in terms of nutrients of concern, I know Steve can speak -- I know you all did not specifically say protein in older adults, but I think, you know, given the levels that -- the prevalence that we’re seeing, I think they probably should be concerned.

MEMBER DEWEY: Kay Dewey. I have another question. I think the list of nutrients where you were examining whether they were of public health concern did not include fats, and is there a reason for that?

MEMBER BAILEY: Yes. We hadn’t looked at that data specifically, but you’ve reminded me that we really need to do that. So we looked at saturated fats, but we didn’t look at other fatty acids so we should absolutely do that. Thank you.
CHAIR SCHNEEMAN: Any comments, any questions?

Regan, do you want to put that draft conclusion statement up, and let’s see if there’s -- ah. Great.

So I guess the question, though, is are we comfortable with this general statement, knowing that the report itself will go through some of the details that Regan has presented?

And again, it’s still a draft, so there’s still some tweaking that probably needs to be done.

MEMBER ARD: So the alternative to that statement is something that then calls out some specific chronic diseases that, you know, may be of more interest or more concern.

So as an example, we had nominated something like dental caries and metabolic syndrome and diabetes as being really concerning, as well as increasing rates of mortality related to chronic liver disease.

So these were things that were, you
know, somewhat striking for us as we reviewed the
data, but they were, you know, our perspective.
And so I guess the question is, do we just leave
this very general and be very generic and point out,
you know, things that we all know?

Chronic disease is increasing and the
disparities, and you know, it’s worse in some
subgroups compared to others, and we could stop
there.

Or we could, you know, incorporate or
call out things that we think are, you know,
particularly concerning, either across life stages
or related to other things that, you know, would
be relevant for some of the questions that other
subcommittees are dealing with, or related to
things that we think are relevant, you know, with
regard to where particular recommendations might
go or be needed for emphasis?

MEMBER DEWEY: Kay Dewey. So one
thought is to at least highlight in some way the
outcomes that are being examined in some of the
literature reviews that the different
subcommittees are doing.

So the certain cardiovascular disease outcomes are part of several of those, and also growth, size, and body composition, so overweight and obesity. All those come to my mind as deserving to be highlighted, because we are going to talk about whether diet is related to them.

MEMBER NOVOTNY: Just in general, I would like us to think about what to do with weight status or overweight/obesity. I see it’s kind of listed as a -- almost like a demographic, and whether it goes along with health condition or whether we have to like call it out as an intermediary, metabolic syndrome was mentioned in your review. It’s closer to the diet than the pathway of many of the conditions. So just to -- I think we should think about where that goes and follow it.

MEMBER TAVERAS: I wonder also if you can group them in that way, that some of these are obesity-related and make the summary a little more -- that the cluster is associated with
obesity?

MEMBER DONOVAN: Yeah. I'm sort of -- I'm just struggling on whether this is appropriate or how to say it, but I guess when I was looking at your comments and you were talking about racial/ethnic differences, that to me, it seems like we also need to include socioeconomic status and potential health care coverage, because I don't think it's just genetics.

Right? And that was kind of led to my respect of the genetic components, but we also disparities in prevention, and I think these differences are because people who have health insurance are getting their medications and they're getting earlier screening of prediabetes and they're getting a lot more prevention, and diet intervenes with that, but it's, you know, a broader issue, and it's quite beyond the scope of dietary guidelines.

But this aspect of, you know -- to give you more work, if we could look at things beyond -- you know, other demographics in terms of
SES or health care or SNAP utilization or -- because to me, that leads directly to potential application of dietary guidelines in nutrition programs.

MEMBER BAILEY: Many of our protocols have food security --

MEMBER DONOVAN: Yeah.

MEMBER BAILEY: -- included --

MEMBER DONOVAN: Right.

MEMBER BAILEY: -- to try to get --

MEMBER DONOVAN: Right.

MEMBER BAILEY: -- a different -- other than just, you know, how much money does your family have? How is that money distributed towards nutrition specifically? So that's ---

MEMBER DONOVAN: That would be great.

MEMBER BAILEY: -- yeah.

MEMBER MAYER-DAVIS: Just looking at this statement, I very much appreciate Rachel's comment -- this is Beth Mayer-Davis -- to pull out obesity, and then frame obesity-related
conditions.

I think that’s part of calling out and being more specific. Thinking back to Jamy’s comment, particularly about, you know, some of the areas that, you know, maybe are not above the radar right at this moment, you know, like increasing mortality related to liver disease, like dental caries.

