

Dietary Guidelines Advisory
Committee Meeting
Day 2

Date: October 31, 2008
Time: 8:35 a.m.
Location: USDA South Building
Jefferson Auditorium
1400 Independence Avenue, SW
Washington, D.C.

Meeting Conducted By: Dr. Van Horn

1 P R O C E E D I N G S

2 DR. VAN HORN: Good morning. We are going to get
3 started again this morning. Thank you all for joining
4 us and Happy Halloween. We actually had one of our
5 members come in here looking a little more like
6 Halloween, but we'll let you guess who that is.

7 We are delighted to have a chance to launch into
8 our second round of discussion, but first, for the
9 purposes of those in the audience, we thought it would
10 be helpful to summarize some of the discussion that
11 took place yesterday. Those on the Committee have a
12 copy of these, but I'll just briefly give you a hint on
13 exactly what the key topics were that were addressed.
14 Some of the major points related to the discussion on
15 nutrient adequacy included the shortfall nutrients for
16 adults and children that continue to be a problem,
17 including calcium, potassium, fiber magnesium and
18 vitamin E; and also for adults, vitamins A and C; and
19 children, especially vitamins A, C and possibly
20 phosphorous were a concern.

21 Shortfalls in terms of food components include
22 fruits and vegetables, especially dark green and orange

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1 vegetables and legumes, whole grains and milk. There
2 is excessive intake of sodium and calories from solid
3 fats, alcohol and added sugars, otherwise known as
4 SOFAAS. I guess we are all calling them that.

5 Then there is the need to emphasize meeting
6 nutrient needs within energy needs by recommending
7 nutrient-dense foods, and perhaps considering new ways
8 to group foods, and that's something that we touched on
9 yesterday that we'll perhaps take up again. I would
10 also add to that actually the whole concept of
11 discretionary calories, which is the flip side of that;
12 the meeting the nutrient density needs, but also
13 considering what discretionary calories might include.

14 Since diet is a complex exposure, there is a need
15 to emphasize dietary patterns. We talked about whole
16 foods and the cultural and social aspects of eating and
17 the needs for including dietary patterns, even along
18 with healthy aging and longevity.

19 Then we raised some potential new questions;
20 looking at the amount and source of protein; the role
21 of bioactive proteins in methyl groups; in the
22 discussion of folate, we talked about potential dual

1 effects on cancer and risk, and needs to look at the
2 positives and negatives of that; vitamin D, of course,
3 which is currently a hot topic and the need to consider
4 it, and to look in combination and coordination with
5 the IOM Committee. Other nutrients that were not
6 investigated in 2005 include things like selenium and
7 possibly other micronutrients. And then, the whole
8 concept of bio availability that we raised. And then
9 also, the need to look at changes in nutrient content
10 over time in processing and the whole concept of
11 globalization of food and nutrient integrity; so, the
12 idea of looking at possible speakers to give us further
13 input on that.

14 Then, in our fluid and electrolyte discussion, we
15 talked about the recent research that continues to
16 recognize the benefits of potassium and reduced sodium
17 in the diet. Consensus still exists on the benefits of
18 lower blood pressure and the fact that blood pressure
19 rises throughout one's lifetime, which is usual, but
20 not normal in that ideal. The adverse effects of
21 excess salt intake, of course, include hypertension and
22 cardiovascular disease and stroke risks; probable

1 relationships with gastric cancer; and there is a
2 relationship -- a suggested relationship of increased
3 risk of osteoporosis and increased left ventricular
4 mass with higher sodium intakes; and then the
5 hypothesized relationship with overweight and obesity
6 as well, recognizing that more food obviously
7 contributes more sodium.

8 So, looking at new evidence and emphases, blood
9 pressure status of Americans is getting worse. We
10 assume its related to the obesity epidemic; evidence
11 and benefit of reducing salt and increasing potassium,
12 as far as cardiovascular disease events; and concerns
13 about blood pressure in children, which continue to
14 accompany the rise in pediatric obesity as well. Some
15 potential new research questions; what dietary factors
16 influence blood pressure in children and young adults;
17 not necessarily assuming that everything that's true in
18 adults is true in children; we need to take a closer
19 look at their diet. Should the target for sodium
20 intake be reduced from 2,300 to 1,500 milligrams per
21 day, or at least in those at high risk or already
22 hypertensive or pre-hypertensive? Looking at the

1 current sources of sodium; the effects of certain
2 beverages, such as coffee and tea on cardiovascular
3 disease and its risk factors; what are the effects of
4 sugar-sweetened beverages and artificial-sweeteners,
5 and water on weight in children and adults. So, the
6 discussion then revolved around concern about blood
7 pressure in children, differentiating inherited blood
8 pressure hypertension versus acquired through
9 environmental exposures, and looking at again, high
10 sodium/low potassium intake. More discussion on
11 coffee, tea and considering other beverage-related
12 questions, as time goes on, related to blood pressure.
13 Also, the effects of reduced sodium on protein balance,
14 iodine deficiency and food safety questions related to
15 making sure we don't compromise any of that.

16 For potassium, does it make a difference, if
17 potassium comes naturally or is added as a supplement?
18 We spent a little time on that.

19 And then additional information may be needed on
20 the composition of processed foods and new fortified
21 products; has food composition data been updated with
22 these new foods, such as chicken injected with brine

1 and some of the other processed foods that we kind of
2 take for granted?

3 So, having summarized all that, I guess we had a
4 pretty productive day yesterday, and I'm sure today
5 will be equally productive. I am happy now to welcome
6 Dr. Pi-Sunyer, as we start off this day's discussion
7 related to energy balance and weight maintenance,
8 management. Oh, sorry. Sorry. Oh? Sure. And Rob
9 Post would like to add a point.

10 DR. POST: Yeah. I'm not sure if this is on and
11 if everybody can hear me. Okay. In the discussion
12 yesterday, I just wanted to add a point, a suggestion,
13 for consideration in the discussion of dietary sources
14 of sodium and processed foods, and there are a couple
15 of points in the summary that Dr. Van Horn just
16 mentioned.

17 To consider that there is an issue in terms of
18 potential compromising of food safety, because a lot of
19 the substances that are used to promote food safety
20 that are antimicrobial in nature, anti-listeria, for
21 example, in certain processed foods are sodium-based,
22 so consider that issue in the discussion of sodium,

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1 that suggestion.

2 DR. VAN HORN: Anything else? I don't want to cut
3 short any additional comments. Sure. Yes.

4 MS. McMURRY: Just one quick update, just to make
5 sure you are all aware of another complementary effort
6 at the Institute of Medicine. They have just recently
7 convened a panel to look at strategies for reducing
8 sodium intake to the 2005 Dietary Guidelines level; so
9 to the extent that it's possible, it would be nice to
10 try to coordinate with that effort too.

11 DR. VAN HORN: Right. There were a couple of
12 recommendations yesterday to look at linkages between
13 this group and IOM, so thank you. Yes, that's a great
14 idea too, Kathryn. Thank you. Okay. Dr. Pi-Sunyer.

15 DR. PI-SUNYER: Okay. Well, good morning to you.
16 Is this on? I am going to begin this discussion on
17 energy balance and then Dr. Nelson is going to talk.
18 She has been a member of the Physical Activity Task
19 Force for the HHS that just finished their report, and
20 so I'm not going to say much of anything about physical
21 activity; she will. And then Dr. Christine Williams,
22 who is a pediatrician, is going to talk about, a bit

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1 about children and adolescents, and finally, Dr. Rafael
2 Perez is going to say a few words about weight gain
3 during pregnancy. And then we'll leave it open for
4 discussion.

5 Just, by way of background, I want to remind you
6 of the trends and age adjusted prevalence of obesity.
7 This is for adults 20 to 74, and you can see from 1974
8 up to 2000 there was an enormous secular increase in
9 the number of people, who were obese, both males and
10 females, and this continued pretty much right up to
11 about 2006. Now, it may be flattening out a little
12 bit. It's not quite clear in the latest NHANES survey,
13 but an enormous change in the population in terms of
14 overweight and obesity.

15 You have all seen the CDC maps and they are
16 graphic, and how they move from a very low rate of
17 obesity to a rate where now up to three states are
18 above a prevalence rate of 30 percent -- 29 percent,
19 and growing rapidly around the country.

20 The same kind of trend you can see in children.
21 This is data from NHANES for children showing you the
22 same kind of increase over the same period of time, and

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1 the children and adolescents, their rate has continued
2 to go up right up to the present time.

3 We are worried about this because of the medical
4 complications, particularly the ones you see on the
5 right; the coronary heart disease, diabetes,
6 dyslipidemia and hypertension. The paper today
7 announces that our rate of diabetes incidences has
8 doubled, so this is a very serious condition. There is
9 more data over the last five years; new data on the
10 relationship of obesity to cancer, and I'll talk about
11 that a little bit; and then there is more data on non-
12 alcoholic fatty liver disease, which turns out to be
13 probably the greatest cause of cirrhosis in this
14 country after alcohol; and then quite a lot of data
15 related to pulmonary disease and sleep apnea.

16 So this really impacts Americans' quality of life
17 and it really impacts the cost of medical care in a
18 very ominous way. We have not only the epidemic of
19 obesity, but this is related to an epidemic of what we
20 call the metabolic syndrome, which is really something
21 that leads to much greater risks for both Type 2
22 diabetes and cardiovascular disease. You can see how

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1 the overweight and obese prevalence on the top -- men
2 in red; women in yellow -- is tracked by the metabolic
3 syndrome down below in white and blue. So, as people
4 gain weight, they also increase the number of them that
5 have the metabolic syndrome, which is clearly a risk
6 factor for both Type 2 diabetes and heart attack.

7 There is increasing data about the relationship of
8 obesity to cancer, and you can see here a study from
9 the European Commission -- showing you on the left,
10 men; and the right, women -- with the kinds of cancers
11 that have an increased incidence that is thought to be
12 related to increased overweight. In men, we are
13 talking about colon cancer, prostate cancer, kidney
14 cancer and gallbladder cancer; in the women, we are
15 talking about breast cancer, colon cancer, endometrial
16 cancer, kidney cancer and gallbladder cancer. So there
17 is, over the last five years there has been quite a lot
18 of new epidemiological; mostly observational -- really
19 observational data linking overweight and obesity to an
20 increased cancer incidence.

21 So what is the established science in this area?

22 Well, it's really pretty simple. I think very few

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1 people would argue that this isn't true; the caloric
2 intake has gone up and physical activity has gone down.
3 The balance has shifted towards greater energy
4 reserves, and as a result, increased weight. There is
5 some argument around the world of how much is related
6 to intake and how much to physical activity, but I
7 think when we are dealing with the issue, we really
8 need to deal with both of them.

9 We know that you can change the way people eat and
10 the way they behave in terms of physical activity.
11 This shows you the data from the NIH Center in Phoenix,
12 which studies the Pima Indians showing you the Pima
13 Indians' body mass index in Arizona and in Mexico; the
14 differences; the Arizonians have very high fat, high
15 calorie, high alcohol, a very sedentary kind of
16 lifestyle; whereas the Mexican Pimas were genetically
17 similar, are much more physically active and have a
18 much more traditional high fiber, high carbohydrate,
19 low-fat kind of diet.

20 In terms of the established science, we know that
21 obesity does lead to a number of major diseases. It
22 can also lead to a number of less prevalent, but

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1 serious diseases. We know the public awareness of the
2 link between obesity and chronic disease is low, and
3 that the awareness needs to be increased sometime,
4 particularly in people who already have risk factors
5 for chronic disease.

6 The American paradox, I think in 2008 is that we
7 have an escalating trend towards poor nourishment and
8 health in a land of plenty, and because of the
9 sedentary lifestyles and poor food choices, many
10 Americans exceed their caloric needs without meeting
11 their nutrient requirements, and this was dealt with in
12 the last Dietary Guidelines Committee, and I think it
13 really needs to be dealt with in ours.

14 What kind of consensus do we have about all of
15 this? Well, first, it's that we are making very little
16 impact; second, we don't seem to have the adequate
17 tools to change lifestyle behavior in the way we would
18 like it to be changed; we need a lot more public
19 awareness of the relation of obesity to chronic
20 disease; and I think the awareness needs to increase
21 particularly in people who already have the risk
22 factors, which is a large part of the population, who

1 think they are healthy.

2 One question I think our Committee needs to
3 address is should we focus on simply on prevention of
4 weight gain, and should we avoid focusing on weight
5 loss? The last Dietary Guidelines did talk a bit about
6 weight loss. I think the really important thing from a
7 public health point of view is to prevent people from
8 gaining weight. It is very difficult to lose weight
9 once you have gained it; you tend to gain it back.
10 It's sort of something that we have not been able to do
11 well. On the other hand, I think if we focus the
12 message on the prevention of weight gain, keeping
13 people within the normal BMI categories, we might do
14 better in the long run. This is taken from the old
15 DRI. It seems logical to base estimated energy intake
16 on the amounts of energy that need to be consumed to
17 maintain energy balance in adults, who maintain
18 desirable body weights; also taking into account the
19 increments in energy expenditure elicited by their
20 habitual level of activity. So, what we are saying is
21 the energy intake estimated should depend on the energy
22 required for somebody in a normal BMI category, and

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1 also taking into advice their physical activity.

2 Now we know that small daily imbalances in energy
3 intake have an effect in body fat mass. I am showing
4 you this data from my colleague from Columbia, Michael
5 Rosenbaum, which shows you what an excess intake of 12
6 calories per day, 25 calories per day, and 125 calories
7 per day can do to change in body fat over one year.

8 And you can see that it takes very little excess intake
9 to really have a major impact on weight gain. And we
10 know that Americans, as adults, continue to gain weight
11 every year from age 20 to age 60. So I think the big
12 message here is how we can get it across to people that
13 with a small change in lifestyle they can have a big
14 impact on preventing weight gain as they move from age
15 20 to age 60.

16 We have a sedentary lifestyle. This is from the
17 Surgeon General's report a while back, but it shows you
18 that both in men and women, in all age groups, the
19 percent not particularly participating in physical
20 activity is extremely high, and this also we need to
21 deal with.

22 What are the issues that need further discussion

1 or further evaluation? One, I think -- and these were
2 actually dealt with, with the last Dietary Guidelines
3 Committee, but I think we need to revisit them, because
4 of new data in all of these. The first is what are the
5 optimal proportions of dietary fat and carbohydrates to
6 prevent weight gain; the second is how is physical
7 activity related to body weight; the third is how much
8 physical activity is needed to avoid weight gain; and
9 fourth is caloric compensation different for solid and
10 liquid foods?

11 Other issues, do energy-dense/nutrient-poor foods
12 displace energy-poor/nutrient-rich foods, and does this
13 lead to weight gain and nutrient inadequacy; and what
14 kind of data do we have for saying this is true?

15 Other questions to address to prevent weight gain,
16 I have already mentioned the optimal proportions of
17 macronutrients; the effect of energy-dense foods; the
18 effect of portion size; the effect of added sugars and
19 the difference between added sugars in liquid and
20 solids; the effect of snacks; and what intake pattern
21 is most likely to prevent weight gain?

22 Finally, you know, what behaviors are most likely

1 to prevent weight gain? We have not been able to
2 figure out how to change lifestyle behaviors to prevent
3 this continuing increase in weight throughout the
4 lifespan in Americans.

5 There are also questions we need to look at to
6 prevent weight gain in special groups; children and
7 adolescents, which Dr. Williams is going to speak
8 about; pregnant and lactating women, which Dr. Perez is
9 going to speak about; the elderly; and particularly,
10 minority women, who have the greatest prevalence of
11 obesity in this country.

12 There are questions about discretionary calories;
13 do Americans really have any I think is the big
14 question? And the second is, is it too difficult a
15 concept? We wrestled with that a bit, you know, the
16 last time around and I think we need to talk about
17 whether this is a concept that, as a public health
18 message, is difficult to get across. And the best way
19 obviously, I think, to talk about it is in relation to
20 physical activity, but we need to address that.

21 Potential guest speakers that I think would be
22 helpful to us in our deliberations that I thought

1 about; one was Adam Drewnowski, who could come and
2 speak to us about nutrient density versus nutrient
3 adequacy; and Barbara Rolls, who has done so much work
4 on liquids versus solid compensation. So I think they
5 might be two guest speakers that we might like to hear
6 from.

7 So, in summary, the dietary factors that affect
8 energy intake that we need to deal with are nutrient
9 composition, energy density, portion size, liquid
10 versus solid, snacks, and then how physical activity
11 impacts the whole issue. So thank you very much for
12 your attention, and I'll pass this on to Dr. Nelson.

13 DR. NELSON: Thank you, Xave. Well, I had the
14 wonderful honor of serving on the Physical Activity
15 Guidelines Committee over the past year. Also, similar
16 to this Committee, we had 13 members, if I remember
17 correctly, and a wonderful group of scientists; all
18 with expertise in physical activity and exercise, and
19 health and public health. And, I won't go into the
20 whole history of why these Physical Activity Guidelines
21 came to be, but I wanted to talk mostly about sort of a
22 process and some of our main findings.

1 A couple of key things that I think are important
2 are that, in terms of history is, when we look at the
3 2005 Dietary Guidelines, you can see that there is
4 actually, you know, quite a bit of information around
5 physical activity. I think that what was important
6 was, because there is a whole -- I mean, really in the
7 last 20 years there is really a real growth in the
8 amount of research in the area and the number of
9 scientists who have really devoted their careers, it
10 seemed very important to actually have a committee that
11 really could look at all of the evidence around, not
12 just around energy balance, but around a number of
13 different health outcomes; and to also put a little bit
14 more information around physical activity beyond just
15 the sort of 30 minutes a day of physical activity.

16 So, background, Americans are largely inactive;
17 many opportunities for physical activity have been
18 engineered out of daily life. I think what's
19 interesting is that if you look at the data around just
20 leisure time physical activity, it's been held pretty
21 constant, but when you look at the number of sedentary
22 activities and the hours of actual sedentariness,

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1 that's what's really gone up. And that physical
2 activity is one of the most important steps people can
3 take for their overall health. So the process, we had
4 quite a -- this committee I have to say is quite
5 luxurious. We have a document from which to work from;
6 we have two years to do our work; we -- our committee
7 was convened with the first meeting, in June of 2007,
8 following the same FACA regulations. We met three
9 times. We met in June, December and February. We
10 reviewed the evidence from 1994, when the Surgeon
11 General's Report came out, to 2008; and if we had more
12 time, our report would not have been 668 pages. I
13 think it would have been more like 350 pages, but -- so
14 our technical report, while it's not printed yet --
15 it's still on-line. It's very accessible and I'll give
16 you the link to that in a bit. So we submitted our
17 technical report in June, I believe, or May, late May
18 of this year, and then HHS had a writing group that put
19 together the actual Physical Activity Guidelines. And
20 I think that's important for us to remember, you know,
21 we don't develop guidelines. We look at the evidence,
22 and from that the guidelines are developed; so the same

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1 with our Committee here.

2 So what's new about the Guidelines? It was the
3 first major science review in more than a decade to
4 address Americans over the age of six in specific
5 subgroups, and it really went beyond just the 30
6 minutes or more of most days of the week, although that
7 was sort of a starting point for us; thus providing
8 greater detail regarding dose and a lot more
9 information around physical activities for Americans.

10 So, our major research findings, we were separated
11 into subgroups that were looking at major chronic
12 conditions, and what we saw was regular activity
13 reduces the risk of most of the chronic diseases, as
14 most everybody knows, and that these are some of the
15 more salient findings that, in fact, some activity is
16 better than none; basically that being sedentary
17 confers the greatest health risk, and that any activity
18 is better than none, but that there is added health
19 benefits occur as the amount of activity increases;
20 both aerobic and muscle strengthening activities are
21 beneficial. Health benefits apply to people of all
22 types, sizes and ages. Health benefits occur for

1 people with disabilities. Physical activity can be
2 done safely. Benefits far outweigh possible risks.
3 Physical activity provides health benefits regardless
4 of body weight changes over time, so at any given body
5 weight, a person who is more active is going to be
6 healthier than a person who is not active.

7 In terms of the sort of dose of activity, a total
8 of two-and-a-half hours a week of moderate intensity
9 aerobic activities substantially reduces the risk of
10 many chronic diseases and other adverse health
11 outcomes. So, this is really back to the 30 minutes a
12 day. I mean, that's where the evidence seemed to
13 really converge now. I will tell you that some of the
14 issues -- maybe I am speaking personally here -- but,
15 because we have had this guideline for so long, most of
16 the good research studies really are designed to look
17 at this dose of physical activity. So, I mean, one of
18 our research sort of questions is, you know, is there a
19 range of doses that confer; but this is what sort of
20 settled out. Again, any activity is better than none,
21 but this is really where most of the health benefits
22 confer, and then as people move from two hours and 30

1 minutes a week towards five hours or up to an hour a
2 day of moderate activity, you get even more benefits;
3 really basically more is even better especially if you
4 have some health concerns.

5 And then the other thing that I think is important
6 is that -- oh, wait a second -- I just wanted to go
7 back -- what I don't have here is in fact that -- well,
8 I'll go with the Guidelines -- just a second. So, when
9 we look at children, and this is really -- there are no
10 great changes from what the Dietary Guidelines did in
11 2005. You are all looking at similar data. But, with
12 children, one hour or more physical activity a day
13 that's at least moderate is beneficial. Most of the
14 one or more hours a day should be either moderate or
15 vigorous, and to do vigorous intensity physical
16 activity at least three days a week, so that children
17 should have some vigorous activity. As part of one or
18 more hours daily activity includes some muscle
19 strengthening activities at least three days a week,
20 and as part of one or more hours of physical activity
21 include bone strengthening activities at least three
22 days a week. And the real key here is that we need to

1 encourage young people to participate in physical
2 activities that are age appropriate; that are enjoyable
3 and offer a variety of activities. We are not talking
4 about sending the eight-year-old to a gym and lifting
5 weights. We are talking about playing on the jungle
6 gym and climbing trees, and doing hopscotch and jump
7 rope, and everything else that young people at least
8 used to do.

9 In terms of adults, 18 to 64, similar two hours
10 and 30 minutes a week of moderate intensity or one hour
11 and 15 minutes of vigorous intensity activity, or a
12 combination of the two.

13 So this was the data. We weren't sure where this
14 was going to show up, I'll tell you, but it was a
15 charge right off the bat that we decided we wanted to
16 look at was this -- is it intense activities or is it
17 moderate activities, and then we weren't sure, and in
18 the end, it can be either or. And I think this was,
19 from a public health message, I think it was really
20 important, because it shows that if you want to spend
21 less time, you can do it more intensely. So, it means
22 that it makes it a little easier for those of us that

1 have challenging jobs to get some of these, meet some
2 of these deadlines. And that -- or these
3 recommendations. And that muscle strengthening
4 activities involve all major muscle groups should be
5 performed on two or more days of the week, and there is
6 a variety of ways to get those activities as well.
7 Additionally, five hours a week of moderate intensity,
8 or two-and-a-half hours of vigorous activity, or an
9 equivalent combination for even greater additional
10 health benefits, especially for people with some
11 chronic conditions, and especially with issues around
12 weight control.

13 So with older adults, the same exact guidelines as
14 for adults, but when not possible, be as physically
15 active as someone's ability and conditions will allow,
16 and this goes all the way up through the oldest of old.
17 And do exercises that maintain or improve balance, if
18 the person is at risk for falling. There is some very
19 good data for individuals who are at risk for falling
20 for reducing falls. And those without chronic
21 conditions and symptoms do not need to consult a health
22 care provider about physical activities. So this

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1 barrier for older adults, especially that they have to
2 see a physician, that we didn't see any evidence that
3 that was absolutely necessary.

