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DIETARY GUIDELINES)
ADVISORY COMMITTEE MEETING)

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P R O C E E D I N G S

(9:30 a.m.)

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3 CHAIRMAN GARZA: I have to congratulate this
4 group. It is one of the most orderly ones I've ever had the
5 pleasure of chairing. People start going to their chairs on
6 the first notice.

7 (Laughter.)

8 Good morning. Very pleased to see so many of you
9 return despite the weather forecast, which for Washington, I
10 know, strikes terror and horror in everyone's heart. It
11 reminds me a little bit of Houston where two snowflakes and
12 people run for cover. So I've adjusted, you can tell, from
13 after 10 years. Now, 12 inches and it looks normal to me.

14 Okay, we have the pleasure this morning of having
15 Dr. Tim Byers, Professor of Preventive Medicine, University
16 of Colorado School of Medicine. We've asked Professor Byers
17 to come and review with us the links between fat fiber and
18 carbohydrate intake. That was the original title. Then I
19 called Tim back about a little bit over an week ago and
20 said, "Tim, can you add alcohol to that, and help us look at
21 the links between those four and the risk of cancer?" And
22 he very graciously agreed.

23 And so with that very brief introduction,
24 Professor Byers.

25 DR. BYERS: Thank you. And you also said "add a
26 little sugar."

27 (Laughter.)

28 So got a little added sugar to the presentation as
29 well.

1 So this is my assignment really, to review
2 findings just in the last four years relating cancer risk to
3 these factors. It is snowing outside, I notice. I'll keep
4 you all informed because I do have a little bit of a view of
5 the outside from here.

6 And to comment as well, I was asked to comment as
7 well on my views about food, the implications for food
8 policy, which is what you all are about, and research
9 approaches, so I'll try to do that.

10 My approach to looking at these four factors was
11 sort of the usual thing, look at publications largely in the
12 English literature in the past four years. My presentation,
13 however, will not be intended to be comprehensive in the 20
14 minutes with these four topics, and I'm going to focus
15 instead on two things: the larger pool studies, or read
16 analyses, and focus on specific controversial areas. So
17 we're sort of more of the same, I'll just say that, and
18 focus really on what I think are the more cutting edge
19 issues.

20 Prior to 1995, there were a few studies about
21 dietary fat and prostate cancer. The emphasis was pretty, I
22 think, weak and inconsistent, and I think since 1995, it's
23 more of the same. I think the range of kinds of findings
24 for prostate cancer is essentially unchanged; that is, that
25 there is a lot of heterogen in either cross studies, there
26 are some very good studies, including one published just a
27 month or two ago indicating fats, in particular, saturated
28 fats, are a risk factor, or a review written by Larry
29 Colonel, published just this last month, I think, covers the

1 topic well and makes a case that there are a range of
2 findings. And if there are relationships with fats, there
3 is probably more evidence for animal sources or saturated
4 fats.

5 The other thing that I think is important is that
6 there is continued to be evidence that there are effects of
7 fats, especially saturated fats, on circulating androgens
8 and perhaps androgen conversion in tissues, and I think that
9 that's an important continuing development.

10 As far as colorectal cancer before '95, there were
11 fairly consistent findings for associations with total
12 and/or saturated fats. Most of the research at that point
13 came from case control studies. Since 1995, the
14 associations have generally been weaker from the larger
15 prospective studies and it's pretty clear that various
16 methods of caloric adjustment in epidemiology largely adjust
17 away fat effects for colorectal cancer.

18 But this is problematic really because the kind of
19 factors we adjust for are not only total calories but also
20 relevant factors for colon cancer risk, including fruits and
21 vegetables, physical activity and red meats, which are
22 themselves associated with fat and saturated fat intake,
23 really, I think, create problems with multi-variant
24 adjustments such that it's really difficult given our
25 current epidemiologic technique to be certain that we're not
26 overly adjusting some of these models, and the new issues, I
27 think, will be screening. In the past, there has been very
28 little screening activity for colorectal cancer. In the
29 future, as we increase screening in this country, it's

1 likely that those people who are screened for colon cancer
2 will also be on more hard, healthy diets, so that will be a
3 new compounder in future studies, I think.

4 With regard to fats and breast cancer before 1995,
5 the evidence was pretty inconsistent and really weak, and
6 came from case control studies. In the last four years the
7 evidence has been more consistent and more consistently now,
8 and the larger cohort studies really are null for the
9 dietary fats/ breast cancer hypothesis.

10 I think there is a new -- as we scramble to try to
11 resurrect the hypothesis, there is a new area of confusion
12 that I want to comment on, and that is some confusion about
13 different types of fats.