So seeing those data, we’re not necessarily surprised, but it’s not necessarily what would have been front-of-mind, and so you know, I think that was really a good comment. So I think taking opportunity to be a little bit more specific here, in that regard.

And then my second part of this comment has to do with being more explicit about health equity and inequity, because that’s really, you know, what we’re talking about.

And I think that’s really important as we think about the Dietary Guidelines with respect to informing federal food policy, which is about food security, access, all those kinds of things.
So I’d like to, you know, see that aspect of health equity brought out as well.

CHAIR SCHNEEMAN: So we’re reaching the end of our allotted time, but one of the things that Dr. Kleinman and I have been talking about is, as one of the chapters, it’s important for us to start integrating the work of all the subcommittees and where do we come to after addressing all of these questions.

And so I’d like to just finish the meeting by maybe going around if we had any particular thoughts about that issue, or kind of what Regan was asking the Committee about. It’s an opportunity for you to get some final comments for today, based on what we’ve been hearing.

And are you beginning to see some things that sort of emerge to top priority from the work that your subcommittees are doing? So it’s always tough to figure out who goes first. I’m going to pick on Kay.

MEMBER DEWEY: Well, I mean, the challenge that we have is integrating across the
B to 24 age group, because this is the first time that recommendations for this age group are going to be this report, and as you all know, we have multiple outcomes for the same exposure, and so we haven’t yet talked about how to integrate across those.

Yeah. Well, one thing I do want to repeat that I said at the last meeting, to make sure everyone is aware, that we are only looking at a subset of all the different types of eating advice that might be given for this age group, and in particular, we’re focusing on the what to feed and not the how to feed.

So a general question then is, how far do we go in even talking about the ones where we have not done systematic reviews. And so any advice I throw back to you that the overall Committee would like to give us on how that gets approached would be very helpful.

MEMBER DONOVAN: I mean, I’ve just been seeing -- we haven’t really had the opportunity to speak in terms of Regan’s committee on intakes and
the prevalence of things like gestational diabetes
and all of those.

So I think that will be important to
start to integrate, and you know, and while
pregnancy and lactation has been a component of
previous Dietary Guidelines, I think pulling out,
you know, this sort of special life stage, as we’re
taking that life stage approach and thinking not
only about improvements for maternal health,
because we certainly know that women with
gestational diabetes are at higher risk for type
2 diabetes later.

So again, we haven’t really had a lot
of time to think about that, I think, in terms of
integrating the data in terms of whether pregnant
or lactating women are actually consuming and
what are the incidence of these health conditions?

But I think what we have seen, you know,
from both in B-24 and Pregnancy and Lactation, we
have a lot of inability to draw conclusions because
the data sets -- the data is just not there.

So clearly, as we move forward -- and
there was a comment earlier about research needs. 
And we'll have very long lists of research needs, 
and so that's -- again, I think at the end, we'll 
be able to make some conclusions, but 
unfortunately, I don't think very many of them will 
be strong, but we'll have lots of recommendations.

MEMBER BAILEY: I really like the life 
stage approach that we're taking, and it's very 
clear for B to 24 and Pregnancy and Lactation. 
They have specific working groups. It's been less 
clear to me how to handle the other life stages. 

And so integrating all the information 
from the different life stages is going to be a 
little bit more challenging, but I think really, 
really important. 

And then the other thing that really 
stuck out to me in going through all of the data 
that we did is that foods and nutrients are 
inextricably linked, when you see that the food 
changes over time, and we see that mirrored with 
nutrients. And I feel it's very important that we 
meet people where they are in terms of
recommendations. So people are consuming a lot of --

MEMBER DONOVAN: Burgers and sandwiches.

(General laughter.)

MEMBER BAILEY: So we have to give them tools and strategies to do that in a better way, not just, you know, you need to eat more of this, eat less of that. But giving them real strategies for success, I think, will be something that is important, at least from my perspective.

MEMBER NOVOTNY: What I'm thinking about is really integration and just trying how to weave this in a useful way, like a sentence, but I think the problem is this last point about socioeconomic status and the -- I know socioeconomic status has been in our models as a variable to consider, but given the potential use of our findings, I'm wondering whether we should be looking at the different subgroups in order to inform policy, and indeed, whether there should have been other kinds of variables in our models
that might have helped us, like food security or
something about health utilization.

I’m not sure also about the
race/ethnicity route, whether that’s going to be
the most helpful way to go about it, but that’s what
I’m thinking about.