4 So some additional considerations, other subgroups
5 of the population and Physical Activity Guidelines
6 included persons with disabilities. Jim Rimmer, who
7 was fabulous, really took this on, talked about a lot
8 of small research studies that are out there, but
9 really compiled a lot of good evidence, and that women
10 during pregnancy and post-partum period, the guidelines
11 for them are really no different; however, they just
12 have to monitor themselves. And that adults with
13 select chronic conditions, especially with arthritis
14 and osteoporosis, we dealt with as well.

15 So a little bit about sort of -- I have to say, we
16 spent a lot of time on the weight control issue. What
17 I will focus on for just a second is the bottom line
18 here; is that the one thing that we, you know, it's --
19 I don't want to say, duh -- but, it was so important is
20 that you really cannot look at physical activity and
21 weight control unless -- without considering dietary
22 intake and energy intake. And so, what we did and what

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1 I hope this Committee will do also is we really said
2 that you have to consider nutrition; and in fact, if
3 the data show us that if you just add physical activity
4 to someone's life, they don't lose weight because they
5 end up compensating by eating a little bit more, so you
6 really -- there has to be a dietary intervention when
7 you are talking about weight loss. So, in terms of --
8 we separated things out into three different
9 categories; weight stability; weight loss; and weight
10 stability after weight loss. And the data on weight
11 stability -- and, Xave, I think this will be the big
12 challenge for us and I don't think we need to look at
13 this data again, because we just have -- we can refer
14 back to the report -- but that weight stability over
15 time, it's really almost impossible to do a well-
16 designed, randomized controlled trial, because you are
17 talking about trying to measure no change, and you are
18 talking about large groups of people, but the
19 preponderance of the data when we looked at it, it's
20 somewhere in the vicinity of 150 minutes of moderate
21 activity or 75 minutes a week of vigorous activity is
22 helpful for people in terms of weight stability, but

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1 again, you can't isolate it out of dietary intake.

2 In terms of weight loss, we are talking about
3 larger amounts of physical activity; and in terms of
4 weight stability, it's even more physical activity.
5 But again, this is where you need to tie the dietary
6 intake together with that.

7 So the sort of -- the theme here is be active your
8 way. I think that one of the important parts is that
9 there is a variety of ways to get physical activity
10 into your life; be active, healthy and happy. The
11 mental health outcomes here were also great. The
12 website is listed here. A couple things I want to say
13 is, first, I really want to thank Rick Troiano, who is
14 in the audience today, because he was the one who
15 really put these slides together. I just edited them.
16 Our whole Committee was provided with slides around the
17 process, so don't think that I was so clever as to be
18 able to put these all together.

19 But, the other thing, a couple of things that I
20 think were important potentially for this Committee as
21 we go into sort of what I would call an evidence-
22 informed process, there was so much different data that

1 we looked at for our Committee, and one thing that we
2 did -- and you can look at it later -- but, in the
3 summary chapter, you have it in part E, page actually
4 E-2, this is in section 4 of your binder, we put
5 together a table, which was a way so that we could,
6 across all of our subcommittees, categorize the kind of
7 science, the research that we were looking at. So we
8 weren't individually coming up with qualitative ways of
9 discussing our research. We -- this was - this sort of
10 comes out of more of the pharmaceutical or heart
11 disease kinds of way of looking at research, but we
12 sort of massaged it a bit so that it could be used for
13 physical activity, and I think we could also use it
14 around nutrition. It's table E-1. So we are looking
15 at the type of evidence, and we categorized the
16 evidence into four different types of evidence, and
17 then we looked at the strength of the evidence.

18 So, in fact, for example, Type 2 could be
19 randomized controlled trials or meta analysis with
20 important, with some limitations or non-randomized
21 clinical trials. Type 1 is randomized controlled
22 trials without major limitations. Type 3 is well-

1 designed prospective cohort trials or other
2 observational trials. And Type 4 is much more around
3 sort of anecdotal evidence or expert opinion, and then
4 the strength of the evidence either is strong
5 consistence across studies; moderate or reasonably
6 consistence across studies; or weak or limited
7 inconsistence across studies and populations. And this
8 was a way for us to categorize the research across
9 subcommittees, and I would hope that we could possibly
10 do a sort of thing as this. We spent a lot of time
11 around this, because again, there was just so much
12 different types of data that we were looking at as
13 just an idea.

14 So, I am happy during the discussion to answer
15 more questions, but I really -- I think it will be
16 important -- I don't believe that this Committee needs
17 to re-evaluate all the physical activity data over the
18 last 14 years, because we have just done it and the
19 report -- there is a lot of information in the report,
20 and I'm happy to be that link between the two
21 committees.

22 DR. VAN HORN: Excellent. Thank you.

1 DR. WILLIAMS: Thank you very much. In my
2 presentation this morning, I would like to address some
3 issues related to childhood obesity and energy balance.

4 We all agree that children need a high quality
5 diet that's sufficient in energy and nutrients, as well
6 as adequate daily physical activity, in order to
7 maintain health during childhood, and also to prevent
8 risk of future chronic disease.

9 One of the most disturbing public health trends in
10 the past few decades, however, has been the increase in
11 obesity in youth. Although the increase in obesity has
12 also affected U.S. adults, and indeed many populations
13 around the world, the magnitude and rapidity of the
14 increase among U.S. children and adolescents has been
15 significant enough to label it an epidemic.

16 Since the late 1970s, the prevalence of obesity
17 has increased more than 100 percent in children six to
18 11, and 200 percent for adolescents 12 to 19. It's
19 generally accepted that both genes and environment
20 contribute to obesity risk, but since the increase in
21 prevalence of obesity was too rapid to be explained by
22 genetic drift, the consensus is that it's the result of

1 a shift in energy balance.

2 Since Dr. Nelson addressed issues related to
3 physical activity and obesity, I'll focus on some
4 changes in dietary intake of children over the past 25
5 years; changes that may have contributed to energy
6 imbalance. A closer look at the prevalence rates for
7 childhood obesity reveal the changes since the 1960s;
8 in the beginning, the slow and almost imperceptible
9 increase before 1980, and then the rapid increase over
10 the next two decades. Here in bar graph form is the
11 same data. You will note that there is some overlap in
12 the dates for the most recent surveys depending on
13 which time points were included in the analyses and
14 reported in the three most reports in the JAMA
15 articles; 2004, 2006, and most recently, in January
16 2008.

17 When we take a closer look at the changes in the
18 prevalence rates, there may actually be three time
19 periods of interest. Following the first period of
20 very slow increase between 1963 and 1980, there was a
21 period of very rapid increase between 1980 and 1999.
22 Since then there is some recent evidence that perhaps

1 the epidemic has begun to slow down or even begun to
2 decrease. During the most recent period, there seems
3 to be a change. When the prevalence rates for 2005 to
4 2006 were examined, they found that they were not
5 significantly different from the rates for 2003 or
6 2004, so that they were combined. There was actually a
7 small, but non-significant decrease in overall
8 prevalence of overweight for 2 to 19-year-olds; from
9 17.1 percent in 2003 to 2004, to 15.5 percent in 2005
10 to 2006, and this was somewhat exciting, because up to
11 this time rates had just continued to increase. The
12 decrease, however, was seen only among non-Hispanic
13 white youth and not among minority children.

14 More about those recent changes. During 1999 to
15 2002, and even through 2003 and 2004 it appeared that
16 the prevalence rates were still increasing, but now
17 that the latest figures for 2005 and 2006 have been
18 added, there is a suggestion that perhaps a change has
19 begun and the epidemic is beginning to level off. In
20 fact, analysis of prevalence rates showed no
21 significant trends between 1999 and 2006. The authors
22 caution, however, that data from 2007 to 2008 will be

1 needed to further examine the trends.

2 So I think its interesting to look at changes in
3 children's diet relative to the time periods when
4 obesity rates were increasing very slowly, and then
5 during the periods of rapid increase between 1980 and
6 1999; and eventually to look at dietary changes in the
7 last seven years, between 1999 and 2006, when rates may
8 have leveled off, or there is at least a suggestion of
9 that.

10 Have there been measurable changes in energy
11 intake in children and youth since the early 1960s?
12 What happened to children's dietary intake between 1980
13 and 1999 when the -- during the period of most rapid
14 increase in childhood obesity; and are there any recent
15 healthful dietary trends that may be contributing to a
16 slow down in the childhood obesity epidemic?

17 The question about energy intake among children;
18 what do the national surveys show, and there is a lot
19 more data that can be added to this chart, but if you
20 look first at the, some of the data from the most
21 recent surveys, 1999 to 2005 and 2006, it looks as if
22 the energy intake is fairly stable during this time.

1 The previous analyses show mixed results; Troiano
2 compared energy intake between NHANES I and NHANES III,
3 and the only change noted was an increase for
4 adolescents, especially adolescent females. But it was
5 difficult to explain this since the increase in obesity
6 over that time had occurred in all age groups, not just
7 teenage girls. (Inaudible) compare energy intake
8 for children between 1977-1978 and 1989-1991; 1994-1998
9 surveys. In this comparison, total energy intake had
10 increased with similar changes for children of all
11 ages. They also reported shifts from at home to away
12 from home food consumption, and from meals to snacks.
13 There are, of course, a lot of methodologic
14 difficulties in assessing dietary intake in children,
15 especially with collecting proxy data from parents and
16 caregivers.

17 Here is a closer look at some of the data between
18 CSFII 1989-1991 and 1994-1995. Aside from looking at
19 energy intake, what about changes in children's food
20 patterns and food choices since the late 1970s? Just
21 briefly, when the dietary intake of children in 1977-
22 1978 was compared with recent intake in NHANES 2001-

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1 2002, some key changes were noted; beverage preferences
2 have changed significantly; children and teens who
3 consumed fruit juices, fruit drinks and ades and soda
4 were drinking more of these beverages in 2001-2002 than
5 they did in 1977-1978. Higher fat food choices had
6 also increased. For the beverages, the changes over
7 the past 25 years since 1977-1978 and NHANES 2001-2002
8 for children six to 11, milk has decreased from 61
9 percent of total beverage intake to 33 percent; soda
10 has increased from 15 to 33 percent of total beverage
11 intake; and for teens 12 to 19, soda replaced milk as
12 the beverage of choice; from 1977-1978, milk made up 51
13 percent of all beverages by gram weight, while soda
14 made up 29 percent. By 2001-2002, this was reversed;
15 soda made up 50 percent and milk 23 percent. The
16 majority of soda, 95 percent consumed by children and
17 teens is regular soda. Fruit drinks increased slightly
18 14 to 20 percent, and fruit juice increased slightly
19 also 10 to 14 percent in children; 9 to 10 percent in
20 teens. Again, if you look at beverage intake among
21 children and teens, you can see the decrease in milk
22 and the increase in other beverages.

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1 Another recent change between 1977-1978 and 2001-
2 2002 has been that higher food choices have increased
3 for U.S. teens and children. Consumption of pizza,
4 tacos and snack food increased dramatically for
5 children and teens between -- in these 25 years. There
6 were large increases in mean intake of savory grain
7 snacks, pizza, Mexican dishes and candy. There was a
8 sizable increase also in fried potatoes, but a decrease
9 in intake of vegetables.

10 So, in general, over the past 25 years that
11 roughly coincides with the rapid increase in childhood
12 obesity, food and beverages choices have changed
13 considerably. Beverage choices shifted from milk to
14 less desirable choices; those which typically have
15 higher caloric content relevant to nutrients they
16 provide showed large gains in popularity.

17 Another major trend during this time was a trend
18 for children to eat more food away from home. Even for
19 two-to-five-year-old children, food consumed at home
20 decreased from 88 percent to 76 percent; and food away
21 from home -- food consumed at home decreased, and food
22 away from home doubled from 12 to 24 percent, and those

1 are for preschool children. This is even greater for
2 teens 12 to 17, where foods eaten away from home
3 increased from 20 to 35 percent over the same period;
4 and for six to 11-year-old children the increase was
5 similar, 21 to 32 percent. The problem is that meals
6 consumed in restaurants are not as healthy as home
7 meals. They are lower in total unsaturated fat and
8 sodium and higher in calcium and iron. Children also
9 are snacking more. I'm sorry -- lower in calcium and
10 iron. Children are also snacking more, tending to
11 replace meals with snacks. Young children gradually
12 increased the number of snacks they eat each day from
13 1.73 to 2.29, so the total energy increased because
14 children are snacking or eating a greater number of
15 snacks each day. The snacking patterns of two to 18-
16 year-old children also increased. More children are
17 snacking now. The average energy intake from snacks
18 increased from 450 to 600 calories, and the energy
19 density of children's snacks has also increased.

20 So the question now is, if the prevalence rates
21 for childhood obesity are beginning to slow down, have
22 food patterns changed since 1999 to 2000? Have there

1 been any improvements? Have there been any changes in
2 energy intake, as well as macronutrient/micronutrient
3 intakes since 1999; or diverging difference among
4 children by race and ethnicity, have choices of
5 beverages changed again? Have the amount of beverages
6 changed recently? Have higher fat food choices
7 decreased? Has there been a change in patterns of
8 eating out or purchasing take home foods? Have
9 economic hard times encouraged more at-home meals? Are
10 children and teens more physically active than they
11 were in 1999? We need a lot more information on these
12 questions, but it's likely based on current knowledge,
13 especially energy balance.

14 The 2010 Dietary Guidelines will need to emphasize
15 key issues that are most likely to help children,
16 adolescents replace some current food choices with more
17 healthful options, including fruits, vegetables, whole
18 grains and calcium-rich foods and beverages. This will
19 be critical in helping children meet nutrition,
20 nutrient requirements and reduce risks of future
21 chronic disease. Thank you.

22 DR. PI-SUNYER: Thank you, Dr. Williams. Now Dr.

1 Perez is going to speak briefly.

2 DR. PEREZ-ESCAMILLA: Good morning. I will talk
3 about recent developments related to gestational age
4 weight gain; maternal and child obesity-related
5 outcomes.

6 In 1990, the Institute of Medicine came up with
7 the actual recommendations for gestation and weight
8 gain, and brought down these recommendations based on
9 pre-pregnancy body mass index. In 1990, the criteria
10 that the IOM Committee used was mostly related to the
11 prevention of small-for-gestational-age babies. And,
12 research by Barbara Abrams and others has shown that
13 these recommendations do work, a very reasonable
14 prevention of small-for-gestational-age weight
15 babies.

16 The problem that we are facing now is that we are
17 now in the midst of a major obesity epidemic that has
18 affected women, to a large extent, and in particular,
19 low income minority women; such as African-American and
20 Latina women.

21 As you can see from the recommendation, in 1990,
22 the Committee decided to recommend that among women who

1 were obese before pregnancy that they should gain at
2 least 15 pounds. Unfortunately, there wasn't a lot of
3 evidence based behind this recommendation, and this
4 recommendation caused -- I mean, has caused an enormous
5 amount of confusion with some coming up with their own
6 recommendations claiming that women should not gain
7 more than 20 pounds all the way to some groups claiming
8 that women should gain less than 15 pounds if they were
9 obese or if they are obese before pregnancy. Because
10 of the context of the major obesity epidemic in which
11 we are, the IOM decided to call for the formation of a
12 committee that is currently reviewing the gestational
13 weight guidelines, and this report is expected to be
14 released in 2009.

15 However, there are materials that can be shared
16 now, because our AHRQ published an evidence-based
17 systematic review on maternal and child consequences of
18 gestational weight gain that is available at this
19 website and that systematic review was released just a
20 few months ago, so, it is very updated.

21 I have identified two key issues that I want to
22 share with the Committee today, and the first one is

1 that it is clear that high pre-pregnancy body mass
2 index is a significant predictor of excessive
3 gestational weight gain and maternal post-partum weight
4 retention and that, in turn, is a significant predictor
5 of serious risks for chronic disease among women.
6 Excessive gestational weight gain independently of pre-
7 pregnancy BMI is a significant predictor of post-partum
8 weight retention, which is a major cycling issue that
9 eventually leads to major obesity problems among women
10 that have multiple pregnancies. High pre-pregnancy
11 body mass index and excessive gestational weight gain
12 are definitely associated with the delivery of large-
13 for-gestational-age newborns, and being a large-for-
14 gestational-age newborn, in turn, increases the risk of
15 childhood obesity, insulin resistance and Type 2
16 diabetes later on in life. So, this issue of
17 gestational weight gain during pregnancy and maternal
18 pre-pregnancy BMI has major implications for maternal
19 health, and also for the childhood obesity epidemic.
20 The implications of this, first the issue is that
21 the fetal nutrition and hormonal milieu is likely to
22 affect the risk of childhood obesity and chronic

1 disease perhaps via epigenetic mechanisms. Secondly,
2 preventing maternal obesity before pregnancy is likely
3 to have a positive impact on both maternal and child
4 chronic disease outcomes. And lastly, I want to
5 mention that many questions remain regarding the safety
6 of promoting weight loss during pregnancy, because with
7 that type of approach we have to take into account the
8 health of the mother, but also potential risks for
9 fetal health if the mom enters a ketogenic status.

10 The second issue that I want to mention is that
11 the AHRQ report fully confirms the 1990 IOM findings
12 regarding inference of maternal underweight before
13 pregnancy, and to what the maternal gestational weight
14 on risk of small-for-gestational-age babies. SGA --
15 since then, however, evidence has been strongly
16 accumulated that SGA is associated also with an
17 increased risk of childhood adiposity accumulation and
18 risk of type II diabetes and cardiovascular later on in
19 life, and there is a good number of studies that have
20 been published since then along the themes of fetal
21 programming or developmental origins of a adult health
22 and disease. And I want to emphasize that the risks

1 for linking SGA with childhood adiposity and chronic
2 disease later on in life seems to be strongly modified
3 by the growth rate during infancy. So I know that the
4 Dietary Guidelines are supposed to look at the U.S.
5 population after two years of age, but it is very
6 difficult to deal with a childhood obesity epidemic if
7 we don't deal with the infant nutritional, early
8 nutritional issues as well.

9 So, the implication of this key issue is that
10 preventing maternal underweight before pregnancy and to
11 what amount the gestational weight gain are likely to
12 also have a positive impact on preventing childhood
13 obesity and subsequent chronic disease outcome. So, at
14 both ends of the spectrum, SGA or LGA, they both appear
15 to have strong implications for the childhood obesity
16 epidemic.

17 And last, but not least, it is my hope that the
18 2010 Dietary Guidelines that impact maternal intake and
19 physical activity during pregnancy should take fully
20 into account the gestational weight gain IOM report
21 that is due next year. Thank you.

22 DR. PI-SUNYER: Thank you, Dr. Perez. Linda,

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1 that's it. We are open for discussion.

2 DR. VAN HORN: Excellent. Well, thank you,
3 everyone. Those were wonderful presentations and I
4 know there will be a rich discussion following all of
5 that. I don't know how you'd like to begin. We can
6 just open it up and take any of the presentations, or
7 do you want to go in order?

8 DR. PI-SUNYER: I think anybody who wants to ask
9 questions or make comments, we'd love to have them.
10 Larry?

11 DR. APPEL: Yes. Those were great presentations.
12 A comment --

13 DR. VAN HORN: Oh, please say your name before you
14 speak.

15 DR. APPEL: Larry Appel.

16 DR. VAN HORN: Thank you.

17 DR. APPEL: I have a -- I would propose a question
18 that you consider, at least on the list, and that is,
19 you know try to make this action-oriented. In terms of
20 behaviors that we use in our weight loss trials; self-
21 measurement of weight; the self-measurement of physical
22 activity; calorie counting and their effects on

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1 preventing weight gain or encouraging weight loss might
2 be questions that, you know, could be answered that
3 would actually have an impact on what people do and at
4 least put those on the list, I would hope. And I also
5 wanted to toss out a concept -- you know, I am sure you
6 thought a lot about the 2005 Guidelines, where we
7 focused on weight and we didn't use calories as an
8 outcome variable that would drive decisions, and you
9 know, I have thought a lot about this. You know if, as
10 you point out, the difference in calorie intake that
11 accounts for the obesity epidemic is 100 calories per
12 day, and if it's multi-factorial -- let's say there are
13 10 factors, each accounting for 10 calories per day --
14 do we have the tools, both epidemiologic -- primarily
15 epidemiologic, to discriminate at that level; and
16 instead of saying, we don't have the evidence, we might
17 want to ask, will we ever have the evidence for, let's
18 say, sugar-sweetened beverages or portion size, and if
19 we don't think we are going to have the evidence, then
20 I think we need to make decisions about whether to act.

21 DR. PI-SUNYER: Yes. Well, I would agree with
22 you. I think it's very difficult when you get down to

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1 under 100 calories to really be able to measure it and,
2 you know, so the evidence isn't really there except in
3 short interventional studies.

4 DR. APPEL: Yes. And we sort of punted saying,
5 well, let's get the evidence, but if it can't, if it
6 never will occur, we might have to make some decisions.

7 DR. SLAVIN: Yes. This is Joanne Slavin. I am on
8 this committee too, but I just want to talk about the
9 Jim Hill stuff, where you look at people that actually
10 are successful and calorie counting and exercising
11 that's what they do. So I think we have -- it's not,
12 you know, exactly the kind of research people are
13 looking for, but it's very clear that if you want to be
14 successful -- and, you know, I always talk about that -
15 - I give the example that french fries at the state
16 fair is 7,000 calories and people eat those french
17 fries. And I say, you've got to walk to Eau Claire,
18 it's 70 miles, so think about. You know, I think
19 linking it is critical to people; there is a price to
20 pay. You might want to eat it right now, but down the
21 line is was it worth it?

22 DR. APPEL: Yes. This also has implications for

1 calorie labeling widespread. You know, if what you
2 people are asked to do; to monitor their weight and
3 monitor their calories, and if they are eating out
4 frequently, you actually have to provide calories for
5 those people who are actually calorie counting.

6 DR. VAN HORN: I'd like to just jump in on that
7 one as well. We were discussing this, Joanne and I, on
8 the way over here, and the whole concept of calories I
9 think is something that we might want to embrace at
10 this point. I personally have heard over the years,
11 well they are too hard to calculate; you know, people
12 won't want to do the math, I mean all of those kind of
13 statements, and I think we have now perhaps a more
14 sophisticated society that, if they can learn to know
15 their cholesterol number they might be able to learn to
16 know their calorie number and what they are supposed to
17 have and how to get them, and how to burn them. So, I
18 would just like to add to that concept of you know
19 perhaps starting to get people thinking more about the
20 energy balance using something more objective, like
21 calories.

22 Sorry. Go ahead.

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1 DR. PEARSON: I think -- this is Tom Pearson --
2 all of you dealt tangentially with the issue of this
3 paradox of, as education and income levels go up the
4 obesity levels go down and this apparent competition of
5 food insecurity programs on the one hand and over
6 nutrition on the other. So I guess one of the
7 questions I had is that there -- is there a literature
8 and evidence base to look at the issue of if you attack
9 the obesity issue in one way, you don't affect the
10 under nutrition issue in another way? We are getting
11 into some economic difficult times. These are some
12 times in which you are going to have larger portions of
13 the U.S. public in need of some food assistance, and so
14 what you have at the same time of this co-existence of
15 the obesity epidemic somewhat of a historically and
16 unusual situation in which you are going to have two
17 mega trends coming together where you want to actually
18 want to reduce consumption at the same time as you
19 really historically are going to be looking at people
20 who are really nutritionally and adequately taken care
21 of. So I guess the question is, what is the evidence
22 base that you can actually do both of those

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1 successfully?

2 DR. PI-SUNYER: Well I think that we can certainly
3 look at that. Dr. Post, do you have any thoughts on
4 that, in terms of the U.S. Department of Agriculture
5 and their programs, and you know, what happens to a WIC
6 woman when she gets pregnant and is getting WIC food?
7 Does she gain more weight when she is pregnant, or do
8 we have any data on things like that?

9 DR. POST: There are some data. We could organize
10 that for you in the next, in the interim certainly and
11 get that to you and this subcommittee, in terms of what
12 we do have.