14 These are the findings from the pooled analysis of
15 seven large cohort studies conducted around the world, and
16 these four were total, saturated, poly and mono, and I
17 didn't label them because it doesn't matter. As you can
18 see, they are all the same. And essentially across a fairly
19 wide range of dietary fat intake findings are null for
20 breast cancer risk.

21 And this range really does, I think, even
22 accounting for measurement error, really does include the
23 range from about between 25 or 30 percent of calories from
24 fat, in the high thirties, 35 to 40 percent of calories from
25 fat, so across the range of intakes that are typical in the
26 diet, but probably not below 25 percent. We can conclude
27 that there is not much for relationship between dietary fat
28 and breast cancer. Of course, the women's health initiative
29 is an experimental study to try to test the effect, possible

1 effectiveness of lower levels.

2 I wanted to comment though on a new problem, which
3 is an analytic problem, I think, and that is, as we try to
4 separate out the effects of different kinds of fats using
5 multi-variant models, there are particular problems that
6 emerge. I think as an example here is a case control study
7 within a cohort in Sweden published just last year in which
8 these are the relationships between saturated, mono and
9 polyunsaturated fats, and across, again, a fairly wide range
10 of intakes, really not much evidence of a relationship. But
11 in multi-variant models in which one type of fat is adjusted
12 for the other, then these fats can separate, and I think
13 that some of this separation of, in this case, mono fats
14 came to be protective, and saturated and polies looking to
15 be slightly risk factors is an unfortunate artifact of a
16 statistical colony. This is before adjustment, this is
17 after, and the headlines from this were that olive oil or
18 monofats protect against breast cancer.

19 I think, as we scramble and try hard to squeeze
20 effects out of fats using multi-variant models, we're going
21 to create some artifacts that I think are, unfortunately,
22 probably not a reflection of true biology.

23 DR. DWYER: All of those risks are below 1.4,
24 right?

25 DR. BYERS: All of the risks are below 1.4.

26 Well, the model fat was a protective relationship
27 that was marginally statistically significant.

28 The increase in risk for saturated and polies in
29 that study was not statistically significant, and it was

1 certainly below 1.4 as an observation.

2 Alcohol and breast cancer before '95, they were
3 fairly consistent, but weak associations. There were a lot
4 of questions though at that time about not only beverage
5 specificity for the effect, but dose response relationship,
6 biological mechanisms and latency. By latency, I mean at
7 what point in life might alcohol really be relevant. Is it
8 in later adulthood? Is it teenage years or whatever?

9 Subsequent to '95, the findings have really been
10 more consistent, and I think we now have some good early
11 answers to all the above questions that I want to comment
12 on. But the new questions and the questions that policy
13 panels such as you have to deal with are the trade-offs for
14 heart disease. So I'll comment on all those things.

15 Here is a pooled analysis from the same seven
16 large cohort studies showed before for dietary fats,
17 indicating that above -- this would be 15 grams or so would
18 be about a drink a day, and certainly above a drink or two a
19 day you start to see a linear increase in risk for breast
20 cancer.

21 The question of latency, I think, has been
22 addressed nicely in three studies that have been published
23 in the last two - three years, and they all agree that
24 alcohol intake -- it appears that alcohol intake later in
25 life is more relevant to breast cancer risk than alcohol
26 intake early in life. One of the questions was, was maybe
27 this weak, inconsistent relationship with alcohol might be
28 sort of a residual effect of heavier drinking earlier in
29 life, and it looks like that probably is not the case; that

1 alcohol, in fact, does increase breast cancer risk --
2 because I believe it does -- then the effect appears to be
3 more proximal to the breast cancer itself.

4 There are a couple of studies of alcohol and stage
5 of breast cancer, and both studies agree really that alcohol
6 drinkers, women who drink alcohol tend to present at later
7 stages of breast cancer. The explanations for that aren't
8 clear cause there is really two possibilities. One is that
9 there is a biological effect of promotion by alcohol and
10 breast cancer. I think that is plausible by affecting
11 estrogens. The other possibility that needs to be teased
12 out is that there may in fact be diagnostic delay, and that
13 women who drink alcohol may be less attentive to breast
14 cancer in terms of screening mammography. That should be
15 fairly easy to answer pretty soon, I would think.

16 The real problem is, as you're well aware, is the
17 trade-off. Here is data from two large prospective studies,
18 the Nurses' Health Study and the American Cancer Society --
19 Cancer Prevention Study II. The solid lines from the two
20 studies indicate this U-shape relationship with total
21 mortality driven largely by benefits to cardiovascular
22 health, of drinking one or two drinks a day in this range.
23 The breast cancer risks shown in the dotted lines, and when
24 you get up to one or certainly two drinks a day, then I
25 think there are appreciable breast cancer risks.