MEMBER STANG: I would follow up
with -- I think we’ve talked about the life span,
but also thinking about the life course approach.
So the fact -- when we were looking at the data
yesterday, we saw these adolescent females with
this constellation of poor nutrition, and I’m
thinking, these are our future mothers.

And so there’s this whole circular
piece that what’s important during pregnancy and
lactation informs what happens to the children,
which then grow up to be mothers themselves or
fathers.

And so somehow to weave that piece
throughout the report that there’s this
generational piece that I think often we miss
because of the way that data is collected or
reported, but it has a lot of contextual implications for the recommendations we make.

MEMBER TAVERAS: I was thinking, I guess, three things.

One, Sharon mentioned, it’s discouraging to see so much insufficient evidence and inability to make conclusions from very little, in some cases, data that is out there and results.

And I think that’s going to be really important as we summarize, because I think there is quite a bit of attention on what is going to emerge from here, and I think we’ll have to be careful with how do we frame this in a way that sets up the next Dietary Guidelines or the next Committee on what were some of these research recommendations, and where we might be able to contribute for research purposes for the next round?

I’m struck, Reagan, with the conversation we just had about so many of these chronic diseases are obesity-related and the increase in trends and the prevalence of obesity,
and I think that’s important.

I think we have to call it out. Even if all the chronic diseases, cancer, CVD, if they all cluster around obesity, I think that it is going to be important that we really drive that home, because in our subcommittee and in many others, growth size and body composition is something we’re paying close attention to.

And then the only other thing that I found interesting is there is a big drop-off -- and maybe because I am a pediatrician and I think a lot about the child diet, but there are some really interesting patterns from two to five to six and older that seems to be this critical point where so many of the other patterns that you showed are deteriorating and decreasing.

And I wonder if there’s a way -- as we talk also about life course and life stage, if we can point out some areas of opportunity in either these critical periods or setting staff to work with people and populations in those critical periods, that there might be more room for
influencing diet in those settings and age groups.

MEMBER DONOVAN: I think -- I looked at that too as kind of the child care to school, and there's different policies in school lunch and in CACP, and so I think that's a really smart observation.

MEMBER MATTES: Yeah. I don't want to just be redundant, but to amplify the importance of paying attention in our discussion sections about future directions, that this is so disheartening.

We spent so much time building these models, to find the greatest science, and we're all ending up with science that isn't answering the questions, and so it's vital that we encourage future researchers to design their trials so that we can get to the bottom of all this.

The only other thing that I felt compelled to comment on, but nobody anywhere has talked about food palatability. I mean, there are certainly issues and disparity issues and so on with regard to access and so on, but the primary
reason that we pick one thing over another is
because we like one thing more than another, and
so I hope we don’t lose sight of that, and somehow
we can leave this in, that we have to pay attention
to that component of food.

MEMBER BAZZANO: So I like the life
course approach and the life stages approach that
we’re taking, and I do think, you know, that it’s
important to distinguish the different periods
because nutrition is different and the needs are
different in different periods of time, and
specifically I think in the older adults, you know,
we need to think about that as well, that so far,
we’re kind of all lumped in as adults. So you know
I think that might be a particular group with
particular outcomes to be focused on.

And then the other thing I wanted to
mention was also kind of along the lines of what
Richard, Dr. Mattes just mentioned, that the data
that we have, all of these studies, we’ve been
reviewing a lot, a tremendous amount of studies,
most of which are not actually designed to study
what we’re trying to use them to -- the question
that we’re trying to use them to answer.

So that’s a different issue, but it does
get to the importance of research for the specific
questions that we want to answer.

MEMBER SABATÉ: I agree with many of
the things that has been said, and I think on the
last presentation, I think it struck me that the
way that the menu must be -- almost everything is
concentrated in sandwiches and burgers.

So it’s culprit of many things, but also
an opportunity. And I think that changes in social
trends from different perspectives, not only from
food, but also from the perspectives of
sustainability and taste. I think this is an
opportunity to try to improve the health of
Americans, but also tackle other social issues that
are concern to today’s society.

MEMBER LEIDY: I’m not sure if I can add
too much to the conversation when we get around to
this point, but there’s just a couple of things that
have come to mind. You know, I think we’ve all said
It’s surprising right now when you see that there’s just a lot of limited evidence, and the evidence that exists seems to be from cohort studies, when we only say we use randomized trials, and I feel like now that kind of just goes to the wayside, because we know we need them.