13 DR. PEARSON: Yes.

14 Dr. POST: Working with the food and nutrition
15 source.

16 DR. PI-SUNYER: Because I think it's an
17 interesting question. I have often thought that we
18 could solve the obesity problem if everybody went to
19 college and got a certain minimum income.

20 DR. APPEL: You may be right.

21 DR. NELSON: But not so much anymore, I don't
22 think.

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1 DR. VAN HORN: Rafael?

2 DR. APPEL: It would be easier.

3 DR. PEREZ-ESCAMILLA: I just want to mention, you
4 know, the food insecurity problem in the U.S. is mostly
5 related to dietary quality and not to quantity, and
6 that in fact, at least among adult women, there is
7 really consistent evidence that food insecurity is
8 related to obesity, not to under weight. So, that's a
9 context in which food insecurity is happening in the
10 U.S.

11 DR. FUKAGAWA: Two comments. I was really -- this
12 is Naomi Fukagawa -- really heartened by the trends in
13 pediatrics and seeing that it potentially is leveling
14 off, which means that somewhere we have been effective
15 at teaching and enforcing the message in at least the
16 age group that is amenable to changing their behavior.

17 One of the questions that I have is, do we have
18 any data as to how the general public where this
19 epidemic is occurring, putting aside you know the
20 concerns about the pediatric age group, as to whether
21 or not they perceive it as a problem? You know, we are
22 telling them that obesity is an epidemic; it's going to

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1 hurt them; you know, there health is going to go
2 downhill sooner rather than later, but --

3 DR. PI-SUNYER: Well I think this relates to Dr.
4 Pearson's point.

5 DR. FUKAGAWA: Yes.

6 DR. PI-SUNYER: I think a lot of people,
7 particularly some minority groups and some lower
8 socioeconomic groups have higher, other priorities that
9 are much more -- that are pressing on them much more
10 than their weight, and so they may have some sense that
11 15 years down the line they might get diabetes, or 20
12 years down the line they might get a heart attack, but
13 that's a long way away and right now they have all
14 these pressing issues, and so the priority is not as
15 high, you know, as maybe it should be in terms of their
16 future health.

17 DR. WILLIAMS: Chris Williams. I'd also like to
18 say that working in the clinical field there is always
19 a lag between the time that materials are available for
20 counseling minority families; and especially in Spanish
21 and other languages. Working with minority families in
22 the area of Columbia, you are always struggling to find

1 materials, because there is a lack of materials for
2 families like that.

3 DR. PI-SUNYER: Again -- excuse me -- in New York,
4 you know, in the public school systems there are 187
5 languages that are being, as second languages, that are
6 being taught.

7 DR. VAN HORN: Shelly.

8 DR. NICHOLS-RICHARDSON: Sharon Nichols-
9 Richardson, just in response to Tom's question. The
10 data that I presented yesterday was taken from the
11 reports on the food stamp program and the school lunch
12 program, and those data were broken down into the
13 different groups. And, if I had to boil that down to a
14 single message, there is not much difference based on
15 participation versus non-participation versus a higher
16 SES status, in terms of diet quality, related to the
17 nutrients that were presented yesterday.

18 DR. ACHTERBERG: Cheryl -- Cheryl Achterberg. I
19 just wanted to toss in the comments too that I think
20 the Committee should look at the data around food
21 variety, and I know that's hard to measure, I know it's
22 difficult, but look at it in the context of these

1 eating patterns that lead to weight gain or not lead to
2 weight gain, especially in terms of the development of
3 childhood obesity. You know, is it a function, in
4 other words, of a harried mother coming home and just
5 throwing a bag of chips at a child and the child eats
6 the whole bag and that's the extent of variety, or if
7 in fact they do consume more variety, is that
8 associated not only with nutrient adequacy, but
9 ultimately a lower calorie intake?

10 DR. NELSON: This is Mim Nelson. I really
11 wholeheartedly support the self-monitoring piece. I
12 think the data is so strong, and it's not just from
13 the, you know, the Jim Hill's work. So, I really
14 wholeheartedly support that.

15 Two other sort of comments. Xave, I am not sure
16 about this linking sort of discretionary calories with
17 physical activities, and I'll tell you why. Because
18 most of the research that we just looked at, when you
19 -- and it could be -- there may be a message in there
20 that we could use, but the problem is most of the
21 research shows that when people increase their physical
22 activity by 20 minutes a day or 30 minutes a day they

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1 don't lose any weight. So, we just have a natural
2 propensity to eat a little bit more to maintain
3 whatever body weight we have. It may help with not
4 gaining weight, but I think that we may be overstating
5 what the research says about if we just increase
6 physical activity a bit, we'll be okay. So, I am a
7 little bit concerned about -- because we just naturally
8 increase our energy intake, although certainly we need
9 to promote physical activity. But the other is -- and
10 this gets back to I think some stuff that Larry said
11 yesterday about some of the cultural, you know,
12 differences that are out there, other cultures. I
13 mean, I am thinking of Japan, and they have this sort
14 of cultural saying, hara hachi bu that is around eat 80
15 percent of what you need. You know, try to stop before
16 you are 100 percent full. And I think that it's really
17 indoctrinated into the culture about you just don't eat
18 until you are full, and I don't think -- I wonder if
19 there are ways to -- you know, when we do weight
20 control studies or weight loss, we talk about trying to
21 just eat a little bit less than you need. And that --
22 it might get to calories, but basically we have to eat

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1 less, and that's I think a message and I think there is
2 some research to back that up that's very important,
3 so.

4 DR. PEREZ-ESCAMILLA: Can I go back to Naomi's
5 question, because I think it's a very important one.
6 You know, my research group has done extensive work
7 with the Puerto Rican community in Connecticut, and
8 this is a community that has been decimated and
9 continues to be decimated by the Type 2 diabetes
10 epidemic, and we have found that they are very aware,
11 the vast majority of them, that it's not a good idea
12 for their kids to be obese and that that is indeed a
13 risk factor for Type 2 diabetes. But, as it was
14 mentioned by Xavier before, there are other priorities,
15 there are major barriers that keep them from doing the
16 things that need to be done for their kids to not be
17 obese. And you know some may be difficult choices, but
18 a lot of them are structural system level type of
19 barriers; and if we don't understand what those
20 barriers are, it's very difficult to make progress in
21 addressing childhood obesity in these communities.
22 But, it is not that they think it's great for their

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1 kids to be obese.

2 DR. NELSON: Can I just respond? Because --

3 IOM has just convened a committee on looking at

4 community factors for childhood obesity prevention, and

5 I'm thinking a lot about some research that a close

6 colleague of mine at Tufts is doing around really sort

7 of environmental interventions, and it's really so that

8 choice is not an issue; it's really that the community

9 is less obesogenic, and it works. And so I think that

10 there are ways that hopefully the policies that come

11 out of this could help with sort of community action.

12 I am thinking about the Physical Activity Guidelines

13 Committee and I show this lovely brochure -- you can

14 see I am doing a big ad for our -- but this is a, you

15 know, a community guide for trying to figure out the

16 built environment, connectivity and things like that,

17 and I really like to think -- I mean, Xave, you brought

18 some other, you know, it's not just about what we eat,

19 but it's also how we eat, and so if we can sort of

20 think creatively about that so that the parent isn't

21 having to make these choices, it just naturally is

22 healthier for the kid to be there. I think there is

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1 good data now and I am happy to share some of our
2 current research on these sort of environmental change
3 interventions that work around nutrition and physical
4 activity.

5 DR. VAN HORN: Okay. I think we should be moving
6 along, but in keeping with the pledge I made yesterday
7 and hearing about all this physical activity, I want
8 everyone to stand up for two minutes, just shake
9 yourself out, take a deep breath, stretch, do whatever,
10 and then we'll start on carbohydrates.

11 (Whereupon, at 9:49 a.m., a brief recess is
12 taken).

13 DR. VAN HORN: All right. Next, we are going to
14 be talking about the role of carbohydrates on health
15 and Dr. Slavin is going to address this topic, and she
16 pretty much has got the show on this one. So, go for
17 it.

18 DR. SLAVIN: Thanks, Linda.

19 DR. VAN HORN: Yes.

20 DR. SLAVIN: All right. My outline -- what I was
21 told to do, and I am from Minnesota so we are very
22 compliant -- we just follow the rules -- was to first

1 summarize the recommendations in the 2005 Dietary
2 Guidelines relative to carbohydrates; do a little bit
3 on the background and then what new information has
4 happened since the 2005 Dietary Guidelines that we'll
5 need to put into our review and consideration.

6 So, Dietary Guidelines for Americans 2005 choose
7 fiber-rich fruits, vegetables and whole grains often.
8 I don't think there is -- I've heard much disagreement
9 to that. Choose and prepare foods and beverages with
10 little added sugars or caloric sweeteners, such as
11 amounts suggested by the USDA Food Guide and the DASH
12 Eating Plan. I am going to talk some more about what
13 has been happening since that point. And then the
14 third one; reduce the incidence of dental caries by
15 practicing good oral hygiene and consuming sugar and
16 starch-containing foods and beverages less frequently,
17 which is also a known and could easily flow right into
18 the 2010.

19 All right. Some other things in the 2005
20 carbohydrates part of the healthy diet; positive
21 association between consumption of sweetened beverages
22 and weight gain -- a study that had come out in 2004,

1 I'll talk a little bit about -- reduced intake of added
2 sugars may be helpful in weight control and achieving
3 recommended intakes of nutrients, and this is kind of
4 the discretionary calories idea that if you don't have
5 any discretionary calories there is not going to be
6 much room for the sugars; and then to reduce the risk
7 of coronary heart disease and promote laxation
8 recommends intake of 14 grams of dietary fiber per
9 thousand K-cals, which came out pretty much directly
10 from the DRIs.

11 All right. I'm going to do a little background
12 and I have to thank my friend, George Fahey. We do
13 this talk together and he and I have put it together,
14 so some of it may have come from him, so I thank him
15 for that.

16 Looking at carbohydrates we usually talk about
17 chemistry. And remember too, carbohydrates are done by
18 difference, so even though carbohydrates I think are
19 really important, it's half our calories, very few
20 people -- you know, measurement -- there are a lot of
21 issues. But, from a chemical perspective, we have
22 monosaccharides, which really don't occur in foods

1 much; sugar alcohols, which we add to food;
2 disaccharides, you know, lactose, sucrose, which are
3 the sugars we eat; tri's, which are pretty uncommon;
4 oligos, three to ten glucose or carbohydrate units; and
5 then polys are bigger than that. So it's mostly this
6 chemistry idea, which is good, but it doesn't help us
7 much when we get into dietary guidance. So, when we
8 look at the nutritional perspective, what else can we
9 talk about with carbohydrates? Do they get absorbed?
10 Do they -- you know, do they get into the body? Are
11 they digestible? The bigger ones, anything past a
12 monosaccharide if it's going to get in, it needs to
13 first be broken down so we can get it in. Once it gets
14 down to the large intestine, the formidability of
15 carbohydrates. And then there are certain fibers or
16 other non-formidable carbohydrates that nothing
17 happens; they pretty much goes straight through. So,
18 if you think of eating sand, if you have a baby in a
19 sand box, it comes through and there are some
20 carbohydrates that end up in the diaper. Nothing
21 happens. They just go along for the ride.
22 Absorbable carbohydrates that don't require

1 digestion are the monosaccharides; glucose, galactose
2 and fructose. They don't occur naturally in foods so
3 they really aren't any of those, or they are very small
4 amounts, in general, very small amounts. A little bit
5 of fructose. We add fructose to foods, so there is
6 going to be some, but typically those are not -- there
7 is not much of that naturally occurring in foods, so
8 there is not much exposure to that. Digestible
9 carbohydrates are the ones that get enzymatically
10 digested. So, you've taken lactose, it gets broken
11 down. Sucrose, the same thing. And remember, you
12 know, lactose, you know, the components of lactose;
13 glucose, sucrose, fructose, glucose, and then just
14 maltose being the disaccharide of starch. And that's
15 most of what -- the carbohydrate exposure we have is
16 starch; most of what we eat typically.

17 The whole thing of fermentable are ones that get
18 down to the large intestinal tract -- so lactose and
19 sucrose perhaps sometimes. If you are lactose
20 intolerant, they definitely get there. Pectin, some of
21 the other fibers; beta-glucans; psyllium; gums;
22 oligosaccharides, those three to tens; some of the --

1 you know, things that are in beans, onions that cause
2 intestinal gas; and then just resistant starch. So
3 starch can -- you know, as long as you digest it and
4 absorb it, you get the glucose. If it's resistant to
5 digestion, absorption, it gets down to the large
6 intestinal tract, functions similar to fiber. Then
7 there are some of these celluloses hemicelluloses.
8 There are some resistant maltodextrins that nothing
9 happens. They really do go in and come out. And, you
10 know, they may absorb some water along the way,
11 increase stool weight, but really nothing else happens.

12 This is another big thing. I wanted to just talk
13 about glycemic versus non-glycemic, and up on the top
14 -- a little hard to see here -- but glycemic, we talked
15 about the free sugars getting metabolized by the liver.
16 There is some thought on lipid metabolism in fructose.
17 A lot of that is in animal studies. Maltodextrin, you
18 know, these starch components and they are either
19 rapidly available or slowly available, and we'll talk
20 more about that; how important that is to glycemic
21 index.

22 All right. And then the whole non-glycemic and

1 the fiber committee dividing it into total fiber; both
2 dietary fiber, which is naturally occurring in foods;
3 and functional fiber, which is isolated fibers and
4 those varied a lot. So, you know, if you look at total
5 fiber, they are very different compounds, so -- but the
6 dietary fiber is a food matrix, so it's actually food -
7 - you know, it has to be in food for it to be dietary
8 fiber. Functional fiber is just fibers we put into
9 food. Go down the way, oligo saccharides, they can
10 both be intrinsic, so if you look at wheat, onions,
11 there is a lot of oligo saccharides naturally
12 occurring; beans obviously -- you can think of things
13 that cause intestinal gas, and that's them. And we can
14 also add those to foods. So oligo saccharides can be
15 added to food. And then just sugar alcohols, another
16 food additive that functions similar to these, you
17 know, they can get fermented and cause intestinal gas
18 and other problems. So there is a lot of exposure to
19 carbohydrates, and this is a slide which just tells you
20 that everyone is really different.

21 So chemical identity we talked about already; the
22 food matrix, how it's put together, and then just how

1 it's consumed in other foods, obviously meal factors,
2 other properties of the carbohydrate, individual
3 variability, and everybody's gastrointestinal tract is
4 different, so we do see some differences there. But,
5 it's either going to get absorbed in the small
6 intestine and then we are going to get glycemic or
7 calories from it, or it's going to get into the large
8 intestine and get fermented and also cause some
9 potential negatives, but a lot of potential positives.

10 So why are carbohydrates important? Why are they
11 half of what we consume? Well, they are sweeteners;
12 food preservation -- you know, some of the discussion
13 this morning was salt and sugar in foods. There are
14 some advantages for sugars -- you know, think of jam,
15 how long it can sit around because the sugar is tying
16 it up. Functional attributes, viscosity, texture,
17 body, browning capacity; a lot of the foods we like
18 because of the carbohydrates have these components in
19 them; energy, and then just this fermentation; what
20 goes on in the large intestinal tract.

21 Is there a carbohydrate requirement? If you go
22 back in nutrition and if you are as old as I am, you

1 can go back and say there is no technical requirement,
2 because we can make carbohydrate from protein. So
3 technically, you could take protein, deaminate it and
4 use it for gluconeogenesis; but, if you go into the
5 2002 DRIs they thought about it some more and said,
6 actually we know that there are parts of the body that
7 do require carbohydrates, so let's put some
8 carbohydrates in, as required, so 130 grams a day, a
9 very small amount, in the DRIs. We don't know exactly
10 -- if you look how much do we need, we don't know
11 exactly how much is necessary for optimum health, but
12 we know -- we had a little discussion about ketones
13 already; that carbohydrates are a good source of
14 energy, and that if there are none around, you do
15 become -- use ketone bodies in breaking down body fats.
16 So, it's worth knowing about that.

17 All right, glycemic index. I'm going to start on
18 some different parts that were within the 2005 and
19 discuss that. If you look at glycemic index, it really
20 gets into that glycemic response. So you eat a
21 carbohydrate and what happens? We always want to
22 measure something. So the nice thing about the

1 glycemic index is it's something we can measure. So we
2 bring in people that are fasted, we feed them a certain
3 amount of the food that associates with the amount of
4 carbohydrate and we look at glycemic response relative
5 to a control, and a control is going to be glucose or
6 white bread; a glucose compound that's quickly
7 absorbed. So you can see white bread gets a score of 1
8 there. So that's our base and then we compare
9 everything to white bread. And if you go up and down,
10 potatoes and rice typically are the ones that are
11 digested and absorbed more quickly, they get a higher
12 score, which is typically considered not positive. And
13 if you go down the line, mixed foods like cheese pizza
14 are going to be less; sucrose is going to be less,
15 because it has some fructose in it, and fructose alone
16 is going to be a lot less. So glycemic index is really
17 driven by the glucose and fructose content of that food
18 and how quickly it's digested.

19 Glycemic load really just corrects for serving
20 size. So carrots -- whenever I give my sports
21 nutrition talks, people say, are carrots bad because of
22 their high glycemic index? And, no, carrots aren't

1 bad, but the food, the carbohydrates in them is mostly
2 starch glucose, so they are going to be higher than
3 fructose. So when you actually calculate out glycemic
4 load, you can see carrots have a low glycemic load just
5 because the serving size is corrected for in glycemic
6 load. And, glycemic load is really a good indication
7 obviously of calories too, so.

8 So, if you go back to the definitions and the
9 recommendations, there is in the DRIS a UL, where there
10 was not a UL based on glycemic index; and it said
11 because the critical mass of evidence necessary for
12 recommending substantial dietary change is not
13 available. There wasn't enough information. But, they
14 did say, principle of slowing carbohydrate absorption
15 is potentially important, further research is needed.

16 This, because of the emerging science, is
17 something to consider, types of carbohydrate or
18 characterizing their glycemic potential is of interest.
19 And I think we definitely see a movement towards types
20 of carbohydrates. We know carbohydrates are a really
21 diverse group, and how to put them into groups that are
22 useful to get at physiological effects and help us come

1 up with recommendations we want to make for
2 carbohydrates.

3 Glycemic response, lots of things affected, just
4 the types of starch that are in the food intact. Large
5 particle size will affect it, if you can't digest it
6 and absorb it, it's going to slow down the process, so
7 if it's intact -- raw starch, you know, the effect of
8 cooking. If you don't cook starch, you eat a raw
9 potato, see what it does to you. It's not good. You
10 don't break it down. You don't get any calories from
11 it, but you get other problems. And just the
12 interaction, the more complicated the food is, you are
13 going to get a lower glycemic response. But there is
14 no recommendation or UL for glycemic index or glycemic
15 load.

16 So what did we get in the DRI report? We have an
17 RDA for a carbohydrate; this acceptable macronutrient
18 distribution range we'll talk about; there is a
19 recommendation for added sugar consumption, but no
20 recommendation for an upper limit for a glycemic index
21 or glycemic load. So, acceptable macronutrient
22 distribution ranges, carbohydrates are most of your

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1 calories, so it's really important -- I always feel
2 like I am the lone person -- here I've got the market
3 share of the calories and nobody cares about me. It's
4 really sad. So maybe I can convince people they should
5 care. But, the recommendation is 45 to 65 percent of
6 calories should come from carbohydrates; and then
7 lipids 20 to 35; proteins, 10 to 35. Why -- where did
8 we get to these levels? We know that below 45 percent
9 I am not going to get my adequate intake for fiber, and
10 based on our discussion yesterday, fiber continues to
11 be a problem. So, getting people to eat more
12 carbohydrates is an important part of getting them to
13 eat the fiber that they need, and just the -- you know,
14 like at the end of the day, we've got carbohydrates,
15 lipid, protein and alcohol, so take a pick. I mean,
16 where do you want to put them in? So, if carbohydrates
17 go down, lipids and protein have to go up. So, there
18 is no real reason to do that. Higher than 65, if you
19 go really high, there is some data on the high
20 triglycerides people get concerned about, and that you
21 decrease fat and protein to too low of levels.

22 Added sugars -- this is very controversial and

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1 difficult, because this is considered sugars and syrups
2 that are added to foods during processing or
3 preparation; soft drinks, cakes, cookies, pies, dairy
4 desserts, candy. You have heard from Christine that
5 that's a lot of what people consume and kids too.
6 Recommendation for added sugars is that they not be
7 more than 25 percent of total calories. And this is
8 looking at -- this is from the DRI report -- looking at
9 added sugar intake combined with nutrient intake data.
10 This is being reassessed in other groups. But, if you
11 look at this calcium intake as they get beyond 25
12 percent of their calories as added sugar, calcium
13 intake goes down; so it's based on that calculation;
14 that if we get sugar above 25 percent of total
15 calories, then it's hard to get nutrients. The
16 significance of added sugar to human health, this was
17 from the 2005 Dietary Guidelines, so they suggested
18 there was a positive association between the
19 consumption of sugar-sweetened beverages and weight
20 gain, a reduced intake of added sugar, especially
21 sugar-sweetened beverages may be helpful in achieving
22 recommended intake of nutrients and in weight control.

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1 So that's right out of the 2005, and it really was
2 based on the study that came out, that's quoted, or
3 that's one of the data points. And this is the nurse's
4 health study where they reported that those who
5 increased their sugar-sweetened soft drink consumption
6 from low to high, which was really one, less than one,
7 greater than one, had more weight gain. So, higher
8 consumption of sweetened, sugar-sweetened beverages,
9 this is associated with greater weight gain and
10 increased risk for Type 2 diabetes.

11 So, discretionary calories really fits into this
12 category here. Added sugars fit into the category of
13 discretionary calories, because they are part of the
14 difference between a person's energy requirement and
15 his essential calories. Persons who are sedentary have
16 very few discretionary calories, and I think you have
17 heard about that already. The Energy Committee that --
18 but maybe this concept is too confusing and not
19 helpful. You know, it seems pretty easy, but probably
20 not.

21 So, where do we end up on dietary carbohydrates?
22 We have an RDA, 130 grams a day; we have a range of

1 intakes, 45 to 65 percent; added sugar, 25 percent or
2 less; and these are existing dietary guidelines DRI-
3 type recommendations; dietary fibers based on K-cals,
4 38 grams for men; 25 for women; and pretty, you know,
5 good support that carbohydrates really are the primary
6 calorie source; that they are less expensive. There is
7 no reason for us to have less than about 50 percent of
8 our calories as carbohydrates. So then the challenge
9 is just managing that 50 percent of our calories, how
10 to make that the best.

11 So we are going to talk about fiber, of course.
12 What is fiber? Carbohydrates and lignin that escape
13 digestion but may get fermented in the gut. According
14 to the 2002 DRIs, it's a nutrient, so fiber moved up.
15 On the nutrition facts panel, 25 grams is the daily
16 value. A marker of a healthy diet -- overall, when we
17 look at healthy diets and a lot of diseases they are
18 plant-based diets, which are higher fiber diets. So
19 fiber tends to be protective for a lot of diseases, and
20 fiber does come along with other things. So whenever
21 -- you know, maybe it's just a marker. Fiber itself
22 may not be the active component. We do have health

1 claims for oats, barley and psyllium, for their
2 cholesterol lowering. I already told you that the IOM
3 separated out dietary fiber and functional fiber and
4 dietary fiber is non-digestible carbohydrates and
5 lignin that are intrinsic and intact in plants. So
6 that's saying we want people to eat a plant-based diet;
7 they should get their fiber from plant foods.

8 Functional fiber can come from anything, so it's the
9 isolated non-digestible carbohydrates that should have
10 a beneficial effect. It can come from plants; it can
11 come from bacteria; it can come from yeast; total fiber
12 is the sum of those.