26 The trade-off then, I think, has to be one, and
27 the tough thing for you all to consider is that there is not
28 only just gender-specific specificity for possible adverse
29 effects of low drinking alcohol for cancer; that is,

1 affecting women with regard to breast cancer, but also age-
2 specificity as well.

3 Here are the U.S. mortality data showing the
4 breast cancer relationship with age, showing the heart
5 disease relationship with age, and a line that I've added,
6 which is breast cancer mortality times two; two times breast
7 cancer risk is something that a lot of women can fairly
8 easily compute for themselves based on their family
9 histories, other risk factors, previous biopsies or, in
10 fact, previous breast cancer.

11 And for breast cancer, it's actually similar or
12 maybe even a higher cause of death premenstaulpausily than
13 is heart disease. For women at higher risk for breast
14 cancer, the breast cancer times two curve, that cross-over
15 really doesn't happen until about age 60. So the tough
16 thing for you is that it's not only issues about gender-
17 specificity, but also age-specificity as well.

18 Just a couple comment on fiber because I'm pretty
19 much going to dismiss it in my conclusions, and a lot of
20 studies have shown this kind of thing. This is a large
21 study out of Italy of about 2,000 cases, 2,000 controls,
22 showing that fiber from grain sources is unrelated or
23 perhaps actually associated with increased risk, but I think
24 most conservatively judged as unrelated to risk, whereas
25 fruit, and especially vegetable fiber, associated with lower
26 risk.

27 Now, a little sprinkling of sugar. Before '95,
28 there were a few studies. After '95, there are still a few
29 studies. Most of the interest in added sugar and cancer has

1 been with regard to colorectal cancer, but I think really
2 the findings are weak. Even as I look at the papers that
3 ostensibly from their titles and their abstracts report a
4 positive finding with sugar or sucrose and colon cancer, I
5 think the findings are even weak within those analyses. I
6 think there has been a tendency to feature on subgroup
7 findings and the big picture is there's not much of a
8 relationship there.

9 There is problems though with studying added
10 sugars in cancer. The biological hypotheses are pretty
11 compelling that either caloric load or maybe stimulation of
12 insulin might help to drive the promotion of neoplasia, and
13 it's biologically appealing. The problems, of course, in
14 epidemiology is it's difficult to quantify many things in
15 the diet, especially things like added sugar, and that
16 sugar, choice of the sweet foods associated with other
17 aspects of behavior and it may, in fact, be an indicator
18 food. So if you ask somebody how often they eat candy, you
19 can make a pretty good guess about some of their behaviors
20 as well.

21 After reviewing about 100 studies on sugar in the
22 diet, Burley actually published a pair of papers in The
23 European Journal of Cancer Prevention, the most recent one
24 last year, and concluded, after reviewing all of them, it's
25 apparent that there is insufficient evidence to conclude
26 whether sugar has a role in cancer at any site, and I would
27 agree with that even though I didn't go through as many
28 studies as Burley did.

29 So just to conclude my comments, first, with

1 research recommendations. These are obviously in a
2 nutshell. I think, with regard to alcohol, the main -- the
3 effect of alcohol in upper elementary cancers is pretty
4 clear, and those are higher doses. The only real cutting
5 edge question is low dose alcohol and breast cancer, and
6 there I think the research really needs to focus on estrogen
7 effects as it has, but I think there needs to be more work
8 on estrogen effects of alcohol.

9 With regard to fats, I think the only critical
10 question now really is fats, especially saturated fats, in
11 prostate cancer, and there I think we need more studies on
12 the effects of fats on androgens in men.

13 I think with regard to fiber, we just need to get
14 over it and focus on foods and not fiber per se in cancer.

15 And with regard to sugar, I think studying insulin
16 resistance syndrome as related to neoplasia, since that's
17 really the primary method, mode that the hypothesis holds
18 for sugar, is probably the way to go, to better understand
19 insulin and missal-like growth factors as they relate to
20 neoplasia, and then to back up from that and make inferences
21 about sugars and starches.

22 So since 1995, there have been two important
23 dietary guidelines issued apart from the ones that your
24 predecessor panel did in 1995. The American Cancer Society
25 a year later, in '96, and The World Cancer Research Fund, in
26 conjunction with the American Institute for Cancer Research
27 a year later, in 1997, issued dietary prevention, or cancer
28 prevention guidelines, and they really overlapped
29 substantially, and the substantial areas of agreement

1 between these two guidelines are just summarized here: Eat
2 more plants, eat less animals, avoid obesity, be physically
3 active, drink little, if any, alcohol. Perhaps you just
4 want to take this exact wording for your next guidelines.