It’s that next step of how do you make that happen, you know, from a funding standpoint of getting that out. But I still think that’s a vital part of trying to answer some of the questions that we have.

So it was more of a surprise. Maybe we’ll all sandbagging writing waiting till the end, until March, when all the data come out. I don’t think that’s the case, but it would be nice if it was. An unrelated issue -- when I look at the food patterns and I think I talked to Regan at lunch a little bit about the different food groups, and I’m struck by the fact that even when we look at whole grains and whether there’s an increase in whole grain consumption, we also see that a lot of whole
grains that are -- whether they're recommended or they're either in schools or what-not, also have an added sugar component to them.

And you know, we'll look at interventions, at least from -- you know, if they're doing added sugars or whatnot, but I think that's just a point that I don't -- we kind of missed that.

We look at the food groups and we see where they're coming from, but I'm not sure if we can then tie that fact into a health outcome to say, you know, whole grains may be beneficial, but as an example.

But if they're, you know, including added sugars, then a lot of those maybe potential health benefits go away, and I don't know -- we probably may not be the group to do that, but it's just something that, even looking at the food groups, if we could really separate them out based on some of the other food components that are part of that, and whether that's dietary patterning, I'm not sure that where that fits. It's just something
that I always see when we look at the different food
groups, that other components, other nutrients
with some of the other healthier food items, and
it’s just hard to tease that out.

And then just a last point. You know,
when we look at the different life stages, and
adolescents, particularly females, are, you know,
the group of interest from a nutrient standpoint,
we also -- I know at least from the literature that
there are very few studies in that population.

So they kind of go hand in hand. You see the nutrition issues, but they’re not always
linked with some of the other health outcomes
usually for compliance or attrition with that
population. But I think that’s a really big area
for future recommendations.

MEMBER ARD: So I think we have not
gotten to the dietary patterns section yet, but if
I would say something that integrates what we’re
talking about, I think tomorrow we should hopefully
be able to have more discussion about the sort of
idea of dietary patterns, when they -- you know,
sort of concept that we can double-down on, and that
was brought forth in the previous Guidelines.

And I think it speaks to several things, you know, what Heather just talked about and what
Regan talked about, where nutrients are not consumed in isolation, and foods are not consumed in isolation, when I have my burger and sandwich, I’m also going to have my starchy potatoes, and my, you know, sugar-sweetened beverage.

Right? You know these things travel together in the American pattern, and we need to acknowledge the idea that across life stages and in the life cycle, these patterns tend to change, and even from, you know, the use of complementary foods, and how, you know, we feed infants -- those things are starting to develop early. And so if we could think about that idea of how we help inform people around those, you know, concepts of foods traveling together.

And then I also think, you know, with regards to the idea of calling out obesity, that’s very important, but we haven’t really talked about
energy intake.

And so at some point, we’ve got to deal with that piece of, you know, how we integrate that into all of what we’re talking about, because I think at the end of the day, quality matters, foods matter, nutrients matter. But energy is very important.

MEMBER DAVIS: I think it’s very important that our report is looking at life stage, and this will be the first time that we’ve ever looked at it from birth all the way up to the elderly population, and I think our doing it that way, presenting our report by life stage, is more useful for the end user, for the public.

And indeed I think we can look at certain things that we’ve seen in dietary intake of the trends over the last few years. For example, there’s been a slight increase in whole grains. Although whole grain intake is fairly low, there’s been a slight increase, and is this because the Dietary Guidelines have reported this, and then I think industry may be reacting to this,
so they’re putting more whole grains into, for example, breakfast cereals and bars, and so forth.

So I think, you know, our report is quite important in informing the public, but also industry that feeds the public.

MEMBER HEYMSFIELD: Tomorrow, I’ll give the report from the Frequency of Eating subcommittee, and I think that’s a very important front-end part of our report, is what you going to generate from NHANES data. So I’m really looking forward to what that will be, because from what we’ve found so far, there’s huge gaps in literature, and we spent a lot of time trying to define what we mean by frequency of eating and digestion and so on.

So it would be very good for you to work with us so we make sure we have the same definition of frequency of eating.

MEMBER SNETSELAAR: I also think that what we’re doing in this Committee in terms of looking at younger age groups is incredibly important, and much of what has come up through this
Committee is the idea that we want to be consistent.

We want to be sure that we’re working together as subcommittees, and particularly, as we focus on these younger age groups, being very careful to ensure that we have conclusion statements and then grading that it is very consistent across committees I think is very important.