13 Issues -- some of the fiber issues intact and
14 naturally occurring in foods -- this makes it kind of
15 hard to measure and put on a label. It's more of a
16 concept than it is something we can put a handle on
17 better than that. Go back to the 1970s, the dietary
18 fiber hypothesis was based on populations consuming
19 unrefined diets that were high in fiber and slowly
20 digested carbohydrates, so different attributes to
21 these diets. Fiber has lots of biologically active
22 compounds, and we know that fiber within the plant's

1 cell structure is handled differently in the body than
2 isolated fiber, so fiber is not all alike, just like
3 carbohydrate is not all alike.

4 The recommendation for 14 was based on this data
5 showing protection from coronary heart disease, so
6 these were three large perspective studies that were
7 put together in this data set, and you can see as fiber
8 intake goes up relative risk of coronary heart disease
9 goes down. This is just from the -- this was from the
10 DRI. We have this recommendation of 14 grams of fiber
11 per thousand K-cals. There is no recommended UL for
12 total fiber, so when the DRI committee got together, we
13 know that there are occasional adverse GI symptoms --
14 that's gastrointestinal, not glycemic index -- observed
15 when humans consume isolated or synthetic fibers. So,
16 this is a review we just published. In looking at
17 different fibers in high enough concentrations they
18 cause problems. When the DRI committee thought about
19 that, they said though that due to the bulky nature of
20 fiber in foods, excessive consumption is likely to be
21 self-limiting. And since fiber intake tends to be
22 really low anyway, I don't think there was any concern

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1 of getting people too high, because most people are so
2 low to start with.

3 New carbohydrate information since the 2010
4 Dietary Guidelines. So that was the lecture. My kids
5 always say, you know, enough. Do it at work. Keep it
6 to yourself. So anyway, what's different? So I have
7 tried to pull together some papers I think that have
8 changed the -- that have been published since the 2005
9 Dietary Guidelines and have -- we should consider in
10 our deliberations. The general areas they are in are
11 sugar, especially fructose; glycemic index/glycemic
12 load -- I want to follow-up on that; dietary fiber,
13 whole grains; and also food form, liquid versus solid,
14 and I know we overlap with other committees on that.

15 Macronutrients And Obesity -- oh -- I actually
16 tried to do a review on this before. It's very
17 difficult obviously, but there is no clear evidence --
18 this is a review that was published in the European
19 Journal of Clinical Nutrition that I thought had some
20 good points; no clear evidence that altering the
21 proportion of total carbohydrate is an important
22 determinant of energy intake. And, if you get into

1 this literature -- there are lots of studies out there
2 -- but, you know -- like proteins versus carbohydrate,
3 if calories are controlled, obviously it is not very
4 clear that micromanaging your macronutrients does that
5 much for weight loss. There is evidence that sugar-
6 sweetened beverages do not induce satiety, and I want
7 to talk a little more about that. There is a lot of
8 controversy there, but quite a few studies in that
9 area. Findings from studies on glycemic index on body
10 weight have been inconsistent. So I think since the
11 2005 Dietary Guidelines that data has definitely not
12 been very positive that that is the way to go, and
13 dietary fiber intake is consistently linked to less
14 weight gain, but you know, we said lots of dietary
15 fiber diets have other positives besides just the
16 fiber.

17 I had to use this slide, because it was published
18 in our journal, and how bad is fructose? It's like
19 when do you stop, you know, when did you stop beating
20 your wife? It's the same kind of thing that, you know,
21 we are assuming it's bad and trying to sort this out of
22 all the different carbohydrates, a couple of other

1 disagreeing viewpoints, so I would suggest you read
2 that one by Dr. Brave. And also Dr. Anderson, in a
3 similar version of the journal said, there is no
4 evidence that the ratio of fructose and glucose
5 consumed from sugars has changed over the past four
6 decades as a result of high fructose corn syrup
7 replacing sucrose in many applications. So trying to
8 get at this data, and I think it would be good if we
9 have some people come in and speak to our committee on
10 this, is that high fructose corn sweetener is pretty
11 much the same as sucrose, so if you are just doing a
12 switchover there aren't big changes in your exposure of
13 fructose and glucose. So, high fructose corn
14 sweeteners does not appear to contribute to overweight
15 and obesity any differently from other energy sources
16 in this review and critical reviews in Food, Science
17 and Nutrition that calories are calories and high
18 fructose corn sweeteners are no different than other
19 calories, calories per calorie, so that was their
20 review.

21 This is another -- sugar-sweetened beverages and
22 body mass index in children and adolescents -- a meta

1 analysis that was recently published, they looked at
2 all the trials they could find. There were 12 trials
3 that were in this reference; 10 were longitudinal and
4 two were randomized trials, of sugar-sweetened
5 beverages and weight gain in children and adolescents.
6 Quantitative meta analysis and qualitative review found
7 that the association between sugar-sweetened beverages
8 and body mass index was near zero. You know, there is
9 not a lot of studies there, and remember too that the
10 data on this, you know, if you think how can people
11 come up with such a difference, calories do count, but
12 in this the sugar-sweetened beverages were not, you
13 know, linked to body mass index.

14 Obesity Review's recent article -- they reviewed
15 associations between intake of calorically-sweetened
16 beverages and obesity relative to adjustment for energy
17 intake. And this is difficult, to try to adjust for
18 energy intake and put that in perspective. They found
19 there were 14 prospective and five experimental studies
20 that were reviewed in their paper. They felt like a
21 high intake of calorically-sweetened beverages can be
22 regarded as a determinant for obesity, but this is --

1 if you read this, this is kind of confusing -- no
2 support that the association between the intake of
3 calorically-sweetened beverages and obesity is mediated
4 via increased energy intake. They suggest there are
5 alternative biological mechanisms. So you can see
6 within the literature there is a lot of disagreement,
7 you know, basically looking at the same data.

8 This is a research editorial also published in my
9 journal, so how discretionary can we be with sweetened
10 beverages for children, and this is a quote directly
11 from it. Based on cumulative evidence, it is
12 recommended that children consume no more than one
13 sweetened beverage per week. There is little room, if
14 any, in the diets of children to replace healthy foods
15 with the empty calories from liquid sugar. So I don't
16 know if -- what exactly -- is that chocolate milk? I
17 mean, what's milk, besides milk has lactose, so is that
18 sweetened or is it only if I put other sugar into it?
19 So, I think there is a lot of -- this whole area of
20 sweetened beverages sugar intake is -- there are a lot
21 of things being published. There is a lot of -- people
22 feel very passionate about it, but for us to step back

1 and take a research-based view of it, I think is going
2 to take quite a bit of effort in looking and reviewing
3 what is out there.

4 I wanted to put this in just because I think --
5 the way I always think of diets is that protein is very
6 important. We never want to lose sight of that. And
7 since we don't have a protein committee, I'm going to
8 be the protein person too. So, I want to point out
9 that diets -- protein is the most important thing when
10 we put a diet together, so we definitely want to make
11 sure we talk about that.

12 On low-calorie diets higher protein intakes are
13 recommended, so as calories go down protein goes up.
14 So, in saying, is it carbohydrate versus protein on
15 low-calorie diets, which we are going to recommend,
16 protein has to become more important. It's going to be
17 a higher percentage of the K-cals that somebody can
18 consume. There are probably some advantages over
19 carbohydrates in satiety, you know, depending on what
20 types of carbohydrates are chosen, increase of
21 thermogenesis, maintenance of fat-free mass. So this
22 review I think did a good job of summarizing that there

1 are definite advantages to high protein diets that we
2 don't want to lose sight of and that, in weight loss,
3 we want to make sure we are not breaking down body
4 protein, because that's going to help people burn
5 calories.

6 This is a little bit on glycemic index. I wanted
7 to start with sugars, and I guess you already know that
8 it's pretty controversial on sugars, but I don't think
9 we have data that suggest that any one sugar is the bad
10 guy, and that if we just get rid of one sugar our lives
11 will be better; that it's calories and needing to give
12 carbohydrates a better way of looking at carbohydrates,
13 but not just picking on one.

14 Glycemic response in health, a systematic review
15 is meta analysis recently published, among glycemic
16 index studies the observed reductions in glycemic load
17 are most often not solely due to substitution of high
18 for low glycemic carbohydrate foods. The big thing is
19 available carbohydrate obviously, if you don't control
20 for that. And also in this review they talk about
21 fiber and unavailable carbohydrate, that that's an
22 important overlay to all of the glycemic index because

1 we know that typically fibers do lower it, but total
2 calories, total carbohydrate the important thing.

3 This was a recent thing looking at glycemic
4 index/glycemic load, and this was in the Women's Health
5 Initiative and what they, over about eight years of
6 follow-up, there were 1,476 incidental cases of
7 colorectal cancer, and they looked at total
8 carbohydrate, glycemic index, glycemic load, intake of
9 sugars, fiber and there was no association at all.
10 They concluded there was no -- results do not support
11 that diet characterized by high glycemic index or load
12 plays a role. And this was in post-menopausal women.

13 Another review just published in Nutrition
14 Reviews, a very extensive review on glycemic index and
15 glycemic load and dietary recommendations. They looked
16 at the epidemiological data and glycemic index,
17 glycemic load and all the relationships, the diseases,
18 heart disease, insulin sensitivity, Type 2 diabetes,
19 dyslipidemia and obesity, with initially healthy people
20 that were followed, very mixed results. The only
21 positive association they found between glycemic index
22 was with the development of Type 2 diabetes, that that

1 was consistent, but otherwise the data was not
2 consistent, and their take home message is it seems
3 premature to include glycemic index or glycemic load in
4 dietary recommendations.

5 I want to just finish on fiber and satiety. So
6 this is all the things that fiber does to affect
7 satiety, and up on the top there you can see hormonal
8 intrinsic and colonic effects. And this is what's so
9 complicated when we look at different types of
10 carbohydrates with or without fiber is they have
11 effects throughout the digestive tract that could help
12 us in making people feel better eating less, kind of I
13 think where we are going to end up here. The left, the
14 hormonal effects; the middle, the intrinsic effects,
15 just the chewing; and then on the right, the colonic
16 effects, and I think we have ignored the colonic
17 effects. Nobody really likes to collect poop, that's
18 what I say, so in nutrition that, you know, like it's a
19 black box and we stay away from it as much as possible,
20 but what's going on down there is actually pretty
21 important.

22 Cereal grains of weight management; whole grains

1 associated with lower body mass index, waist
2 circumference, risk of being overweight -- so there is
3 pretty consistent fiber and whole grains preventing
4 weight gain, helping with weight loss. This was from
5 the Women's Health trial, post-menopausal women at 12-
6 month intervention, and this is this mixed diet idea
7 that it's not one particular thing. They are on a low-
8 fat diet and that the dietary correlates, everything
9 comes along for the ride. So, it's low-fat, it's high
10 fiber from a mixture of whole grains, fruits and
11 vegetables; a higher fiber intake; lower body weight;
12 and there were some improvements in biomarkers, so.

13 This was a study done -- and this is difficult,
14 because a lot of studies with whole foods are not real
15 successful. This one was done at Penn State, Penny
16 Kris-Etherton. They looked at obese adults and gave
17 them a whole grain diet of refined grain. One thing,
18 and it's really right down there on the bottom, both
19 diets improved CVD risk factors, and this typically
20 happens on even refined -- you know, people think,
21 okay, what's the difference between whole grains and
22 refined grains -- both groups when you put them on

1 these controlled diets do better. So, the whole grain
2 diet did a little better in C-reactive protein and a
3 little bit of difference in body fat, but overall they
4 were both showing improvements.

5 And this is another study that was published since
6 2005 on whole grains, you know, where they actually
7 gave people whole grains or refined grains for six
8 weeks and saw virtually no changes at all. And, you
9 know, these diets are not that different. You know,
10 whole grains are important, you get more fiber. But,
11 in these types of studies, you see improvements just
12 when you put people on these refined grain diets that
13 are typically better than the diets they are on.

14 Okay. Satiety -- I want to just finish up on that
15 -- what makes people stop eating and feel that they are
16 full, and it's a self report. Usually you ask people a
17 combination of questions, hunger, satisfaction,
18 fullness, desire to eat? I already talked a little bit
19 about this, but these things can happen; fiber effects
20 throughout the digestive tract. So where it's having
21 its effect we don't know, but throughout the digestive
22 tract we see differences. And it's usually -- this is

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1 what it is; it's a visual analog self-report and you
2 compare different treatments. Because, you know, I
3 always get this question, well aren't people always
4 hungry or always full? Yeah, you know, there are
5 differences among people, but if you use the same
6 person in these trials and they come in fasted, they
7 are given the treatment, it's how to -- what's the
8 acute effect of that treatment. I go back really --
9 even though I know I am supposed to stay within 2005, I
10 really like old stuff. I am more of a history buff
11 than anything, and this is a poorly, you know 10
12 subjects, but I think it's kind of a neat little study
13 where they compared apples, apple puree and apple juice
14 and they found that -- you know and it was 60 grams of
15 available carbohydrate, the juice could be consumed 11
16 times faster than the intact apples and four times
17 faster than the puree. So just kind of, you know, what
18 makes people slow down and not eat so much, you know,
19 fiber and whole foods. And when they actually
20 controlled the rate of ingestion so that it was all
21 equalized, the juice was less satisfying than the puree
22 and that was, you know, less than the apples.

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1 There is a more recent study in carrots that kind
2 of gets at this physical structure of real food, and in
3 this they were given either carrots at 200 grams or
4 whole carrots, blended carrots or carrot nutrients. So
5 carrot nutrient is like a carrot cocktail. You figure
6 out all the nutrients that are in it, throw it in a
7 drink and it's a carrot cocktail. What they found,
8 whole carrots and blended carrots resulted in
9 significantly higher satiety, and when you look at food
10 intake throughout the rest of the day, the carrot
11 nutrients didn't really affect it at all, so it was the
12 fiber content and the structure of the food that were
13 important.

14 This is another recent one on whole grains that I
15 wanted to bring in, and this is another thing where
16 they controlled different types of breads. So it was
17 white bread, whole meal, wheat bread and then whole
18 kernel bread, which is like bread with chunks in it,
19 and what they found is the whole kernel bread resulted
20 in significantly higher satiety than the whole meal,
21 wheat bread or white bread, so actually having some
22 structure into the food. And they saw no differences

1 in blood glucose, so it wasn't related to the blood
2 glucose response.

3 I'm going to end up a little bit on viscosity just
4 because we know that these viscous fibers do help
5 enhance satiety, so that kind of gives some structure
6 within the digestive tract. If you take a thing like
7 guar gum that's very viscous and you hydrolyze it, it's
8 not as satiating, even though the fiber content doesn't
9 change. So the fiber is the same, but when you modify
10 it, it doesn't have the same effect on satiety. And,
11 if we look at gastric emptying, that's not the whole
12 explanation, because there are studies where they have
13 control for that and it doesn't explain it all.

14 A little bit on protein and fiber -- if you look
15 at satiety besides fiber, there is quite a bit of data
16 on protein, and this lupin-enriched bread that was
17 done, it's higher in both fiber and protein, so we
18 don't know which it is that's having the effect, but
19 this lupin bread it was higher satiety and after -- you
20 know, you look at energy intake at lunch, they ate less
21 after they had the lupin bread, and there were some
22 changes in gut hormones also with grellin (ph) in that

1 lupin.

2 Does dose matter? Absolutely. You know, little
3 bits of fiber really don't make a difference. Here is
4 4.5 guar, which is a very effective fiber in a
5 breakfast bar, but no differences. Usually it's high
6 doses. When we look at the DRI for fiber and we think,
7 wow, 38 grams is a lot of fiber, but to really
8 increase fullness you see higher doses over the day
9 being more successful than lower doses.

10 And these are the ones that have been shown. We
11 did a review trying to look at all the fibers. The
12 viscous fibers for sure worked. Wheat bran definitely
13 works. The pea fiber, which is more of an insoluble
14 fiber; cellulose, soy polysaccharide have some data,
15 but it's generally in higher doses.

16 I'm going to finish up on fiber intake in the U.S.
17 is low. It's about 15 grams per day; recommended
18 intakes, 25 to 35. Most fiber-containing foods are
19 pretty low; one to three grams of fiber. USDA data
20 shows that white flour and white potatoes provide the
21 most fiber in the U.S. diet, not because they are
22 concentrated fiber sources, but because they are widely

1 consumed. So we haven't made a lot of progress in
2 getting people to eat high fiber foods. So any push
3 towards that -- and because of that there is interest
4 in the addition of functional fibers; if we can't
5 change people's food intake, let's change their food.
6 So -- and this is all reviewed in this Health
7 Implication of Dietary Fibers paper.

8 A little bit on just -- I want to finish up on
9 satiety -- just I stuck these in here since I am
10 talking about satiety. In this research they compared
11 beverages with sucrose or high fructose corn sweeteners
12 on hunger, satiety and energy intake; no differences
13 between sucrose and high fructose corn sweeteners,
14 which really, if you think about it, how, why would you
15 have? I mean, it's chemically the same, so not too
16 surprising. Diet cola and no beverage -- if they
17 weren't given calories they ate more at lunch, which
18 also seems pretty obvious, but you know, the calorie
19 dose they got, whether it was from corn sweeteners or
20 sucrose, no differences, but they ate less at lunch
21 with that.

22 And this was kind of interesting that was just

1 published, because they compared high fructose corn
2 sweeteners, sucrose and milk pre-loads. In this study
3 they added a milk -- and these are isoenergetic drinks,
4 so they controlled calories and they saw no differences
5 on changes for gut hormones between these different
6 drinks. So, you know, looking at all these differences
7 there were no differences at all in satiety and gut
8 hormones.

9 So, just to finish up, high carbohydrate diets are
10 recommended by the DRIs. I don't -- I think that's --
11 we really need to support that. It makes sense overall
12 to be up in that range. Dietary fiber intakes are less
13 than half of recommended levels, so getting progress on
14 that is important. Some of the definitions from
15 before, from the 2005, I definitely want to include the
16 legumes. They didn't get listed, but I think they need
17 to be up there as a good fiber source. And just
18 choosing carbohydrates wisely; getting people to take
19 the higher fiber ones. And I think that there is every
20 reason to push towards whole grains as a grain category
21 just because there is more fiber, there are more
22 nutrients in that category. Measures of carbohydrate

1 quality remain elusive, so trying to give carbohydrates
2 grades is really tough and people feel strongly about
3 it, but I don't think glycemic index or glycemic load
4 are going to help us categorize. Thank you. And I
5 have other people on my committee, other comments, I
6 welcome.

7 DR. VAN HORN: Great job. That was absolutely
8 wonderful. Other people on the committee that either
9 were on the subcommittee. Rafael?

10 DR. PEREZ-ESCAMILLA: Joanne, you presented data
11 regarding the consumption of sweetened beverages by
12 children and relationship or lack of a relationship
13 with body mass index, but what about insulin resistance
14 Type 2 diabetes in childhood; have you examined that
15 data?

16 DR. SLAVIN: Yeah. Go ahead. Yeah, the question
17 had to do with is there data in children with
18 resistance and consumption of sweetened beverages. I
19 think it's probably in those reviews if there was any
20 data out there, but I don't have it off the top of my
21 head.

22 DR. PI-SUNYER: There is certainly no longitudinal

1 data. There is some association data across the
2 spectrum that is determinately good. So I think the
3 evidence is pretty weak mostly because there is --
4 there haven't been enough studies that have been either
5 interventional or observational among children.

6 DR. VAN HORN: Certainly the diet data suggests
7 that children are equally poor in meeting the, you
8 know, fiber goals as the adults, and they too don't eat
9 enough of it.

10 DR. PI-SUNYER: I wanted to comment -- this is
11 Xavier -- I wanted to comment on Joanne's mention about
12 the importance of protein, but you know, I think the
13 mix-up of the message to tell people, you know, lower
14 your calories, but take more protein, I'm not sure is a
15 reasonable message. First of all, Americans are eating
16 more protein by far than they need, on the whole. And
17 I think if you give an across-the-board recommendation,
18 it's usually easier than trying to specify specific
19 groups or macronutrients. So, I'm not sure that we
20 would be doing anybody a disfavor if we said, you know,
21 across the board cut back your portion sizes if you
22 have an appropriate proportion of fat, carbohydrate and

1 protein and not try to push protein a little bit,
2 particularly since with protein very often comes fat
3 and saturated fat.

4 DR. SLAVIN: I just -- I wanted to comment on
5 Christine's with the milk consumption though, because I
6 think the foods that we choose are really a problem,
7 and if kids were consuming milk, that's a protein
8 source. But, as you look at her data, as they have
9 switched out of that, protein needs in general, the
10 average is good, but there are people that just aren't
11 getting enough protein and a lot of it has to do with
12 bad food choices. So, I think, as the Dietary
13 Guidelines group, we have to keep that kind of front
14 and center; that as people cut calories, you know, we
15 don't really want them -- and that's kind of why I like
16 the discretionary calories idea that we want them to
17 cut calories that aren't essential nutrients, which
18 protein is.

19 DR. VAN HORN: One thing that I am aware of that
20 perhaps we need to bring the data up to speed since the
21 2005 Guidelines is the importance of plant-based
22 protein, and the data -- certainly the data that I am

1 aware of, both with Cardia and Eric, I think, and
2 Intermap definitely illustrating that higher -- that
3 plant-based protein is interestingly associated with
4 both blood pressure, cardiovascular disease and
5 obesity. So, you know, it would appear that the
6 message of more plant-based foods transcends not only
7 the message of, you know, improving carbohydrate
8 intake, et cetera, but also by preferentially
9 increasing protein from plant-based sources, there is a
10 benefit as well. Yes.

11 DR. NELSON: I agree with that. Thank you so
12 much. This is Mim Nelson. So, I think that somehow we
13 do -- I guess I'm trying to put a point on some stuff
14 you said, but we have to figure out a different way to
15 frame carbohydrates, because I think that this message
16 that, you know, we need to have a diet that's rich in
17 carbohydrates hasn't worked. And, it's because when
18 you think about it, I mean, the carbohydrates we are
19 trying to get them to eat; fruits and vegetables, low
20 and, you know, non-fat dairy, whole grains, legumes,
21 you know, all the sort of more nutrient-dense
22 carbohydrates, we obviously aren't getting there. And

1 it's the abundance of high-calorie caloric -- whether
2 it's sugar; whether it's just refined grains; whether
3 it's, you know, snack foods, everything else, all of
4 these other carbohydrates that really would go into --
5 you know, white bread, white pasta, you name it, sort
6 of that goes into the discretionary carbohydrates. If
7 we do just talk about carbohydrates, in a sense, there
8 is -- the balance is just so off from what the American
9 population is eating that I think we do have to be
10 really careful about how we present them or look at the
11 evidence, and you know, really around those food groups
12 that are so important for health. And the other, you
13 know, just look at the grocery store and the way our
14 food supply has changed. It's really the preponderances
15 in these snack foods and refined everything else and
16 that's -- I think it's a real problem in energy intake.
17 We just eat a lot more of those foods and not enough of
18 the good ones. So I think we have to be very careful
19 on how we frame carbohydrates, as opposed to just
20 carbohydrates, it's more these food groups and not
21 these.

22 DR. SLAVIN: Yeah. And we were talking on the way

1 over just about complex carbohydrates, you know, like
2 how do you make the point? And glycemic index is good
3 in some ways, because it does -- you know, it doesn't
4 solve the problem, but it does tend to get you more
5 there. But I agree that, you know, with -- and that's
6 why I guess I put in this idea about proteins is that a
7 lot of times we want people to reduce calories, but
8 really not across the board; that as you reduce
9 calories, protein -- the percentage has to go up. So
10 -- especially for kids. I just think -- and you know,
11 I think that the vegetable protein data is interesting,
12 but protein quality, you know, if you are going to
13 limit protein and limit calories, then protein quality
14 becomes a more important data point that we have to
15 consider, especially for children, for kids, pregnancy.