5 Food policy implications of the finding in the
6 last four years, I've summarized here. For alcohol, I think
7 the policy challenge for you all is that I think the best
8 evidence now is that there does need to be some gender and
9 age specificity now in alcohol guidelines, how you translate
10 that into words or how we translate it into bumper stickers
11 or other messages for the U.S. population is indeed a
12 challenge, but I think that science dictates this.

13 For fats, I think the types of fats to avoid to
14 lower cancer risk, either for colon or prostate cancer,
15 where I think there are still some questions especially
16 about saturated fats are, fortunately, consistent with heart
17 disease prevention recommendations. So I see no problem or
18 no conflict here. I think cancer can take a back seat to
19 heart disease when it comes to types of fats, so I think
20 that's appropriate, just as in the past.

21 With regard to fiber, I think we should really
22 avoid the term in recommendations and focus instead on the
23 foods that contain fiber and, I think, other nutritional
24 aspects of those foods that are more relevant for cancer
25 anyhow.

26 And for sugar, I see really no need to add cancer
27 to reasons to limit sugar intake.

28 So those are my comments. I'd be happy to take
29 any questions or comments.

1 CHAIRMAN GARZA: Any questions? Comments?

2 Dr. Meir?

3 DR. STAMPFER: That was really great, Tim. I
4 think that was the best 20-minute diet cancer summary I've
5 ever heard. Just a comment and a question.

6 The comment is I agree with you on the fiber, but
7 just to emphasize that this was for cancer and that there
8 may be other health benefits for fiber besides cancer.

9 My question is, Tim, for the breast cancer what's
10 your -- the current guideline is one drink per day for
11 women. That's the current dietary guideline, and what's
12 your take on the level of risk for breast cancer at that
13 level of consumption?

14 DR. BYERS: The med analysis indicates that at
15 precisely one drink a day the risk is very small; maybe 10
16 percent range. But within that -- I said "med analysis" --
17 within that pool of analysis of seven studies, as you know
18 since you're a co-author of it, is heterogeneter cross
19 studies to where some studies indicate higher and some
20 lower.

21 So at exactly one drink a day, I think, I think
22 there is some elevation of breast cancer risk but it's
23 probably of that order.

24 CHAIRMAN GARZA: Dr. Grundy.

25 DR. GRUNDY: Obviously, there are people who
26 believe very strongly about diet and cancer, and it was, you
27 know, believed so strongly that they initiated that major
28 study and millions of dollars are invested. What was the
29 scientific data or has that changed?

1 DR. BYERS: Which study are you referring to?

2 DR. GRUNDY: Well, I'm taking about like the
3 women's health study where they are going to have a low fat
4 diet and obviously that was -- to mount a study like that
5 there has to be a lot of presumptive evidence and cross-
6 culture and all that.

7 DR. BYERS: Well, I think the best summary is --
8 Roy provided by the National Cancer Institute itself, the
9 rationale for it. There has been repeated papers. Most
10 recently, Peter Greenwall restated the rationale just a
11 month or so ago. I think it's in JNCI or one of the
12 national journals.

13 I think the rationale for that experiment is that
14 you need to get to lower levels. There may be a threshold
15 below which there is an effect, that's it hard to really
16 measure that in observational studies. I wouldn't want to
17 argue strongly for the rationale, but this is sort of water
18 over the dam from a decade ago, and the trial is well
19 underway, and the difficulty in interpretation of the trial
20 is it is not just a low fat trial; it's sort of a total diet
21 trial. But that's good enough for me.

22 I actually think the Women's Health Initiative
23 Study is a reasonable thing to do.

24 DR. GRUNDY: Why is that? I mean, from what you
25 presented, it would have been hard to convince me to invest
26 the money to do that?

27 DR. BYERS: I think if we would have had these
28 data in hand 10 years ago when the debate -- I guess it was
29 10, it seems like 10 years ago when the debate was going on

1 -- that trial may not have gone forward. But at the time I
2 think there was uncertainly, certainly uncertainty in the
3 extent to which we should believe observational
4 epidemiology, uncertainty that there may have been a lower
5 threshold.

6 My guess is that the trial is not going to be
7 particularly positive, but nonetheless I think it's -- I
8 thought when the decision was being made that it was a
9 reasonable trial to do.

10 DR. GRUNDY: The idea of thresholds and systems
11 like this is very problematic, isn't it; a threshold concept
12 is questionable?