MEMBER MAYER-DAVIS: This is Beth Mayer-Davis. Just wanted to note that to some extent by design some of the questions that we’ve addressed have had quite small numbers of studies that sort of made it all through.

But to some extent, that was by design so that we would get our systems in place, and made sure that we were proceeding, you know, in appropriate fashion.

So I can at least tell you that, for Beverages and Added Sugars, some of the questions to come have much larger bodies of evidence, so they will not all be three studies here or four studies there, for better or for worse. Right?
So you know, again, not wanting again
to steal the thunder from Carol’s report on dietary
patterns, you know, that subcommittee talked about
yesterday, and it is relevant, I think, at this
point.

Jamy alluded to this a little bit as well. I think by way of integration across
subcommittees, having a framework of thinking
about dietary patterns and what we’ve been thinking
about by way of hierarchy of dietary patterns,
foods and nutrients, I think that will help with
some cohesion, including how we integrate across
subcommittees, you know, so for example, thinking
about the Beverages and Added Sugars committee.
Thinking about seafood and fats, and how, you know,
those elements come into play with the dietary
patterns.

So I think that will be an important
aspect that will help us in terms of integration.

MEMBER BOUSHEY: I could say wow
everyone said it. But no I have a list, and actually
it’s a list that supports things that have really
been said, and I -- Steven, Beth -- you know, Beth is working on this beverage guidelines.

I thought going through and describing all those beverages, that’s like doing minor surgery. We really have an issue with vernacular, and a lot of it is driven by popular words, but I think we do need to concentrate a bit on how we can make sure that what we’re doing now will be repeatable, that we use language that does describe what people are eating and the activities.

And part of the challenge of this is think about your beverages. Twenty years ago, we only had like one soda that you could select. You know, so really, we’re facing a new world where we get really new foods, you know, almost every year.

And so that’s a burden on our Committee, and we have to somehow think of how to make all that make sense and to be able to bring it all together, because this idea of Kay’s -- you know, really we do need -- Kay said we need to put together all of our work across all of our groups.

And I thought that was a great
suggestion. And, Rick, about your palatability, I think it’s surprising that burgers and sandwiches are so high, and yet we have, you know, this low whole grains. It just doesn’t make sense, does it? So we really have a lot to do to make these guidelines exciting, that people want to follow them, that people see them as something -- hey, I’d like to do that, but -- and I’m not sure that we can do that, but let’s try to think that we can.

Thank you.

CHAIR SCHNEEMAN: So I’m going to give you the last comment.

VICE CHAIR KLEINMAN: So I think that we’ve worked very hard to describe the food patterns or consumption patterns at all of the different life stages that we’ve talked about. But one way to integrate this is to talk about how these patterns change over time, and we’ve also examined health consequences at these various life stages of the foods that are being consumed, and that’s another opportunity for
integration, if we look at cognition, cardiovascular disease, and hypertension and the various other outcomes, and look to see how these relationships change over time as well.

So I think -- I liked what everybody had to say. Jamy mentioned the generational aspect of this, and I think we need to weave that into that conversation about change over time.

And then I think it's really important that we couch all of this as food as one of the social determinants, but there are others, and to the degree that we can link food consumption patterns at least to some sense of economic status, that will help us a lot in completing the story and bringing it together so that we demonstrate where the real opportunity here exists.

And I'll stop there.

CHAIR SCHNEEMAN: Well, these comments are very helpful; I have been scribbling notes here.

So I think at this point we're adjourned for today. I'm looking at Eve. Do we need to
highlight anything?

So again, we will reconvene tomorrow morning at nine o’clock. We have several more subcommittee reports to go through, and then we will have the period for public comment, which I’m looking forward to.

So I hope you all have a good evening.

Thank you.

(Whereupon, at 4:28 p.m., the meeting was adjourned, to reconvene at 9:00 a.m., Friday, January 24, 2020.)
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Systematic Review:

- Subgroup 1: 73 subjects
- Subgroup 2: 73 subjects

Summary:

- The overall study involved 146 subjects from 2021 to 2025.
- The results were systematically reviewed across all studies.
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In the matter of: Public Meeting

Before: 2020 Dietary Guidelines Advisory Committee

Date: 01-23-20

Place: Houston, Texas

was duly recorded and accurately transcribed under my direction; further, that said transcript is a true and accurate record of the proceedings.

[Signature]

Court Reporter