16 DR. NELSON: But I would say equally as important
17 is carbohydrate quality.

18 DR. SLAVIN: Yes, yes, for sure.

19 DR. VAN HORN: Cheryl.

20 DR. ACHTERBERG: Thank you. Cheryl Achterberg. I
21 wanted to reinforce what Mim said, and I think maybe we
22 should be brave enough to consider looking at starchy

1 food components and consider putting potatoes and bread
2 together, at least look at that as an option, since
3 most of the world does already.

4 Two questions really more than anything else. I
5 know, Joanne, you presented a very very comprehensive
6 review, but I also know that in Europe they seem to be
7 a little less squeamish about doing gut health-type
8 studies and that I think the Committee needs to take a
9 good look at what some of those European studies are
10 saying about fiber. I just think they have a lot more
11 data that's come out recently, but I am not sure that
12 Americans are that aware of it yet.

13 I also want to ask the question, if you are aware
14 of any studies so far -- I noticed on one slide you
15 mentioned resistant starch -- whether any studies have
16 been completed yet that look at resistant starch in the
17 context of a whole diet, as opposed to a meal or a
18 food, and what we might make out of that?

19 DR. SLAVIN: Well, you know, I think that gets
20 into this resistant -- that starch complex
21 carbohydrate, you know, what's good about complex
22 carbohydrate that it's got resistant starch in it. So

1 there are some data on intakes. Most of the biological
2 data is short-term, but you know, if you look at people
3 that have high resistant starch diets, there are
4 people, kind of like the 1970s fiber hypothesis;
5 unrefined plant-based foods are going to be high in
6 fiber and resistant starch. There are method issues
7 that have limited just databases on, you know, intakes
8 of resistant starch, but it definitely goes along with
9 high fiber and more plant-based diets.

10 DR. ACHTERBERG: And there are some people trying
11 to tie it to perhaps preventing Type 2 diabetes?

12 DR. SLAVIN: Yeah. You know, I think that the
13 whole -- I think with all the information we have
14 that's a number really important with our group to
15 consider. The carbohydrate message, trying to separate
16 it from over -- you know, because we are most of the
17 calories that people eat, so within -- if we limit
18 calories and control calories, which carbohydrates
19 would be the most protective, and it, you know,
20 obviously fiber for sure and resistant starch, which
21 isn't going to affect glycemic response. I mean, if
22 it's a truly resistant starch it goes straight through

1 and it doesn't change insulin; it doesn't change blood
2 glucose, so it would be a positive for sure.

3 DR. VAN HORN: Roger?

4 DR. CLEMENS: Joanne -- Rog -- thank you very
5 much for that excellent presentation. I appreciate the
6 comments that, in fact, not all carbohydrates -- some
7 carbohydrates function as preservatives. They actually
8 lower the AW and so prevent microbial growth. And I
9 appreciate your comments about the betaglucans and the
10 structural differences there. As we all know, not all
11 betaglucans are the same. Some are immunomodulatory
12 from different sources; others may affect dyslipidemia,
13 as we are all concerned about.

14 Also, some amino saccharides have been
15 demonstrated to inhibit dental caries and actually
16 inhibit the adhesion of streptococci through the
17 dentition, something we might want to consider or at
18 least mention in our comments.

19 And lastly, I would like to comment, as you
20 pointed out so nicely about the short-chained fatty
21 acids, and as we know, the micro flora in the G.I.
22 tract has a tremendous impact on gut health and overall

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1 health, and that too may be a common area our group
2 wishes to address then as we look at more and like
3 impact of whole grains in affecting our microbial
4 health in the G.I. tract.

5 DR. SLAVIN: I appreciate that. Thank you.

6 DR. VAN HORN: Chris?

7 DR. WILLIAMS: Christine Williams. I wanted to
8 comment on dietary fiber intake in children, which of
9 course is very low compared with the National Academy
10 of Science Guidelines of 14 grams per 1,000 calories.
11 On the other hand, there isn't a whole lot of
12 scientific evidence that those guidelines are
13 appropriate, especially for young children, which might
14 be one thing we might want to look at. Dietary fiber
15 certainly has the same health benefits in children as
16 in adults and we need to encourage higher levels.

17 DR. SLAVIN: Yeah. I appreciate that, Christine,
18 because the -- you know, since those recommendations
19 were made on 14 grams per thousand K-cals, it was just
20 a math, so you know, it was never really thought about
21 for kids. So the ones for little kids are way too
22 high, and I think the old recommendations of age plus

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1 five actually make a lot more sense than the ones based
2 on K-cals.

3 DR. NELSON: Right.

4 DR. VAN HORN: Larry?

5 DR. APPEL: Yeah -- Larry Appel. I was just
6 curious about this review in which you said that the
7 only consistent association of G.I. was with incident
8 Type 2 diabetes, which you know, I had thought was -- I
9 thought that it was pretty inconsistent results. So, I
10 mean, is this independent of fiber volume mass index?
11 I mean, is this something we are going to act upon?
12 Because I was -- I mean, obviously Type 2 diabetes is
13 important. It's on the front page of the newspaper
14 today.

15 DR. SLAVIN: Yes. No. I saw that, and I -- you
16 know, I think we need to consider whether, like in that
17 review, the fiber and other things were taken out.
18 Because I think, you know, glycemic index, if it can
19 help us we should absolutely use it. But I think
20 overall, especially on weight control, that it -- you
21 know, because the problem you have in an intervention
22 study, which people do, is the easiest way to get GI

1 down is to put fructose in the diet. So that's, you
2 know, a lot of the intervention studies that are done
3 are really not consistent with the epidemiology, so.
4 But, you know -- yes?

5 DR. NELSON: Just a caution about fiber, and I'm
6 not a fiber expert, but I -- you know, one can't help
7 but look at what's happening in the food supply. You
8 know fiber is now being added to, you know, artificial
9 sweeteners, and it's being added to everything, and
10 while I think that the data is so strong around fiber
11 and health that I want to make sure that whatever we
12 consider that we are thinking more of it as part of our
13 food supply as opposed to as an isolated additive to
14 the food.

15 DR. SLAVIN: And, you know, I think we can use the
16 DRI Committee for that too; that they are saying that
17 not all fiber is the same; the dietary fiber in foods,
18 push that, push that, there is no real disagreement on
19 that. The functional fibers, unless they are shown to
20 do something, we shouldn't just accept them as good;
21 that they actually need to have a physiology effect
22 before we green light them.

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1 DR. VAN HORN: One more question and then we'll
2 take a break.

3 DR. RIMM: Go back to the -- this is Eric Rimm --
4 go back to the issue that Larry brought up about GI and
5 diabetes. I think one of the issues with glycemic
6 index and one of the challenges of using it
7 epidemiologically or otherwise is that it's probably
8 much more important to people who are insulin-
9 resistant. So, if you look at the glycemic index of
10 somebody who doesn't exercise or has a BMI of 29, it's
11 probably a much more, a much stronger predictor of
12 diabetes and other outcomes than it is among someone
13 who is a marathon runner or does strength training, or
14 has a BMI of 22. So, it really is sort of an effect
15 modification by where you are at; and given that 60
16 percent of the population is now overweight it is maybe
17 important enough at the population level.

18 DR. VAN HORN: All right. Well that was
19 absolutely wonderful. Thank you very much, Joanne, and
20 everyone for your input. We are scheduled to take a
21 break. We had a little one before, so let's keep this
22 to about ten minutes, and then we'll start back on

1 fatty acids. Thank you.

2 (Whereupon, at 10:53 a.m., a brief recess is
3 taken).

4 DR. VAN HORN: All right. Thank you all. We are
5 going to get started now with Dr. Pearson leading a
6 discussion about fatty acids, along with Drs. Rimm and
7 Clemens.

8 DR. PEARSON: Thank you, and on behalf of my
9 subcommittee, Eric Rimm and Roger Clemens, we are going
10 to have a little different --

11 DR. VAN HORN: They can't hear in the audience.

12 DR. PEARSON: Okay. Thank you. On behalf of our
13 fat subcommittee, which is Roger Clemens and Eric Rimm,
14 we are going to have a little bit of a different format
15 to where they facilitate a discussion. I am going to
16 stand up here so I have my pointer. We could go
17 through this quite large and complex issue of fats.

18 But just to start out with maybe a little bit of a
19 comment, and that is, a year or so ago -- actually, for
20 the two years before that -- we had a project in which
21 we sought out a low incidence coronary population to
22 really examine how one could in fact prevent the onset

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1 of a cardiovascular epidemic and ended up with a
2 population-based study on the country of Grenada, and
3 basically spent three years looking for a heart attack.
4 We failed. We talked to the physicians. We talked to
5 the nurses. We did a 2,100 person survey, et cetera.
6 We didn't talk to the cardiologist, because there was
7 none in the country. There were no cardiac surgeons.
8 This is a country without a cardiovascular epidemic.
9 And so, really the question one leads to as hypothesis
10 is that there is something that has caused that. This
11 is a country with a reasonable amount of obesity,
12 certainly a lot of diabetes, a lot of hypertension from
13 their Afro-Caribbean genetic basis perhaps, but no
14 coronary disease. So, the hypothesis you are left with
15 says the dietary consumption of saturated fat and
16 cholesterol is really the key determinant for raising
17 serum LDL cholesterol levels about the threshold
18 necessary to induce atherosclerosis on a population
19 basis, and this, for the U.S. Dietary Guidelines, leads
20 to the question of what should that threshold be and
21 what are we going to do nutritionally to get to that
22 point, where our leading causes of death, which is

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1 atherosclerosis, coronary disease and stroke are able
2 to, on a population basis, be controlled? So what you
3 are left with here then are many examples from a
4 population basis, in which you have excess caloric
5 intake, decreased caloric expenditure, high sodium
6 intake, perhaps heavy alcohol, with hypertension and
7 diabetes, also populations with a lot of smoking, but
8 in fact, if there is not the saturated fat and
9 cholesterol getting the LDL above a certain threshold,
10 you do not proceed into an atherosclerosis situation.
11 And this is not just the country of Grenada, but of
12 course, the countries in East Asia, Africa, et cetera,
13 in which you have very high levels of hypertension in
14 smoking, et cetera, but unless you have an LDL
15 cholesterol of a certain level, you are really not
16 developing a coronary epidemic.

17 So I think the real question is, is that can we
18 develop and look at in somewhat of a controversial
19 issue the dietary patterns in which to lower the
20 cholesterol, the total cholesterol in the United
21 States, to about the levels of Japan, et cetera, 160
22 milligrams per deciliter, so -- through the Dietary

1 Guidelines of saturated fat and try to get below that
2 threshold, where we can start to see big changes in the
3 levels of atherosclerotic cardiovascular disease.

4 So what we are going to do with our facilitative
5 discussion here is talk about dietary lipids; and you
6 can see that this is the splitter's view of dietary
7 fats, similar to what Joanne showed with fiber, which
8 is just a huge number of compounds; try to go through
9 this in a systematic way in identifying the various
10 components we might want to work with. Now, just one
11 point is that there are a couple of instances with the
12 whole area of fats, in which this has gone into the
13 pharmacologic realm. This particularly deals with
14 plant sterols and stanols and omega-3 fatty acids,
15 which are oftentimes given in pharmacologic doses, and
16 we are really not going to talk about those. This is
17 really a -- it's really almost a pharmacologic issue.

18 So let's maybe look at this relatively complex
19 view of dietary fats and maybe focus over here on the
20 sterols, and just make a comment on the plant sterols.
21 There has been some discussion on sterols and stanols,
22 particularly in pharmacologic levels that there would

1 be the prevalence of some of the -- of the
2 polymorphisms, which allow increased absorption of
3 plant stanols and sterols, but I think this is really
4 too low of a prevalence really to be a public health
5 issue, and obviously we are all going to be emphasizing
6 whole foods, fruits and vegetables for that. So, I
7 think the plant stanols/sterols really is a
8 discussion probably limited to some further discussions
9 in relatively unusual genetic subgroups.

10 The animal sterols, of course -- the plant sterols
11 obviously are another issue, and the current
12 recommendations for dietary cholesterol of course is
13 300 milligrams a day, in general, for the population
14 and 200 milligrams a day, if you are interested in
15 lowering your LDL cholesterol, and I guess one of the
16 questions is should we look at that again given some of
17 these population issues relative to the level of
18 dietary cholesterol we have. This was reviewed in the
19 2005 a bit, but there may be, with low cholesterol,
20 dietary cholesterol diets, some opportunities to reduce
21 that further and maybe ask Roger and Eric if you have
22 any comments on the dietary cholesterol issue.

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1 DR. RIMM: Yeah. This is Eric Rimm. I guess the
2 biggest issue with all the dietary lipids, that the
3 biggest -- there we go -- this is Eric Rimm -- I think
4 the biggest issue with all of lipids, and maybe this
5 will come more into play with fatty acids and other
6 fats more than sterols, is what can we do differently;
7 what science has been done in the last five years that
8 we can really advance what's already been there? And
9 it may be that for some of the plant sterols that there
10 is a fair bit of evidence in the last five years that
11 we may want to change or may want to add some
12 additional information. For cholesterol, I'm not sure
13 if there has been a substantial amount of new
14 literature that's going to change the 300 milligram or
15 200 milligram recommendation, but you know, I guess
16 that will -- we'll have time over the next year to
17 really search that out, to see if it's worth trying to
18 make a difference, and maybe there will be differences
19 for kids versus adults, but I think there is a lot more
20 new evidence in the fatty acids than the other fats
21 area than there is for cholesterol.

22 DR. PEARSON: Roger?

1 DR. CLEMENS: Likewise that when we pick up --
2 Linda had mentioned earlier about plant protein,
3 obviously plant proteins don't carry cholesterols, so
4 that may be another implication for us to look at plant
5 sterols that have implications on fatty acid profiles,
6 as well as cholesterol.

7 DR. PEARSON: And just to point out, the
8 guidelines are, of course, is that the 300 and 200 are
9 maximum amounts, and there certainly, I don't think, is
10 any evidence for there being any basement effects.
11 Unlike some of the fatty acids, there really isn't a
12 lower danger point in dietary cholesterol.

13 I think there are some other issues with other
14 sterols that are going to be covered I think in the
15 vitamin D and then with other fats in foods, the
16 retinols and some other fat-soluble vitamins, but I
17 think those will be covered by other parts of the
18 working groups.

19 I think where the issues come up a little bit more
20 has to do with the fatty acids. The current
21 recommendation is 20 to 35 percent of calories from
22 fats. Below 20 you do get into some of the issues of

1 essential fatty acid deficiencies and some clinical
2 syndromes related to that above; also have the
3 opportunity to have really excess carbohydrates, as Dr.
4 Pi-Sunyer had mentioned, a high carbohydrate diet is if
5 you don't have the calories from fat. At the upper
6 end, you get into the risk of very high fat diets of
7 hypertriglyceridemia, et cetera. But obviously this is
8 a situation in which the type of fatty acids have been
9 particularly important in that it may not be that the,
10 just the total fats -- percent of calories is the
11 issue, but rather, their make-up between saturated,
12 monounsaturated and polyunsaturated, and wanted to talk
13 a little bit about the saturated fatty acids.

14 We have talked about the short chain fatty acids a
15 little bit already. We talked about it relative to
16 colonocyte health and colonocyte differentiations, et
17 cetera. There is also some evidence that short chain
18 fatty acids may modulate into HMG CoA-reductase
19 activity at the liver level, so there may be a variety
20 of issues, but I think these are largely carbohydrate-
21 oriented rather than dietary fat-oriented.

22 We have the cholesterologenic fatty acids, and with

1 these -- with the C-12 or C-16, obviously there is a
2 large literature on them being cholesterogenic, and I
3 would imagine that there is not a lot more that we need
4 to say about those relative to their abilities to raise
5 LDL. As pointed out in the 2005 Guidelines, stearic
6 acid, C-18, is an interesting fatty acid, which is
7 currently included with the saturated fats, but has
8 metabolically distinct activities and does not have the
9 LDL raising effect. There is one paper cited in terms
10 of a possibly pro-thrombotic effect, and perhaps we
11 should -- one recommendation may be to look at this
12 issue again relative to its inclusion with the
13 cholesterol-raising of fatty acids or thrombogenic
14 fatty acids, and talk about its inclusion as one of the
15 percent of fatty acids that we'd like to reduce or not
16 reduce relative to that percent of calories from
17 saturated fat. So I think there is an issue out there.

18 Any other comments from Eric or Roger on this
19 issue?

20 DR. RIMM: Yeah. Well, I think -- I think -- this
21 is Eric Rimm -- I think this is an important issue
22 where there will be a fair bit of new evidence to look

1 through. You know, it's funny that Joanne mentioned
2 that she feels like the poor relation, because people
3 get -- they just talk about calories with fat and
4 protein and sort everything else falls into her bucket
5 at the end even though that she has most of the
6 calories, but because of that we are almost
7 overstudied. People really have, you know, teased
8 apart every possible calorie that you can get from fat
9 and look at where it's from. So -- and I think a lot
10 of the 2005 technical report did focus on LDL. There
11 was some discussion of LDL and HDL, but I think we
12 can't forget that while LDL is quite predictive, if
13 specific saturated fatty acids also increase HDL that
14 that should be really a very important consideration,
15 because overall, predicting cardiovascular disease it's
16 much stronger if you have a ratio where you have some
17 combination of those markers, as opposed to just LDL.
18 So, looking across the board I think that will be the
19 case, not just in saturated fats, but to specific types
20 of mono fats and polys and their impact on LDL and HDL
21 is quite important.

22 DR. CLEMENS: The interest at that time as well,

1 we have seen -- this is Rog -- we also have seen a
2 number of papers out there relative to the
3 polymorphisms of synthesis and degradation of LDL and
4 the lipoprotein fractions, and those areas may be
5 considered as well.

6 DR. PEARSON: So the current recommendations for
7 saturated fat are less than 10 percent for a standard,
8 with less than seven percent in a situation of
9 therapeutic reduction in LDL. It would appear that
10 both in children and adults we are currently at around
11 12 percent, and so there is certainly some
12 implementation issues that we not need to, to get to
13 relative to reducing those.

14 Let's move over to the unsaturated fats, and in
15 the monosaturated fats, of course we have the cis
16 versus the trans. There was quite a bit of attention
17 given to the trans fatty acids in the 2005. I would
18 imagine that there has been some additional studies in
19 this regard. Obviously there is the synthetic versus
20 the natural trans fatty acids, which may be a nuance to
21 look into, but obviously the ability of trans fatty
22 acids to not only raise LDL but lower HDL has been an

1 issue. There has also been related some morbidity
2 mortality studies, and so the question really is, is
3 that, you know, have all the nails been put into the
4 coffin of trans fatty acids, or is there some
5 additional activity, research activities that we should
6 know about? The current recommendation is less than
7 one percent of calories from trans, and so maybe some
8 discussion about what further we should do in this
9 area. Eric?

10 DR. RIMM: Yeah. I think -- this is Eric Rimm --
11 I think we have a great opportunity to give a very
12 focused direct message on trans, because there still
13 are lots of -- I think there still is a bit of
14 confusion. There are only a few cities that have
15 banned it and, you know, some sort of discussion on a
16 local level of getting rid of partially hydrogenated
17 oils, and I think that there is more evidence now.
18 There are animal studies suggesting that giving, you
19 know, an animal isocaloric diet, but one that's higher
20 in trans over the course of four or five years led to
21 weight gain in monkeys. So, I think there is something
22 about trans and getting trans in the cell membranes

1 that does impact metabolically some very important
2 functions. And, if our focus is going to be on weight
3 gain, I think that we could give a very focused message
4 on trans so that people can look at the 2010 Dietary
5 Guidelines and say, look, this is an important message,
6 because I don't think the, at least at this point, I
7 haven't heard that the IOM is planning on reconvening
8 the macronutrient panel, so at this point, you know, I
9 think that our message may be that, you know, the more
10 focused message as opposed to the 2002, 2002 IOM
11 report.

12 DR. PEARSON: Roger, and then Larry.

13 DR. CLEMENS: I think some additional information,
14 if you look at the national current trends of fatty
15 acids that should be discussed at this time, and food
16 is global. We have to look at the implications that's
17 going around and what it will impact on the diet drink
18 intake here, in the United States.

19 DR. PEARSON: Larry?

20 DR. APPEL: Yeah. A view from the 2005
21 Guidelines. You know, in the Blue Report, I think when
22 we actually recommended one percent, but that got -- it

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1 didn't get translated into the other reports, and so if
2 there is additional evidence that supports this, then
3 it might actually lead to the downstream effects that
4 we want.

5 The second thing, and I wanted to just follow-up
6 on something that Eric said, and I think this is
7 relevant to the committee, is what are the surrogate
8 outcomes that we are going to use for decision-making?
9 And you, Tom, said LDL cholesterol. I think that we
10 didn't really have as formal a discussion in 2005 as we
11 should have about whether we would use HDL as a
12 surrogate outcome for decision-making, and I think that
13 also applies to triglycerides, and that we should
14 really make the decision upstream, and then, you know,
15 do the evidence downstream, but we have to make that
16 call.

17 DR. PEARSON: That's an excellent point. Mim?

18 DR. NELSON: This may be a little off point, but I
19 think it's important, because I think one question to
20 ask is, there is so much awareness about trans fats now
21 in the public, and we have several cities that are
22 banning it, but what's happening with the food supply

1 is so many manufacturers have just substituted palm
2 kernel oil for trans fat, which is basically just as
3 unhealthy, and there has been -- I mean, close -- I
4 mean, it's not terribly healthy, so -- but there has
5 also been a fairly large environmental impact to the
6 palm kernel oil growing, so I don't know. We talked a
7 little bit earlier about some environmental, you know,
8 issues, but this is one I think in which, you know,
9 there is fairly large ramifications for sustainability
10 and things like that.

11 DR. CLEMENS: At this time, I thank you for that
12 comment then, Mim. We have also seen a great deal in
13 the last five years changes in the kinds of fatty
14 acids, in addition to the palm kernel that you just
15 mentioned. We know a number of companies actually have
16 developed the 18-1 and related compounds so that they
17 are doing a lot of substitutions. As a result, we have
18 a seen a marked reduction in the trans acids
19 incorporated into different foods stuffs.

20 DR. PEARSON: Naomi?

21 DR. FUKAGAWA: Yes. Naomi Fukagawa. I also
22 wanted to bring up the point about trans fats though,

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1 that we must remember that natural products are also a
2 source of important trans fatty acids, and in some ways
3 we shouldn't send everything out, you know, don't -- to
4 say that it's all bad, because they also are an
5 important source, you know, from our dairy products
6 and --

7 DR. APPEL: In dairy, fish, poultry --

8 DR. FUKAGAWA: Yes, dairy, fish, poultry. Right.
9 Right. So we have to have that balance.

10 DR. PEARSON: Just to comment on Larry's point, in
11 terms of the end points, I think, and Eric had alluded
12 to this, is that the LDL cholesterol was quite a
13 primary focus of the 2005 Guidelines. There is a
14 movement to redo the, from NHLBI, to do, redo the
15 National Cholesterol Adult Treatment Panel Guidelines,
16 and so those would be probably coming out in a year or
17 two, possibly within the range here, but certainly the
18 coordination of these Dietary Guidelines to, and to
19 adequately support those would be a good thing to do so
20 that those would be consistent. So, I can't really
21 comment or even speculate about where HDL and
22 triglycerides were, but they certainly are going to be

1 there somewhere. So I think, Larry, your point is well
2 taken, even though the interpretation of some of those
3 other end points is probably a little more complicated,
4 at least the HDL is, in terms of what an HDL
5 cholesterol level means, but I think it's a good point
6 for discussion.

7 Let's go on to the cis monounsaturated fats, such
8 as the, those have been, particularly with the adult
9 treatment panel, three guidelines have been the winner
10 of the recommendation in terms of going up a little bit
11 with the total fat to, up to 35 percent, but not
12 obviously doing that with saturated fats, but rather
13 with monounsaturated fats, and I think there is
14 obviously a lot of metabolic data relative to the
15 protection of your HDL and triglycerides through the
16 increase of monos versus the increase in carbohydrates.
17 But I don't know of any particular advances other than
18 showing those in many, many feeding studies. Eric or
19 Roger, any comments on the monounsaturated fats?

20 DR. CLEMENS: I think the only interesting one
21 that came out just about a month or so ago, Tom,
22 dealing with some cis fatty acids relative to satiety,

1 and it might be interesting to discuss as a group.

2 DR. RIMM: Yeah. I think, Rog mentioned that, in
3 an effort to get rid of trans fatty acids, the industry
4 has changed and can't come up with new seed and new
5 ability to make vegetable oils that are high monos,
6 since a lot of that is cis. So I think that it's in
7 the food supply and there is a fair bit of literature
8 on it, so I think it is worth looking at the impact of
9 high monos, not only on HDL and LDL, but on clinical
10 end points. Now we are focusing mostly on coronary
11 heart disease because I think that's where the
12 literature is, but I think it would be worthwhile to
13 try to summarize that, because I think the literature
14 is getting stronger on the benefits of high mono diets.

15 DR. PEARSON: Let's move over to polyunsaturates
16 and maybe talk about omega-3 fatty acids first. That
17 literature I think has been in a couple of areas; one
18 on epidemiologic studies treating, relating to dietary
19 patterns with high fish consumption; and the other with
20 relatively pharmacologic doses of fatty acids in
21 randomized trials of clinical subgroups with some I
22 think very encouraging findings relative to efficacy;

1 things like heart failure recently published in the
2 Lancet and a variety of issues. I think these
3 pharmacologic trials also though provide some
4 opportunities for looking at safety and other issues,
5 other related things at higher levels that we would
6 probably see at the population level, so maybe in a
7 tangential way be informative. But, Roger, I know you
8 have had a lot of interest in this area. Any other
9 comments on the omega-3s?

10 DR. CLEMENS: I think part of the education --
11 this is Rog Clemens -- part of the education on omega-
12 3s may hinge as well on omega-9s and omega-6s, as well
13 as their relationship. It is interesting to see that
14 we might want to address enviro burden. I know that
15 the 2005 Guidelines addressed the interconversion. We
16 talk about from flax seed oil to alpha linolenic acid,
17 for example, all the way down the pathway that the
18 bioconversion in a number of population groups is
19 somewhat compromised, that could be quite an impact.
20 Then we go all the way down to omega-3s and the DHA at
21 the end of the metabolic pathway. We don't all get
22 there at the same time, and that may be a point of

1 discussion for our group as well. We certainly see
2 that while the focus of the 2005 Guidelines was on
3 cardiovascular disease and cardiovascular health, you
4 certainly see in the last five years a merging of data
5 that look beyond that particular area of health and one
6 of those areas, such as macular degeneration, as well
7 as dementia. So I think we want to broaden our focus,
8 as well as to broaden our particular end points that
9 Larry mentioned.

10 Most recently there was -- I think within the last
11 year there was a paper by Bill Landis, if I recall
12 correctly, to address the omega-6, omega-3 fatty acid
13 profile, as well as the ratio relative to the incidence
14 of cardiovascular disease and survivability. That may
15 be a point of conversation for our group as well.

16 DR. PEARSON: Eric?

17 DR. RIMM: Yeah, I agree. I think the evidence on
18 omega-3s from DHA and EPA has gotten even stronger. We
19 have done a meta analysis on that and I think that
20 there is no question the importance of that on reducing
21 overall mortality, as well as sudden death. You know,
22 the issue of the omega-6 to omega-3 ratio to me may be

1 a little more troubling just because I hate for people
2 to reduce the amount of omega-6 in their diets. I
3 think people who have sufficient amounts of omega-3s,
4 the ratio actually is not that important; that there is
5 sort of a hypothesis about the potential increases in
6 oxidation, but most of the data suggests that the best
7 inflammatory profile is when you have high omega-3 and
8 high omega-6. So, I think it would be worth reviewing
9 that data, especially the human data, because that's
10 where you see there is the greatest benefit and the
11 ratio is actually not that important. I think it can
12 become important if people have very, very low levels
13 of both EPA, DHA, as well as alpha linolenic, so I
14 think there is a great opportunity from new research in
15 the last five years.

16 DR. PEARSON: Indeed. Other questions on omega-3s
17 from the panel?

18 DR. APPEL: Yeah. Just a -- will your group
19 actually deal with fish, because I think that's
20 actually we -- we got in a vicious circle actually in
21 2005 trying to figure out, you know, fish
22 recommendations, so.

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1 DR. RIMM: We have two experts. We'll take on
2 fish.

3 DR. CLEMENS: Yeah. We'll definitely take on
4 fish.

5 DR. PEARSON: I've got a few more slides for you
6 too. The fish will swim in.

7 DR. VAN HORN: Well, and also, the issue related
8 to the confusion I think out there about plant-based
9 versus fish-based sources in omega-3 and that the, you
10 know, the total amount needed to, you know, be
11 comparable is extreme.

12 DR. CLEMENS: Yes, it is.

13 DR. VAN HORN: So, I don't think the public
14 understands that.

15 DR. PEARSON: Eric.

16 DR. RIMM: Eric Rimm -- I wanted to make a radical
17 point, one for which I'll probably get kicked off the
18 stage, but the whole issue of total fat and the 20 to
19 35 percent of calories from fat is one that has
20 troubled, I guess has troubled me, because partly
21 because I sat on that 2002 IOM Committee where we tried
22 to come up with a range for fat, and ultimately we

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1 decided there is not one point which is the healthiest
2 point, which is why we came up with the range concept.
3 But the high end, you know, why we set 35 percent of
4 calories from fat, actually was not really based on
5 much science; it's based on the fact that we don't have
6 a lot of -- at the time we didn't have a lot of science
7 beyond 35 percent, and there was a concern that higher
8 fat diets would lead to obesity. I think if you look
9 at the science, there is actually no good human data to
10 suggest that higher fat diets lead to obesity. If
11 anything, higher fat diets, at 35 to 40 percent, lead
12 to lower triglycerides because it's a lower
13 carbohydrate intake. So, I think we should -- I am not
14 saying that at this point we should just say everybody
15 eat as much fat as we want, but I think there is the
16 dogma and that low-fat diets are beneficial, and you
17 can go in the grocery store and see a lot of low-fat
18 foods that are essentially just put in with high
19 carbohydrate, highly processed sugars. So my concern
20 is that we, over the last 30 years, have created the
21 dogma that all fat is bad, and I think that that high
22 end of 35 percent of calories from fat is artificial.

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1 And, if you look at some new data that has come out
2 from dietary patterns among people in Greece or
3 European countries, in fact they don't have higher
4 rates of heart disease, yet they have healthy fats. So
5 I think we have identified, you know, on your right
6 side of your dietary lipids some fats which can be
7 beneficial and which we can -- industry has figured how
8 to put in our diets in the U.S. that, in fact, if we
9 have good fats there is no reason to think that we need
10 to necessarily have that high end set at 35 percent.
11 So, I hope all of us can at least look at the science
12 before we come up with that sort of artificial end
13 point.

14 DR. PEARSON: Thank you. Let's move on to the
15 omega-6 fatty acids. Obviously the recommendation is
16 less than 10 percent of calories from that, mostly on
17 the basis of some concerns about, at least relative to
18 monos, the lowering of HDL and some data relative to
19 increased tumor production in high omega-6 groups. Any
20 other comments from the panel on that, Eric or Roger?

21 DR. FUKAGAWA: What are your -- this is Naomi.

22 DR. PEARSON: Naomi.

1 DR. FUKAGAWA: -- your thoughts about the
2 fortification of infant formula? Is that something --

3 DR. CLEMENS: This is Rog. That's a group that
4 wasn't within our age review. I certainly could
5 address it, but it's not within our charge.

6 DR. APPEL: Unless they are still drinking formula
7 at age 2.1?

8 DR. CLEMENS: Yeah.

9 DR. FUKAGAWA: That's true.

10 DR. CLEMENS: And actually from a regulatory
11 standard, is it geared for people two years of age, so
12 it's a regulatory thing.

13 DR. SLAVIN: I think that -- this is Joanne Slavin
14 -- I think it's a question, you know like if breast
15 milk is 55 percent of calories come from fat --

16 DR. RIMM: It's from fat.

17 DR. SLAVIN: So you start out on a high fat diet,
18 and just, you know, for kids not getting our fat
19 restrictions as tight as sometimes they are that kids
20 really need to be on a high-fat diet, which kind of a
21 conflict with obesity. You are like, well we'd better
22 not do that, but --

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1 DR. RIMM: Yeah. I mean, I think that the problem
2 is there is not a lot of great data that kids who have
3 more fat are more overweight. I think the problem is
4 if they are put on pretzels, which have no fat versus
5 how they process carbohydrates, and that leads to
6 potentially to overeating. So, it would be a good area
7 to look at. I don't think there is a ton of data,
8 prospective data on fat in kids. I could be wrong.

9 DR. VAN HORN: Well, having just been involved
10 with an NHLBI pediatric panel, I think probably the
11 biggest contribution to the data within the last five
12 years has been the strip study recognizing that, you
13 know, taking children from birth and actually having
14 them on a lower, total fat lower saturated fat diet has
15 yielded no adverse conditions, and in fact improved
16 lipid levels in these children, who are now seven. So,
17 the prospective data are really quite impressive, and I
18 think could and probably should be reviewed and
19 included in this presentation or in this discussion.

20 DR. CLEMENS: I see you point, Linda. I think
21 there is some emerging data on cholesterol. We know
22 that breast milk is naturally high in cholesterol.

1 DR. VAN HORN: Right.

2 DR. CLEMENS: And what the impact is neurological
3 development as well.

4 DR. WILLIAMS: Chris Williams. Actually, from
5 that strip study now they are showing lower rates of
6 obesity in the children, so.

7 DR. VAN HORN: Yeah, right, exactly, both, both
8 obesity and lipids. Right.

9 DR. WILLIAMS: And that's with lower fats, low
10 saturated fats and a little lower total fat.

11 DR. FUKAGAWA: But we also want to know what they
12 are doing cognitively later.

13 DR. WILLIAMS: They are doing well.

14 DR. FUKAGAWA: They are doing well? Okay.

15 DR. CLEMENS: Good point.

16 DR. VAN HORN: Yeah. No adverse events at all on
17 anything so far.

18 DR. PEARSON: One other point for discussion is
19 the individual fatty acids, which fall under all of the
20 unsaturated ones. Obviously there has been a
21 reasonable amount done on elaidic, as kind of your
22 poster child for your trans, and on oleic, obviously is

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1 your arch typical mono, but does anyone else have any
2 comments on specific fatty acid issues that you want to
3 address, other than as the group, you know, mono, poly?

4 DR. CLEMENS: I think in dietary guidelines we
5 obviously have a mixed message, and I will address
6 that, when it comes to food safety. One side we say we
7 should be consuming more fish so we get omega-3s; on
8 the other side we are scaring people because of the
9 methyl mercury story, and as a result, many OB/GYNs
10 have advised their patients to stop eating fish when
11 they are pregnant and when they are lactating. It
12 would be nice if we could encourage the appropriate
13 amount of -- and the ADA has done a wonderful job of
14 trying to promote the appropriate servings and the
15 appropriate types of fish during this vital period of
16 development.

17 DR. PEARSON: Yeah. I was going to get to fish
18 consumption in a little bit; maybe I will just hold my
19 comments because we are going to get off that.

20 DR. CLEMENS: By all means. I would suggest that.

21 DR. PEARSON: All right. So here is the Lumper's
22 view of dietary fats, and that is that there have been

1 also research literature done on specific fat and
2 sterol nutrients, but rather, on food and diet-based
3 issues, which really do deal with conglomerative fats,
4 so this is -- and so fish is on the top of that list,
5 and certainly I have been impressed with the
6 epidemiologic and I think some clinical trial data on
7 fish consumption relative to neural development.
8 Certainly our environmental health group had a study in
9 Saychelles, in which the hypothesis that the relatively
10 high mercury fish that was consumed by that population
11 would impede neural development, and in fact the
12 relationship between fish eating and neural development
13 was significant and direct rather than inverse. So,
14 Mim, I was right, fish is brain food, it appears. And
15 so the other question is food-based is that there are
16 some other things in fish of interest than just the
17 omega-3 fatty acids. There is some taurine data and a
18 variety of issues, as well as a good protein source,
19 but I think our group was interested in taking fish on
20 as one of the, again a food pattern discussion rather
21 than an individual macronutrient. Any other comments
22 on fish? I think we should come up with -- I think,

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1 Roger, your point about people being confused, I think
2 you are correct on that.

3 DR. CLEMENS: And I think they are confused as
4 well the type of fish that may be consumed to provide
5 these types of healthful benefits; whether these are
6 cold water fish, warm water fish; whether they are
7 farm-fed or whether they are wild. We might be able to
8 help in that regard in clarification.

9 DR. VAN HORN: Can I just ask a question that I
10 honestly don't know the answer to this? Because of the
11 interest in fish and the fact that we keep advocating
12 it, and you know, I'm all for that, is it within this
13 group's pervue to talk about the need for safe fish
14 farming and the fact that, you know, as time goes on,
15 if we want to keep recommending that the population
16 consume more fish realizing that we have a limit and
17 recognizing the importance of farming, that the mercury
18 issue is, you would think, would be something that
19 could be addressed in terms of safety of fish farming?
20 I don't know. Is that something that this group can --

21 DR. WANSINK: Sure. Yeah. This is Brian Wansink.
22 Yeah. That's a very -- within the pervue and it's a

1 good thing to do too.

2 DR. VAN HORN: Okay. Great.

3 DR. RIMM: Yeah. This is Eric Rimm. I think
4 there actually has been a fair bit of interesting data.
5 If you look at some of Emily Okin's recent data on
6 child development in mothers eating fish, that the
7 mothers that ate fish it was very important for the
8 cognitive development of the child, and if they ate
9 fish and had high levels of mercury in their hair that
10 actually hindered the development somewhat. So I think
11 we can give a clear message that fish, you know, have
12 been analyzed to death now. We sort of know how much
13 mercury is in which fish, and it does vary a bit, but
14 it's not a perfect measurement, but we still do know
15 that swordfish has a lot more mercury than salmon. So
16 I think that, you know, I think that we can make a
17 stronger statement than they could five years ago. Now
18 we should probably do it in coordination with the EPA
19 and everybody else who is trying to say the same thing
20 at the same time, so that there is a clear message and
21 there is not confusion.

22 DR. VAN HORN: Right. Right.

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1 DR. PEARSON: Another food-based issue are nuts,
2 and this obviously goes to omega-3, vegetable-based
3 omega-3 rather than marine-based omega-3 alpha
4 linolenic acid and some of those micronutrients, but it
5 might be some other issues as well relative to those.
6 But there is really quite a literature from small
7 randomized trials certainly with lipid end points, and
8 so this may be some comment, as well as it relates to
9 omega-3 non-marine sources. Any comments on nuts?
10 Yes?

11 DR. APPEL: How about the whole issue of mothers
12 being worried about allergy in children?

13 DR. PEARSON: Certainly there is with particularly
14 peanuts, but I guess that would relate to other nuts as
15 well. I'm not a --

16 DR. CLEMENS: Yes. We would include food
17 allergies.

18 DR. PEREZ-ESCAMILLA: This is Rafael. You know,
19 several scientists I remember before the last dietary
20 guidelines were issued were calling for creating a
21 separate group for nuts and not having together with
22 the protein group, with everything together. And I

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1 wonder if this is a question that this committee should
2 take on again at this time.

3 DR. NELSON: Maybe we should have a carbohydrate,
4 nut and protein, or protein, fat -- one committee?

5 DR. VAN HORN: That's an interesting thought. One
6 thing I would just add is that was actually the comment
7 that I made earlier. It would take -- I did this
8 calculation. I think it's something like you need
9 eight cups of walnuts to be equivalent to one ounce of
10 fish, in order to get the same amount of biologic value
11 omega-3. So again, the calorie, you know, contrasts
12 are huge, and so, you know, we would need to keep that
13 in mind too. Larry?

14 DR. APPEL: Yeah. Correct me if I am wrong -- oh,
15 this is Larry Appel -- I don't think we actually, in
16 the 2005, looked at the epidemiologic data between nut
17 consumption and/or work on CVD. So, recognizing what
18 you just said, we didn't -- it took a boat load -- a
19 more modest amounts of nuts, from what I understand are
20 actually associated with a reduced risk of CHD in
21 almost every study. So, we have to decide whether we
22 want to do it on, you know, have that as a research

1 question.

2 DR. VAN HORN: Right.

3 DR. PEARSON: And I guess it was my contention
4 that although certainly there are calories and
5 carbohydrates in nuts, but I thought this was largely
6 in the fat vehicle realm, in terms of what the
7 bioactivities --

8 DR. SLAVIN: This is Joanne Slavin. I think of
9 nuts more of a whole food, like whole grains that --

10 DR. PEARSON: I agree.

11 DR. SLAVIN: You know, fat is important, but there
12 is fiber in it and other micronutrients, so that's why
13 it is so protective in epidemiologic studies is the
14 package not any particular part of it.

15 DR. VAN HORN: Right.

16 DR. PEARSON: So -- well, maybe our Chair and Co-
17 Chair can assign the nuts.

18 DR. VAN HORN: You are what you eat, is that what
19 you are saying, Tom?

20 DR. PEARSON: Olive and canola oil are perhaps a
21 little bit more clearer in terms of major sources of
22 monounsaturated fats and obviously there have been a

1 number of trials with these, and obviously we have been
2 advocating fruits and vegetables. I think there
3 certainly are some obviously relative to low fat or
4 certainly high monounsaturated fats diets, as well as
5 protein sources that don't have a lot of saturated fat
6 associated with them. Obviously these have been some
7 food-based issues.

8 In terms of diet-based, many of the specialty
9 diets I think there have been some opportunities to
10 reduce a variety of nutrients, calories, carbohydrates,
11 et cetera, but many of these also focused on fats as a
12 whole food. The Mediterranean diet particularly with
13 high omega-3 fatty acids, high monounsaturated fatty
14 acids, as a characteristic. The high protein, low
15 carbohydrate diets obviously having to do with also
16 frequently high saturated fats in the setting of
17 everything, and then of course, the low fat, low
18 cholesterol diets, which have been looked at,
19 particularly given their higher carbohydrates, had
20 some, you know, increase in triglycerides and decrease
21 in HDL, as a negative consequence. Anyone who would
22 like to talk about some of these specialty diets? We

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1 hadn't really talked about these in the various places,
2 so one of the -- in an effort to be comprehensive and
3 since they are out there and frequently used at a
4 population level, I did want to bring it in, because it
5 is, to some extent, is a dietary fat issue. Eric?

6 DR. RIMM: Yeah. This is Eric Rimm. I think one
7 issue, and we don't have to put names on them, but they
8 are -- I mean, in terms of, you know, who sells the
9 book to sell the high protein diet or a low fat diet,
10 but the fact since the last Dietary Guidelines, there
11 are now a number of trials obviously, including the
12 Women's Health Initiative, where there is -- you know,
13 I think that there what will be of great interest, if
14 we want to have obesity an end point, because in the
15 end, most of them don't work that well, and it's really
16 just how well you can stay on the diet that triggers
17 the how well you -- how much weight you lose. So, I
18 think it would be a good thing to look at, and I think
19 we should focus more on longer trials than those that
20 have just gone for six months, because six months you
21 actually do see great differences depending on what
22 trial you use. And so, it's only where people can

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1 sustain a diet for over a year or two years where you
2 actually see that there is not great differences. So,
3 I think that's a very important thing to consider, and
4 a lot of that research has been done in the last five
5 years.

6 DR. PEARSON: Has there been consideration where
7 to put those in terms of the review? Is that a whole
8 -- is that a whole food issue? Because I think they
9 are out there. I think that Eric is right in terms of
10 the longer trials. Some of them actually come up with
11 some safety issues as well.

12 DR. RIMM: Yes, they do.

13 DR. PEARSON: And so, like you said, a six-month
14 look is oftentimes not adequate to look at some of
15 these other issues.

16 DR. NELSON: Mim Nelson. Well, I guess I'm still
17 advocating that we add yet another subcommittee that
18 looks at behavior, meal patterns, things like that,
19 because I think that maybe it's too hard to put them
20 into one of the other subcommittees, because it's
21 really more around the whole quality of the diet and
22 the way we eat and gets at, you know, the ease. So,

1 I'm still hoping that we can think about a subcommittee
2 that is innovative that way and then it would fit in
3 this. And my sense with most of these diets is what
4 you said, Eric, but it's really you can create a really
5 wholesome, whole foods kind of diet; whether it's low
6 fat, high protein, you know, it's more the quality of
7 the foods that are in it and whether someone can eat it
8 for a long time. So --

9 DR. VAN HORN: Well, and just the whole question
10 you just raised really, and I don't know how much data
11 there are on this subject, but the question of perhaps
12 individual preferences -- I know certainly some of the
13 work that Barbara Roles and others have done related
14 to -- you know if you are looking for eating as much as
15 you want, you know, then obviously you are going to go
16 to a high complex carbohydrate approach because you get
17 volume; but if you are going for, you know, intense
18 flavor or something like that, perhaps then a higher
19 fat type of diet but smaller portions would be your cup
20 of tea, so to speak.

21 DR. NELSON: Right. Exactly.

22 DR. VAN HORN: So, whether there are data that

1 would differentiate for people, you know, a pathway
2 that as long as the total energy intake is reduced,
3 your, you know, your approach to it could be
4 individualized, as long as you don't exceed your
5 calorie needs. That could offer people some hope, as
6 far as being able to eat the foods they really like
7 without having to, you know, totally compromise.

8 DR. RIMM: Yeah. I think there is no one diet for
9 everybody. I think there are 10 different things that
10 can work, and it just has to be something that you are
11 compliant with and that you are not eating too many
12 calories.

13 DR. SLAVIN: And I think it goes back to the
14 nutrient adequacy subcommittee too, that if you are on
15 a low carbohydrate diet, you can't get enough fiber,
16 you know? So, it does affect nutrients; that even if
17 you are losing weight, if long-term, it's not a good
18 diet because the nutrients won't go along with you.

19 DR. RIMM: Yeah.

20 DR. PEARSON: Joanne?

21 DR. FUKAGAWA: Yes. I like the concept of lumping
22 for the diets, because I think we spent the last decade

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1 or so really becoming more and more reductionists and
2 thinking in isolation of one particular nutrient, one
3 particular group, one particular vitamin or, you know,
4 source, et cetera. And I think what we're all hearing,
5 at least I'm hearing, is that it's really that
6 integration of getting back to the basics of energy as
7 conserved; that really, you know, how much you take in
8 and how much you expend is really what's going to end
9 up with the outcome of better health. And so I think
10 that's one of the challenges we have is whether or not
11 we continue to sort of stay somewhat un lumped or we
12 find a way to lump the columns.

13 DR. PEARSON: Well, yeah, this is -- we should --

14 DR. NELSON: Maybe it's a lumper's subcommittee?

15 DR. PEARSON: Well we should get back to Mim's
16 proposal there and that is to really consider what we
17 are going to do about these, because I think, at least
18 my looking at the 2005, they were really very much
19 reductionists, and it may be something maybe we want --

20 DR. VAN HORN: You know it's possible and just one
21 idea and the group can certainly discuss this or think
22 about it as time goes on, because obviously we have our

1 plate pretty full just looking at the eight
2 subcommittees that we have so far, but in some ways it
3 sounds like, from all the presentations we have heard
4 so far, that this is a cross-cutting issue that really
5 interacts with every single subcommittee that we have.
6 So perhaps one of the things that the science review
7 group could take on, as well as bringing in individual
8 representatives from each of these subcommittees is
9 exactly that. These Guidelines mean nothing if people
10 can't follow them. And so it seems to me that, you
11 know, we really owe it to the Secretaries, who both
12 gave us that mandate, to come up with something simple,
13 but you know implementable; that we would look at the
14 behavioral side of these things as well. Larry?

15 DR. APPEL: Yeah, just one comment. I spoke with
16 Trish Britten yesterday about our subcommittee
17 structure. It turns out we actually needed a
18 subcommittee on selected food groups and that emerged
19 about halfway through the process, and so maybe
20 starting earlier is better.

21 DR. VAN HORN: Okay. Good.

22 DR. PEARSON: Okay. In our one minute between us

1 and lunch, I did have one other view of dietary fats,
2 and that's the mechanistic view, and this actually has
3 already been raised by Larry about the end points, and
4 just to say that in the literature one could use the
5 variety surrogate end points to look at the fatty acid
6 effects. And, in doing this by subcommittee, I have
7 already got a reasonable amount of feedback, and so I
8 think maybe I didn't put enough question marks on
9 there, because there seems to be some difference of
10 opinion, even just the three of us about some of these.
11 Part of this has to do with relative to what, and a lot
12 of this is relative to monounsaturated fats, which is
13 kind of the standard versus more carbohydrates as the
14 source of the calories. But the point of this slide I
15 think really is the extent to which we are going to be
16 interested in some of these metabolic intermediary end
17 points. There are a number of studies of endothelial
18 dysfunction which have come up since 2005. Certainly
19 there was a very large literature, which has been added
20 too relative to the lipoproteins. There have been some
21 additional thrombosis studies; been a lot of studies on
22 information relative to the interest in a variety of

1 bio markers, c-reactive proteins, other kinds, et
2 cetera, but we should remember on the bottom line is
3 that this is all a high-density, high energy density
4 food and all of them have obviously the higher energy
5 density that obviously it needs to interface with our
6 caloric balance folks. Eric or Roger, any comments on
7 mechanism?

8 DR. CLEMENS: You know, I think it's really great
9 that we have, are going to take on these particular
10 areas of mechanisms, in particular, the general
11 consumer is very interested in this inflammatory
12 process. We see a lot of products out there about
13 boosting the immune system, and all of us knows -- each
14 of us knows that boosting the immune system is not what
15 you always want to do. It would be wonderful if we had
16 a good position, at least a collection of data to
17 demonstrate modulation of the inflammatory processor of
18 the immune system is appropriated, and from a dietary
19 perspective this is how it could be done.

20 DR. PEARSON: Larry?

21 DR. APPEL: Yeah. I don't know. I think we have
22 to be very careful about this point, getting back to

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1 the surrogate outcomes issue. I mean, I even think for
2 inflammation, you know, we are not quite sure, you
3 know, how to interpret these things, and we can, you
4 know, we can spend a lot of time sort of, you know,
5 doing literature searches on mechanisms, but never --
6 and not really change our final decision. So, I think
7 we -- I like mechanisms myself, but I'm concerned about
8 getting bogged down.

9 DR. PEARSON: So perhaps use this as more of a
10 confirmatory or --

11 DR. APPEL: Biological clause.

12 DR. PEARSON: -- not the main agenda, but --

13 DR. APPEL: A biological clause is recommended.

14 DR. NELSON: Yeah.

15 DR. APPEL: You know, these are possibilities.

16 DR. PEARSON: Exactly. Eric?

17 DR. RIMM: I'd like to second that motion. I
18 think -- this is Eric Rimm -- I think there is a lot of
19 data now on clinical events and I think that's probably
20 the most important thing that we should be looking for.
21 You know, the issue of N-6, you know, decreasing HDL
22 and increasing inflammation, and yet there is a

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1 plethora of data on benefits of N-6 and cardiovascular
2 disease. So, I think that, you know, there are a lot
3 more question marks that we can put here than arrows
4 essentially, so I think that this may be good to
5 support what we want to say, but we should stick with
6 good end points where we have them.

7 DR. PEARSON: Other comments?

8 (No audible response).

9 DR. PEARSON: If not, I think -- I want to thank
10 my partners on the subcommittee, Eric and Roger, for --
11 we had met before about this and the associated
12 discussion. I don't know. I have about 12 areas of
13 recommendations we can certainly sink our teeth into.
14 And I want to thank the group for their addition to
15 this facilitative discussion.

16 One of the areas that I think we'd like some input
17 on is outside individuals that we may want to bring in
18 for a consultation here, and so we are certainly open
19 to suggestions. Dr. Van Horn?

20 DR. VAN HORN: Right. Thank you. I think each of
21 the groups is probably looking for similar input, so I
22 think we all ought to be open to those ideas.

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1 I want to thank the committee -- Tom, you and your
2 group -- but everyone this morning. I think it's been
3 absolutely a rich and full, and incredibly valuable
4 discussion, so I want to thank all the panelists for
5 the excellent work they have done. And now we will all
6 adjourn for lunch and return at 1:30. So thank you.

7 (Whereupon, at 1:14 p.m., a lunch recess is
8 taken).

9 DR. WANSINK: Last week, at the American Dietetic
10 Association, I was there and this person came up to me
11 and said, I think you are sending a terrible signal by
12 having the Dietary Guidelines Advisory Committee meet
13 on Halloween. Ahhh. She says, just think about, just
14 think about it. And I did think about it, and what I
15 thought is that this is the only holiday that has its
16 only food pyramid. So thank you very much for taking
17 your Halloween and the trick-or-treating with the kids
18 to be here today. Linda?

19 DR. VAN HORN: Well, on that note, I think we'll
20 talk about Ethanol. Okay. We are good to go. We are
21 going to talk about Ethanol, and Dr. Rimm is in charge.

22 DR. RIMM: Thank you very much. All right. What

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1 I'd like to do for my talk is take a format -- I'm also
2 from the Midwest, so like Joanne I'm going to just do
3 it as I was told. We are going to take the format of
4 reviewing what was done in 2005, questions asked and
5 conclusions from the Technical Report, and then go to
6 the 70-page key recommendations, and then talk about is
7 there new evidence for 2010, and if there is are there
8 are there new questions that we could ask?

9 So, in 2005, several questions were -- the
10 Committee came up with several questions, and I'll put
11 together the evidence to answer these questions. The
12 first was, among persons who consumed four or less
13 drinks per day, what is the dose response between
14 alcohol and health? A pretty global question, but I
15 think what came out of it was what is now, I think,
16 quite well accepted in the scientific community is that
17 one to two drinks a day lowers total mortality; one to
18 two drinks per day is associated with lower risk of
19 coronary heart disease; one drink slightly increases
20 breast cancer; and alcohol risks and benefits do not
21 differ between middle age and elderly people, but there
22 is little if any benefit for younger people. This is

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1 actually verbatim from the -- these are the words and
2 the word-smithing that went on to fit into each one of
3 these categories. So, I think this is important
4 especially the last point, because I'd like to bring
5 this up again when we get to the questions asked.

6 The next question asked is what is the
7 relationship between consuming four or fewer drinks and
8 macro or micronutrient profile in overall diet quality.
9 I think we have touched on this several times today
10 about looking at the overall diet, and the question
11 that was posed here was, if someone does drink four or
12 fewer drinks, does it actually impact the rest of their
13 diet. So not biologically what happens, but if someone
14 drinks, do they have an insufficient micronutrient
15 intake, or do they change the composition of their
16 macronutrient intake? And, what they found based on
17 national data is that individuals who drink one to two
18 drinks per day, it is not associated with an inferior
19 diet quality. And this was done through several
20 different measures, but specifically for macro and
21 micronutrient intakes or profile, they found that
22 people who drink don't have necessarily any worse off

1 diets. I don't think that's a proper terminology, but
2 you get the idea.

3 So going back to the technical report into the key
4 recommendations, this actually made it in I think as
5 almost one of the next to last chapters, chapter 9 on
6 alcoholic beverages, and so the recommendations there
7 were those who choose to drink alcoholic beverages
8 should do so sensibly and in moderation. And,
9 interestingly, if you go back to the history of the
10 Dietary Guidelines, back to the first one in 1980, this
11 is one of the few guidelines that has almost not
12 changed since 1980. Again, there has been a little bit
13 of word-smithing that has gone on. Now it says
14 sensibly. I don't think it said sensibly back in 1980.
15 But, for the most part, the guideline has stayed the
16 same. The wording that has come after it has changed a
17 lot. In just terms of -- just because there has been much
18 better science in the last 25 years to study the health
19 effects of alcohol.

20 And then consumption is defined as one drink a
21 day; moderate consumption is defined as up to one drink
22 per day for women and up to two drinks per day for men.

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1 And again, that's meant to be per day and not
2 necessarily an average per week where someone could
3 have all their consumption on Friday and Saturday
4 night. I didn't mean for that to be a joke, but thank
5 you very much for joining in. Alcoholic beverages
6 should not be consumed -- again this is verbatim --
7 should not be consumed by some individuals, including
8 those who cannot restrict their alcohol intake; women
9 of childbearing age, who may become pregnant; pregnant
10 and lactating women; children and adolescents; and
11 individuals taking medications that can interact with
12 alcohol. And again, this has changed somewhat over
13 time as there has been more evidence, but for the most
14 part, in general, this has covered similar populations.
15 And then of course, those also with specific medical
16 conditions. Alcoholic beverages should be avoided by
17 individuals engaging in activities that require
18 attention, skill or coordination, such as driving or
19 operating machinery, or potentially sitting on a
20 Dietary Guidelines Committee.

21 So the definition for moderate drinking is defined
22 as 12 ounces of regular beer; five ounces of wine; and

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1 one-and-a-half ounces of 80-proof distilled spirits.
2 And in the technical report they go through the
3 discussion of how much is consumed in this country and
4 how that has changed over time. What I find is
5 interesting, if you can see this, is this actually was
6 in the 70-page Dietary Guidelines, is that several
7 different beverage types are listed, as well as -- if
8 you look at the last two columns -- the average portion
9 size -- so that's 12 ounces for beer; five ounces for
10 wine; three ounces for sweet dessert wine and one-and-
11 a-half ounces for 80-proof distilled spirits. And the
12 last column is the calories and the calories, if you
13 can read the small print way down at the bottom, are
14 based on release 17 of the USDA nutrient database or
15 standard references. So I looked this up, they are now
16 up to release 21, and release 21 actually -- because
17 the alcohol content of beverages has increased in the
18 country, beer is now over 150 for a regular beer; red
19 wine and white wine are now up at like 120 to 130
20 calories per same five-ounce serving, only because the
21 amount of alcohol has changed. Obviously, if you are
22 measuring 80-proof distilled spirits, it's still going

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1 to be the same calories because it's 80-proof distilled
2 spirits. So the question is, what are people
3 consuming, and in fact, we should at least make, take
4 this into consideration when looking at total caloric
5 intake, if we are talking about average consumption of
6 alcohol.

7 So the research recommendations for 2005 - and
8 Larry mentioned that you and your committee at the time
9 didn't spend as much time talking about research
10 recommendations, this was mostly saved for the last
11 meeting so, you know, this may not be as important. If
12 they wanted to investigate the relationship between
13 alcohol consumption and obesity, this obviously is
14 quite important, and the evidence they had concluded at
15 that time was that there was insufficient evidence. It
16 didn't look like alcohol in moderation was associated
17 with obesity. They also had some more policy issues,
18 which was to investigate the impact of adding calorie
19 information to the labels of alcoholic beverages,
20 including whether, for educational purposes it would be
21 sufficient to include only calories. I know there has
22 been a movement afoot to do that. Thus far I don't

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1 believe that calories are added -- are mandated to be
2 added to the labels of alcoholic beverages.

3 Investigate the impact of banning alcohol advertising,
4 when and where it might increase underage drinking,
5 during college sporting events, for example. This
6 obviously is a very important issue that universities
7 struggle with across the country as the problem of
8 excessive or binge drinking among college students,
9 either of age or not of age. And also, to investigate
10 the impact of a unified federal message on alcohol and
11 health through increased collaboration across agencies
12 or consolidation of authority under one federal agency.
13 I am guessing this is way beyond the purview of the
14 Dietary Guidelines, but it was an interesting thing
15 that was brought up in 2005.

16 So, 2010 Guidelines, is there evidence; are there
17 new questions to be asked? I think there is a bit more
18 evidence on drinking patterns. This is a challenging
19 one, because when people -- when you say drinking
20 patterns, the first response is, are you talking about
21 binge drinking? And that is one aspect of drinking
22 patterns. I think another aspect of it is people who

1 drink every day versus many who drink many days per
2 week but don't drink every day. The issue is of, you
3 know, transitions from certain drinking, types of
4 drinking patterns to actually excessive alcohol
5 consumption and alcohol abuse.

6 Just a few things that we have looked at and our
7 cohorts -- this is a cohort of 50,000 men looking at
8 alcohol and coronary heart disease -- and on the bottom
9 axis you can see the number of days they reported they
10 consumed alcohol, and you can see that the most benefit
11 came when you drank at least every other day, and there
12 wasn't additional benefit from drinking more days. And
13 even among the categories of days per week, you can see
14 the amount they were drinking actually didn't make that
15 much difference. It was mostly important to get
16 alcohol in the system at least every other day to -- I
17 shouldn't be saying that with a toxicologist here --
18 it's not meant as a pharmaceutical, but more as people,
19 you know, who were drinking at least every other day
20 were getting a benefit, and drinking beyond that there
21 was not further benefit. We also looked among people
22 that were the healthiest of healthiest in our cohort.

1 Of the 50,000 men, only 3,195 men were not overweight,
2 didn't smoke, had a healthy diet and had regular
3 exercise. Even among those men there was about 200
4 cases of MI (myocardial infarction) over the course of
5 16 years of follow-up, and we still found that days per
6 week of alcohol consumption lowered the risk of heart
7 disease among these very healthy individuals, who
8 otherwise didn't have standard risk factors.

9 So, I put this slide here mostly to remind me of
10 the other question, which is, is there new evidence of
11 health effects in vulnerable populations? That was
12 among the healthiest of healthiest, but with 60 percent
13 of the population overweight and more people with
14 diabetes and hypertension, there hasn't been, I don't
15 think, necessarily a single message of what to do, what
16 to tell people about alcohol among individuals who are
17 otherwise compromised or at higher risk for other
18 chronic diseases.

19 This was a recent metabolic study or a clinical
20 trial of alcohol that was done in Israel, and it's
21 interesting. They actually randomized people who
22 seldomly drank alcohol, so they weren't lifetime

1 abstainers, but they were people who seldomly drank
2 alcohol, and they were randomized into either one drink
3 a day on the right side, or a controlled beverage which
4 did not have Ethanol in it. And this was just looking
5 at their fasting plasma glucose. And you can see that
6 on the left side in the control group, they started on
7 average at a fasting glucose of 136.7 and went to
8 138.6. These are among all diabetics in Israel, so as
9 it is, their glucose levels are quite high. The
10 control group was not affected by alcohol. If you look
11 at the right side, those that consumed one drink per
12 day, they had about a 20 milligram per deciliter drop
13 in their glucose after a 12-week period. So this is
14 just to say that it is interesting to think about the
15 impacts of alcohol among a high-risk population. The
16 American Diabetes Association actually takes on the
17 same stance as the Dietary Guidelines; that is, those
18 who drink can do so in moderation.

19 Health effects of new drinkers. This is a very
20 challenging question which we may not ever have
21 evidence from a clinical trial looking at the impact of
22 alcohol long-term among new drinkers. There was a

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1 recent study published from the ARIC study. This is
2 not my study even though the name is quite similar. It
3 actually stands for Atherosclerosis Risk In Community
4 Study. It's done from four different communities
5 around the United States representing different
6 ethnicities. And it's a little tricky, but essentially
7 what they had is a lot of -- they looked at the non-
8 drinkers at baseline at visit one, and visit three six
9 years later, actually some of them had started to
10 drink, either moderately or heavy, and they were able
11 to follow them after that time period to see who
12 developed coronary heart disease. And, believe it or
13 not, there were some people who were middle-aged, who
14 started drinking who previously had been non-drinkers,
15 and if you look across at their odds ratio among
16 moderate drinkers, their odds of developing heart
17 disease was .62. So this was, I think, one of the
18 first studies that had enough statistical power to look
19 at what happens if non-drinkers take up moderate
20 drinking in mid-life. I had very few people who became
21 heavy drinkers, and therefore the comparison levels
22 were quite wide, .41 to 4.9. So really I can't think

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1 -- I think we can't draw much from this. The author's
2 conclusion from this was that there aren't many people
3 who become heavy drinkers, who convert -- who start
4 drinking in middle age.

5 So, you know, there is not enough data here to
6 come up with a strong statement, and I don't think we
7 ever would come up with a statement that people should
8 start drinking alcohol, but at least there is some
9 evidence that among middle-aged individuals there is
10 not a lot of people who go on to become heavy drinkers,
11 who started moderate.

12 So, new questions to be asked? We had a quick
13 circulation of emails among individuals on the
14 committee, Larry Appel and Tom Pearson, and I guess
15 some of the challenges, one of them that I thought we
16 should look at is that, well one to two drinks per day
17 is not associated with a poor quality diet, as
18 concluded in the 2005 Guidelines. Biologically alcohol
19 does impact a lot of important metabolic systems, and
20 specifically we know, even though you may have the same
21 diet, that drinkers actually may not absorb folate as
22 well or alcohol may actually interfere with use of

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1 folate. And there is a number of other biological
2 systems, which I think there is now beginning to be
3 enough data that we could look at. I don't think we
4 would make special, necessary special dietary
5 guidelines for drinkers, but I think it's enough to be
6 -- there is enough data out there that we should be at
7 least aware of the fact that alcohol does, even at
8 moderate levels, interfere with metabolism and
9 absorption of micronutrients. So that may be one area
10 that we can ask.

11 The second, another area that we thought of is
12 that, who should not drink. I provided a list of
13 groups or categories of individuals that should not
14 drink, and there may be growing literature on other
15 individuals or people with certain family histories
16 that also should be given guidance potentially not to
17 drink or to drink less. And I think related to that,
18 are the Guidelines explicit enough on contraindications
19 for alcohol consumption?

20 The next area which I guess maybe I disagreed with
21 the Guidelines in 2005 is, does one to two drinks per
22 day really have little if any benefit for those less

1 than 30 -- less than 45 years of age? That statement
2 was based mostly on the benefits for cardiovascular
3 disease, and there is very little cardiovascular
4 disease among individuals less than 45, so while I can
5 see where that conclusion came from, if we think about
6 most people who have heart disease when they are 50 or
7 60 or 70, if they are getting benefit from alcohol,
8 it's very unlikely that all of them started drinking
9 when they were 45. It's more likely that they started
10 drinking at an earlier age where they already were
11 accruing some of the benefits; the increased HDL
12 cholesterol; the decrease in clotting; and the probable
13 benefit on atherosclerosis. So, I think that there
14 probably is -- cardiovascular disease takes 30 or 40
15 years to develop, so I think to say that there is only
16 benefit among people over 45 may be too broad of a
17 conclusion.

18 But also importantly and related to the headlines
19 today on diabetes, there is now at least 10 studies,
20 which show prospectively that individuals who drink one
21 to two drinks per day have about a 30 to 40 percent
22 reduction in risk of developing Type 2 diabetes. And

1 that may be related to lower levels of glucose, an
2 increased insulin sensitivity related to moderate
3 alcohol consumption and alcohol blunting the glycemic
4 effect of a meal.

5 So, that's sort of where we stopped. There
6 probably are other questions that could come up with
7 alcohol. I don't expect that the bottom line will
8 change much but I think we could explore some
9 questions and potentially expand our guidance for some
10 certain subpopulations and for certain age ranges. So,
11 I will leave it there. I hope I went quick enough to
12 stay within my allotted half hour Ethanol time?

13 DR. VAN HORN: Excellent. Does anyone else on the
14 subcommittee have anything, Larry or Tom?

15 DR. APPEL: Yeah. This is Larry Appel. I --
16 thanks, Eric. I had one question that's a little bit
17 out of the box, but it pertains to the fact that
18 alcohol is something that you are not supposed to
19 consume and then you do consume it, and we know that
20 this transition period is a big problem. And one
21 question that, if I could, I threw out, to paraphrase
22 this, are there healthy patterns of starting drinking

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1 and what are those, which is a little bit different,
2 but it relates to behavior issues, but that might be
3 something we should at least put on our list to
4 consider.

5 DR. VAN HORN: Good point. Tom?

6 DR. PEARSON: For the nutrition committee of the
7 American Heart Association, I wrote some guidelines
8 about 10 years ago on this subject, and we actually did
9 an analysis addressing the CDC's recurrent information
10 about 100,000 excess deaths in the United States due to
11 alcohol consumption every year. Looking at also if we
12 assume that everyone were a tee-totaler, what would be
13 the effect, and it's about an 86,000 excess deaths. So
14 it's about a wash. So you have this U or J, or
15 whatever you want to call it, on both sides of it. So
16 it makes it, for a messaging issue, very difficult.
17 The problem is that the MI benefits are all in the
18 middle-aged and older individuals, and many -- not all
19 -- but many of the auto accidents, violence and issues
20 are in the younger people. So this issue that Larry
21 raised I think is very important, and I may need to go
22 up one more step and maybe at this point look at the

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1 evidence to say, are there any interventions in which
2 young people's behavior can be changed so that they are
3 using this as a beverage and not a drug? Because
4 that's really the issue. Is it the -- the excess
5 mortalities is when alcohol is used as a drug and not
6 as a beverage. And I think what the nutrition
7 guidelines ought to do is to see as many people who
8 could be curtailed from using this as a drug and moved
9 over into the one or two drinks a day and really have
10 it all beneficial. So, I would go one more step than
11 what Larry said, and that was not only to the
12 observational studies, but if there had been any
13 interventions.

14 DR. VAN HORN: Very good.

15 DR. ACHTERBERG. Cheryl Achterberg. I liked those
16 comments. And this might be a marginal addition, but
17 nonetheless if we are going to be looking at these
18 drinking patterns and so forth, it might be worth
19 noting somewhere that there are over 21 university
20 presidents now working together to create a
21 recommendation to lower the drinking age to 18. So as
22 this committee is working, we might want to be

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1 monitoring that, if we decide to make a statement.

2 DR. WILLIAMS: Christine Williams. I have a
3 question about, with the growing numbers of overweight
4 and obese individuals and the non-alcoholics data on
5 hepatitis, what is the effect of moderate alcohol
6 intake on those individuals?

7 DR. RIMM: Yeah. You know, I think that's a
8 really important question. I think there is sort of
9 two separate issues biologically what's going on, I
10 mean, what has been studied in very clinical detailed
11 clinical studies, and then epidemiologically, what do
12 you see if you study hundreds of thousands of people?
13 And the hundreds of thousands of people ultimately are
14 at risk for diabetes first, and in those populations
15 alcohol actually is beneficial when consumed in
16 moderation; and when not consumed in moderation
17 actually is detriment. And so it is still -- that has
18 not changed. The underlying population at risk has
19 changed, and so generally what you see is a greater
20 benefit than you would have seen in a lean population,
21 but you also see potentially greater risk, because they
22 are already at -- they already have (inaudible) related to

1 obesity, so it makes it even more of a challenge to
2 come up with sort of a simple, you know, phrase that
3 you can do to capture the entire population.

4 DR. VAN HORN: Tom?

5 DR. PEARSON: Another implementation issue really
6 is to not only look again at the algorithms or
7 whatever that would identify people who shouldn't drink
8 at all. I mean, certainly there are some people who
9 shouldn't drink at all, but the extent to which they
10 are actually implemented, I mean I think -- I think
11 most people find out they shouldn't drink at all by
12 drinking excessively and then ending up in our
13 hospitals with pancreatitis or addicted to alcohol, et
14 cetera. And it would be helpful if we could come up
15 with ways that work so, you know, we don't have that.

16 DR. FUKAGAWA: Is it also -- this is Naomi -- is
17 it also possible to consider where these three carbon
18 fragments go in terms of its impact on lipogenesis or
19 lipid metabolism, because you know it is another source
20 of Acetyl CoA, which could go down fatty acids side? I
21 don't know.

22 DR. PEARSON: I think the issue is lipogenesis

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1 that we need -- it affects lipogenesis about 14
2 different metabolic steps and --

3 DR. WILLIAMS: Yeah. Okay.

4 DR. PEARSON: I think that's pretty well worked
5 out. What I am not sure is that unless there is some
6 new health effect of one of these carbon fragment
7 metabolites that I don't know about -- I think it's
8 been pretty well identified. You are right it is a
9 source of Acetyl CoA for sure.

10 DR. WILLIAMS: So having it as a source -- okay.
11 Yeah. Yeah.

12 DR. PEARSON: And obviously there is a lot of
13 empty calories there, but I'm not sure there is
14 anything new.

15 DR. WILLIAMS: Right.

16 DR. RIMM: Right. I mean I think that's what the
17 last Dietary Guidelines started with is, you know, are
18 there empty calories, does it really impact everything
19 else?

20 DR. WILLIAMS: Yeah.

21 DR. RIMM: They are not going to be worried about
22 the fact that people are displacing it for fat,

1 carbohydrate or protein. It didn't look like that when
2 you are drinking in moderation. Obviously that's not
3 the case when you are drinking more, and it does start
4 to impact metabolism and fatty acid metabolism, as well
5 as displacing micronutrients and interfering with
6 micronutrient absorption. So, I think at the high end,
7 the message definitely shouldn't change. The message
8 was quite strong in the last Dietary Guidelines about
9 drinking excessively, so.

10 DR. VAN HORN: Good. Any other comments? All
11 right. Very good. Thank you very much.

12 And for our last discussion this afternoon, we are
13 going to hear about food safety and technology, and
14 that will be Roger and Rafael. Right. Okay.

15 DR. CLEMENS: Thank you, Linda. This is a joint
16 project with Rafael, so we have a good space of
17 population representation, with any luck at all. We
18 realize too that food safety is paramount and everyone
19 wants to be responsible and everyone should be
20 responsible. We noticed that in the opening remarks by
21 both Secretaries and the Under Secretaries that food
22 safety was part of their presentation. It's on the

1 tips of everybody's tongue. It's a major
2 responsibility within all the major agencies, so we --
3 from farm to fork, from good agriculture practices to
4 the dining room table, food safety continuum is
5 everyone's responsibility.

6 As we look at the Dietary Guidelines that we
7 presented last 2005 certainly these are the major
8 bullets that were presented in that fine report. In
9 this particular case we look at separate, as Rafael and
10 I collaboratively discussed the options, we find that
11 particularly in low income areas that the separation
12 and the appropriate use of utensils and cutting boards
13 alike sometimes don't permit or do not execute the
14 separation of foods, therefore leading to contamination
15 between raw foods and contamination of cooked foods,
16 and that's totally inappropriate.

17 The Guidelines do a very nice job in outlining
18 what the responsibilities are and what each step
19 represents. From an international perspective, these
20 are adopted icons. We notice that the last report did
21 not use these icons. They were just recently
22 developed. As I and Rafael have traveled

1 internationally, we find that these icons are now being
2 used more and more in various professional
3 organizations and in restaurants and hotels alike to
4 encourage the locals to wash their hands; to wash food
5 contact surfaces and so forth. Importantly we find
6 that a temperature and time of proper cooking is an
7 issue, both domestically and internationally, and
8 certainly this is a big message that has been purveyed
9 across the consumer groups. What we see now is we look
10 at a number of groups and the last icon is showing the
11 temperature range which is considered safe versus the
12 temperature range which is considered dangerous. There
13 has been a change in that range in the last five years,
14 which would be pertinent information to be discussed
15 and presented for the next Dietary Guidelines.

16 Another wonderful project that was supported by
17 the last Dietary Guidelines and also with the FDA, the
18 USDA is -- these various bullet points -- clearly the
19 food storage and spoilage we often forget about
20 spoilage organisms, and if you look at the history of
21 food usage that many cultures, and depending on your
22 lineage and your heritage, you will see that you may

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1 have a spoiled cheese, but often we fail to discard it.
2 We just cut off the obvious and get rid of it and don't
3 get rid of it rather. We know that can cause some
4 issues as well. We are very pleased to see that
5 listeriosis was brought to the attention the last time,
6 and we want to look at other microbes that may be
7 affecting the food supply as well, and particularly in
8 the home. As we mentioned earlier, that methyl mercury
9 and one area that we examined most closely and we hope
10 that we will examine more carefully this time around as
11 well is that one side we are saying we are concerned
12 about the methyl mercury in fish and the other side we
13 are saying we should be consuming more fish for a
14 number of health reasons. In my own case, and my
15 daughter just delivered our first grandchild, her OB/G
16 said don't consume any fish; at any other time we say
17 we should consuming fish. So you see we get mixed
18 messages on the healthful benefits of fish because of
19 the potential methyl mercury.

20 There have been a number of new data that have
21 come out that are now available and it's incumbent upon
22 us to re-examine those data to health risk benefits.

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1 And a really nice survey that was conducted
2 recently by the International Food Information Council
3 published just earlier this year, you will see that
4 some of the messages in terms of food safety
5 preparation in the home, and in fact that message is
6 getting out to the audience and we are really pleased
7 about that. We see that people are now washing their
8 hands at least more frequently. And when it comes down
9 to one of our pets, that is, Rafael's and mine, that
10 separation is not quite clear amongst the population
11 and group. And this we suspect is quite obvious
12 regardless of your socioeconomic status in various
13 cultures. We need to identify this in some way to
14 communicate this more effectively to the population
15 groups.

16 Very few are using the thermometer, whether they
17 have it over a grill or in a microwave. You see it
18 down there at the bottom of this particular graphic.
19 Thermometers and microwaves historically did not mix,
20 yet today's technology says that thermometers are quite
21 available, and in fact, you can monitor the internal
22 temperatures of foods that are being cooked and cooked

1 properly.

2 Overall, what are the consumer expectations in
3 terms of food safety? The consumer expectation is that
4 all food should be absolutely safe. A wonderful ideal
5 -- not possible. We know that all foods carry a
6 natural or unnatural -- carries some form of risk,
7 therefore, the other button that is pushing loudly with
8 most consumers is it's all natural. Most consumers
9 feel today that natural means safe. Those of us
10 involved with food toxicology are realizing the fact
11 that natural does not necessarily mean safe and we can
12 identify a number of those areas. This may be an area
13 for us to add to the chapter in terms of food safety.

14 Consumers want convenience and yet fresh.
15 Sometimes those are like oxymorons. What is fresh? It
16 has not been defined. But they do want convenience,
17 and partly -- and maybe part of that, they want to cook
18 a meal in ten seconds and then enjoy that meal. And
19 that's probably with fast food, rapid preparation,
20 hurry up and go. We need to sit down and really enjoy
21 the meals together like we have enjoyed the lunches
22 together.

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1 Positive messages? There are a lot of negative
2 messages relative to food or food components. We would
3 like to consider a positive message what foods deliver,
4 and today this group is looking at foods in total as a
5 positive way to deliver food messages.

6 We know that there are a number of technologies to
7 improve the quality of life, as well as the quality of
8 food. We are going to address that in the next
9 graphic, but also address the movement, in terms of
10 locally grown, in an effort to control costs and food
11 quality. Well some of those issues are right here, in
12 terms of the home kitchen is the last line of defense.
13 It's up to the individual -- it could be -- well, the
14 home kitchen, it could be the back seat of the car that
15 also has some impact on food safety.

16 Well a number of big organizations are conveying a
17 message, and our hope is that we would have a message
18 in food safety that is consistent with this and
19 supported by the National Restaurant Association, in
20 terms of serve safe all restaurants, all professional
21 chefs, all major food companies that are involved with
22 this safe serve application. And then various messages

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1 here we want to be sure we get our data out to the
2 general consumer as well.

3 In addition, there is a great website, wonderful
4 information available through www.foodsafety.gov,
5 wonderful pull-down messages. Again, we want to be
6 certain that we have continuity and harmonization
7 messages to be delivered between the report that we are
8 generating and the messages that are available at this
9 website.

10 Well, where are we going? The technology will
11 hopefully help the consumers of tomorrow and the
12 consumers of today with these type of tools. We know
13 that the films and the saran wraps, if you will, of
14 today will be different of tomorrow. We know that the
15 packaging materials will be different tomorrow. Those
16 packaging materials and wraps and films will actually
17 improve stability. They will include oxygen
18 scavengers; they will include various sensor noses, if
19 you will, so they will automatically tell you whether a
20 product has spoiled. It will tell us the heat shock,
21 the heat exposure and thermal profiles that our foods
22 have undergone so that in fact we can ensure a safe

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1 food supply, particularly in the home. At the end of
2 the day we want to be sure that food safety drives the
3 technology and resources, sanitation resources that are
4 in the kitchen.

5 And lastly, we want to know, perhaps not addressed
6 by the last group, that is, what is natural and does
7 natural always mean safe? Can we give some guidance in
8 this area relative to the safety of natural foods and
9 really educate the consumer about this very important
10 topic. We know very much that organic foods have
11 received about four percent or so penetrations in the
12 U.S. market. The national organic program is very
13 helpful in educating the consumer, as well as the
14 farmer, and yet we also know the research that I have
15 done and many other -- and Joanne brought up some of
16 this information yesterday -- that organic does not
17 necessarily mean safe. And the only work that we have
18 done at the USC, examined the bio burden of organic and
19 in conventionally farmed foods, and in this particular
20 case we demonstrated in many cases that organic foods
21 have a much larger bio burden than those which are
22 conventionally produced.

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1 We want to look at that. In addition to some of
2 the methodologies that are being used today to assess
3 microbial loads, we note that we found at least that
4 many of the organisms that we see are much slower
5 growing than the BAM actually will detect, and could
6 that in fact suggest we may want to look at additional
7 methodology, maybe outside the pervue of this group,
8 but yet it is something for us to discuss.

9 Buy local -- this is clearly a big movement across
10 the country and around the world. While we support the
11 buy local movement the buy safety is really more
12 important. We know that many of the farms do not
13 practice good agricultural practices. When you ask
14 them in the farmer's market, what is organic, they
15 don't know what organic means. They just display the
16 placquard. So we need to educate the farmers, as well
17 as educate the consumers, when they buy local be sure
18 that they buy safe.

19 Another area that's important to us, and this is
20 mentioned by the opening remarks by the Secretary is
21 that the generation, the baby boom generation is about
22 to retire. In that retirement generation we see that

1 these individuals are polypharmate; they are taking
2 multiple medications. The message of the food
3 interaction, particularly the nutrient interaction with
4 the medications is not well-known. I reflect back on
5 the days that Daphne Rowe started wonderful work at
6 Cornell. Unfortunately, a traffic accident took her
7 life much too soon. We know that antibiotics certainly
8 deplete the microflora in the G.I. tract. We know
9 certain that antihypertensive medications obviously
10 affect electrolyte balance. We know that some
11 antibiotics affect the mineral absorption. We know
12 that some anti-seizure medications affect folic acid
13 metabolism. The length of and degree of what we know
14 about medications is growing. Maybe we have an
15 opportunity here to educate the aging population so
16 that as they take medication, we can work with the
17 professionals and provide this great educational
18 opportunity.

19 And lastly, well who can do it? Well, we know
20 that Dr. Carl Winter, who is a noted food safety expert
21 and toxicologist has a good handle in really studying
22 the dynamics of the increased fruits and vegetable and

1 grain production and consumption may have in terms of
2 exposure to not only environmental toxins, but in
3 toxins that are naturally occurring in foods that you
4 and I have come to enjoy.

5 In addition, Dr. Peter Preston, who is a
6 Lieutenant Commander with the U.S. Navy, with the
7 Medical Corps in Jacksonville, Florida, has
8 considerable expertise of food drug interactions. It's
9 those kinds of interactions that we need to have a
10 better grasp.

11 With that, Rafael, any remarks, please?

12 DR. PEREZ-ESCAMILLA: Thank you for an excellent
13 presentation, Roger. I want to add that although for
14 some food safety behaviors the awareness may have
15 increased in the continuum of consumers, national
16 surveys continue to show that we consumers
17 underestimate considerably the risks of potential food-
18 borne outbreaks at home. And what complicates matters
19 very much is that a lot of these outbreaks are never
20 reported, so from the epidemiological point of view, we
21 need better methods to do better monitoring and
22 surveillance of these food-borne illness outbreaks at

1 home. And this is very important, because if this data
2 confirms that a very good percentage of food-borne
3 illnesses originate at home, that is likely to change
4 the attitude of consumers towards to food safety, and
5 our research shows with microbiological health outcomes
6 that the attitude that people have towards food safety
7 matters in terms of their practices. So, in terms of
8 changing food safety behaviors, addressing changes in
9 the attitude towards the risk of home-based food-borne
10 outbreaks I think is -- food-borne illness outbreaks is
11 important.

12 I find the messages and recommendations from the
13 2005 Dietary Guidelines that I have in front of me very
14 complex. I think we need to do a better job at
15 explaining to the public what cross-contamination
16 means. If you saw in the data presented by Roger that
17 separation which is aggressive cross-contamination is
18 one of the behaviors that is practiced the least and
19 probably not understood very well. Thank you.

20 DR. CLEMENS: Thank you, Rafael. Comments?

21 DR. VAN HORN: Tom?

22 DR. PEARSON: Yes. Is there an issue of the

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1 globalization of the food supply -- this is Tom Pearson
2 -- the globalization of the food supply and issues that
3 you are going to bring up relative to food safety? I
4 mean, obviously a number of both environmental and
5 individual product safety issues might be attended to
6 here, but not necessarily elsewhere, so.

7 DR. NELSON: Could I? I mean, I would just add, I
8 don't know that it's the globalization, but it's also
9 the sort of the food processing, the way, I mean
10 especially meat and other things, you know there is a
11 real food safety issue that -- it's sort of -- I mean,
12 it's sort of lumped together with that. This is Mim
13 Nelson.

14 DR. CLEMENS: Sure. The same issue. Yeah.

15 DR. NELSON: Yeah, the same issue. Just, I am not
16 sure we want to link organic and local food in the food
17 safety piece. I think it's another issue. I think
18 overall food safety cuts across all types of foods.
19 So, I think it might be a little bit sort of strange to
20 put them in there. I don't know.

21 DR. VAN HORN: Go ahead.

22 DR. POST: And I thought you were getting at issues

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1 like nitrates, you know? Is that something that -- I
2 mean, I don't think that's covered any other place, you
3 know. Does, you know, barbequing certain meats lead to
4 a certain, you know, an increased risk of certain forms
5 of cancer.

6 DR. NELSON: I think that -- this is Mim Nelson
7 again -- but we spoke a little bit in one of our
8 subcommittee meetings about phone calls just, just all
9 the things that are being added to foods in terms of
10 the functional foods and everything else. I mean, a
11 lot of them we don't know what the safety is and that
12 seems like it probably should be in this committee.
13 Would -- I mean not that we are trying to give you more
14 work, but it's, you know, it's the food additives of
15 all sorts I think is the range.

16 DR. CLEMENS: Yeah, and maybe -- this is Rog --
17 maybe the food additives -- this is really interesting,
18 all of the food additives we see here in the United
19 States are of course considered safe. What's
20 interesting now is that more and more we see a number
21 of herbs and spices and the blends of some of these
22 things are almost at the pharmacological dose. And as

1 we look at the pharmacology of some of the components
2 and the mixture of these components that may raise a
3 number of eyebrows and deserves further attention.

4 DR. FUKAGAWA: This is Naomi. I think I think
5 back on Julia Child's later interviews and she was
6 asked, what has changed the most about eating and food
7 in her career, and she said it was really the issue
8 that people have become afraid of their food. And
9 that's perhaps the biggest change that she has seen,
10 and it's more than just the food safety, but being
11 afraid of fat; being afraid of salt; being afraid of,
12 you know, insufficient this or insufficient that. So I
13 think if we do something that's potentially valuable
14 for the public, it would be trying to narrow and
15 simplify our messages.

16 But I would like to follow-up on Mim's thing about
17 organic and lumping together local food and, you know,
18 your concern about bio burden, and that in many ways
19 some of the issues that we are dealing with is the fact
20 that the boomers have gotten even afraid of microbes.
21 And microbes and bio burden is not necessarily bad for
22 general health, and in some ways, some of the problems

1 we have encountered, especially with resistant
2 bacteria, you know, in the hospitals and things are
3 related to over concern about this type of hygiene. So
4 I think we have to sort of try to balance, you know,
5 that entire message.

6 DR. PEREZ-ESCAMILLA: This is Rafael. I want to
7 also mention that as we are aware there have been
8 recent of food-borne outbreaks related to the
9 consumption of fresh vegetables, and some of that food
10 has come from outside. Some of those outbreaks, like
11 the spinach one, was food grown in the U.S. But the
12 point is that the washing, proper washing of the fresh
13 produce I think is an issue that is much more urgent to
14 be addressed now than it was perhaps even five years
15 ago.

16 DR. SLAVIN: This is Joanne Slavin. I think
17 especially as we promote fresh, we forget about that
18 food processing cleans things out, you know, like why
19 -- people always say, why did we take whole grains and
20 make them less healthy? Well, whole grains from the
21 healthy are not very clean, so there is a lot of
22 processing that's really positive, so to not lose sight

1 of that.

2 DR. NELSON: Mim Nelson. I have a question.

3 Where are we going to be dealing with all of these

4 things that are now added to a lot of different foods,

5 like isolated isoflava -- I mean, isoflavones -- and

6 soy protein? I mean, there is like all these things

7 that, because there has been one study or something

8 that are now added, and I think it's a real concern of

9 mine, because we don't really know some of the safety

10 of these -- especially for different populations. Is

11 that going to be -- Sorry, I keep adding stuff, but is

12 that in the food safety, or would it be in the

13 nutritional adequacy? It seems like it's a food safety

14 question, but.

15 DR. CLEMENS: It could very well be, because we

16 don't have an RDA or DRI for these things --

17 DR. NELSON: Right.

18 DR. CLEMENS: -- so they will fall into these

19 functional food components, if you will, the bioactives

20 that everyone has spoken about, so maybe food safety is

21 to examine some of the bioactives that are really for

22 which most of the consumers are concerned. For

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1 example, we know that the American Heart Association
2 pulled back their support for soy earlier this year, if
3 I recall correctly, in March, if I remember right. So,
4 they said, do we change some of that? Do we want to
5 educate the consumer on behalf of the latest
6 information? Maybe this is an opportunity for us to
7 examine it. Good point.

8 DR. NELSON: Right.

9 DR. VAN HORN: Yeah. I think that's an excellent
10 idea and to move it ahead. Robert?

11 DR. POST: If I could suggest too, to consider
12 food safety then has a couple of perspectives and
13 perhaps it's evolved to be more than just microbes, and
14 you are talking about food components that might very
15 well have adverse health affects and that will cause
16 sensitivities, and that includes allergens.

17 DR. CLEMENS: Yes.

18 DR. POST: So that might do it. Those might be
19 areas to consider, and even broadening the view of what
20 food safety involves.

21 DR. NELSON: And genetically-engineered foods now
22 that we have the potential of purple tomatoes.

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1 DR. POST: Well, internationally food allergens is
2 a very big issue and now there are icons, it may be
3 another way to educate consumers after we had a
4 regulation in 19 -- 2006, if I remember right. So if
5 we include allergens in cross-contamination again with
6 that particular area is going to be important.

7 DR. VAN HORN: All right. Well, I want to thank
8 the committee. And we have now come to the point where
9 we need to express our consensus about whether we
10 believe that this Dietary Guideline review should
11 continue, and if there is enough evidence that we think
12 we need to pursue a new set of guidelines. Is there a
13 proposal, I guess, a motion from the group? Is that
14 what we want?

15 DR. CLEMENS: We move to we need a new set of
16 guidelines.

17 DR. VAN HORN: A new set of guidelines. Is there
18 a second?

19 DR. PEARSON: Second.

20 DR. VAN HORN: Okay. Any discussion? Does anyone
21 have any last minute comments on anything that we have
22 said today?

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1 (No audible response).

2 DR. VAN HORN: Okay. Well, with that I guess
3 we'll just do it the old-fashioned way. All those in
4 favor, just raise your hand.

5 DR. VAN HORN: All right. Very good. It looks
6 like it's unanimous, we believe we should go forward
7 with a new set of guidelines, and we will do our best
8 to be up to the challenge. I'm sure I speak for
9 everyone here. I guess our next meeting will be
10 sometime in January. We have just been talking about
11 that, and while the official dates have not yet been
12 expressed, that will be coming up. It will be toward
13 the end of January, we think about the 28 or 29, but
14 those plans are still underway. And I guess with that
15 I just want to thank everyone and --

16 DR. PEARSON: Subcommittees.

17 DR. VAN HORN: -- subcommittee discussion we will
18 present at this point, or just who the subcommittees
19 are. I guess the audience is not aware of who they
20 are. So, pretty much it has to do with the same group
21 that was presenting during these last two days; the
22 Chair of the Energy Balance and Weight Management Group

1 is Dr. Pi-Sunyer, with Drs. Nelson, Williams, Perez-
2 Escamilla, Slavin and Achterberg on that one; Food
3 Safety and Technology is Dr. Clemens and Dr. Perez-
4 Escamilla; Fluid and Electrolytes, the Chair is Dr. Dr.
5 Apple, with Drs. Williams, Pearson and Nichols-
6 Richardson.

7 DR. NICHOLS-RICHARDSON: Can I make a request?

8 DR. VAN HORN: Sure. To come off of fluid and
9 electrolytes since there will be quite a bit in
10 nutrient adequacy, if that's -- if there is no
11 objection?

12 DR. VAN HORN: Sure. Okay. Okay. That sounds
13 good.

14 DR. NICHOLS-RICHARDSON: Okay. Thanks.

15 DR. VAN HORN: We will remove you from that one.
16 Ethanol is Dr. Rimm with Drs. Appel and Pearson;
17 Nutrient Adequacy, the Chair is Dr. Nichols-Richardson
18 with Drs. Fukagawa, Achterberg, Slavin and Nelson;
19 Fatty Acids, the Chair is Dr. Pearson, with Drs. Rimm
20 and Clemens on that; Carbohydrates, the Chair is Dr.
21 Slavin with contributors, Drs. Achterberg, Pi-Sunyer,
22 and I'll help out a little on that one; and then our

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1 Science Review Committee will be myself with Drs.
2 Fukagawa, Appel and Pi-Sunyer. And that represents the
3 subcommittees that we will be convening in between now
4 and the next meeting, and we look forward to launching
5 in those directions and --

6 DR. NELSON: Can I comment?

7 DR. VAN HORN: Yes.

8 DR. NELSON: I still want to put a plug in for
9 consideration of adding a subcommittee on behaviors and
10 food patterns. And we might consider -- we might just
11 wait until the next meeting to discuss that. We have
12 time, but.

13 DR. VAN HORN: Right. I think that's a good
14 point; however, I also feel that it is the charge of
15 every one of these subcommittees to take on a
16 behavioral component, because it seems to me, and I
17 think we discussed this earlier, that each one of them
18 has some aspect of translational effort related to it,
19 and therefore, it should be addressed in each of the
20 subcommittees as well. Anything else?

21 (No audible response).

22 DR. VAN HORN: All right. Well, I guess we are

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1 adjourned. Thank you very much. Have safe travel.

2 (Whereupon, at 2:23 p.m. the hearing concluded).

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