13 CHAIRMAN GARZA: Dr. Lichtenstein.

14 DR. LICHTENSTEIN: A very informative
15 presentation. You said that there had been some work done
16 on the relationship between alcohol intake and breast cancer
17 as a function of, I guess, age of women, age of diagnosis of
18 the breast cancer.

19 I'm just wondering, has there any work been done
20 on tumors that are estrogen-sensitive to estrogen-
21 nonsensitive and any alcohol intake?

22 DR. BYERS: Yeah, one would think that the alcohol
23 effects would be more specific for the estrogen-receptive
24 for positive tumors.

25 I'm sure somebody has looked at it. Meir, do you
26 know the estrogen on that? I'm not sure what the
27 literature -- what research has been done on that, to tell
28 you the truth.

29 DR. STAMPFER: There is no big distinction that's

1 been noted so far.

2 DR. BYERS: You mean it hasn't been studied or
3 there is no difference?

4 DR. STAMPFER: No, it has been studied in -- it
5 has been studied, although not thoroughly, but the studies
6 that have been done don't show a difference.

7 DR. LICHTENSTEIN: So then that would argue
8 against the relationship between alcohol and estrogen or
9 estrogen metabolism?

10 DR. STAMPFER: No, not necessarily, because
11 estrogen sensitivity changes in the course of tumor
12 progression.

13 DR. BYERS: So there may be effects on a tumor
14 that as it becomes apparent looks to be estrogen receptive
15 negative.

16 CHAIRMAN GARZA: Dr. Dwyer?

17 DR. DWYER: Just a quick question, Tim, on the
18 alcohol breast cancer. Has anyone seen any relationships
19 between hormone replacement therapy and the effect or lack
20 of effect of alcohol?

21 DR. BYERS: I think that was included in the
22 pooled analysis, HRT, and the report I'm understanding in
23 general is that there was not much difference across a
24 number of other risk factors.

25 Was HRT one of those?

26 DR. STAMPFER: They're independent. They both
27 raise risk, but there is no significant interaction. So
28 women who drink and take HRT have a higher risk than women
29 who drink and don't take HRT.

1 CHAIRMAN GARZA: Dr. Johnson.

2 DR. JOHNSON: Thanks, Tim.

3 I was curious about what you said about added
4 sugar because I know in reading the World Cancer Research
5 Fund and American Institute for Cancer Research, that book
6 that came out, that they came to the conclusion in that
7 section on colorectal cancer that sugar, particularly
8 sucrose, was associated with colorectal cancer.

9 DR. BYERS: I think they gave it some sort of
10 guarded category.

11 DR. JOHNSON: Yeah. And, in fact, among the
12 recommendations one of the -- in the end, when they give
13 their recommendations, one of them is to limit consumption
14 of refined sugar, and I'm going to show that in a minute.
15 So I'm just curious if you could comment that you've clearly
16 come to a different conclusion that that group did about
17 sugar.

18 DR. BYERS: Yes. As I look at the studies,
19 especially the bigger, stronger studies that ostensibly have
20 themselves concluded that there is a relationship, it seems
21 to be just within subgroups, so you get an effect in young
22 men, but not older men, and no effect in women and so forth.
23 And so I think if there is a relationship, it's pretty weak.

24 Now, the weakness of that relationship may be a
25 function of the difficulties I listed as how to measure
26 sugar and analyze it and so forth, but that's my own take.
27 I would be interested to hear yours.

28 DR. JOHNSON: Well, do you think some of -- do you
29 think it's possible that it's a displacement of the fact of

1 fewer fruits and vegetables in the diets, or was that well
2 controlled in those?

3 You know, I'm wondering is it the sugar or --

4 DR. BYERS: It was controlled. It was well
5 controlled. I don't know, to the extent which we can
6 measure things, I don't know.

7 CHAIRMAN GARZA: Dr. Kumanyika.

8 DR. KUMANYIKA: Tim, do you know if there has been
9 any dietary pattern analysis on any of these cancers?
10 Because since obviously these are all related, has anyone
11 done any of the scoring, index --

12 DR. BYERS: Yeah, there have been some, and some
13 attempt at sort of cluster or factor analysis, and you can
14 come up with clusters or factors, and you can attach names
15 to them, but just what they mean, I mean, it takes somebody
16 smarter than me to figure out what those clusters really
17 represent.

18 There have also been some other analyses looking
19 at patterns, but you get sort of a predicable thing like
20 with fruits and vegetables especially. So I think that the
21 food pattern approach and analysis that's been done to date
22 has not really added to the field very much.

23 CHAIRMAN GARZA: Dr. Weinsier.

24 DR. WEINSIER: I realize that you're charted to
25 not include the relationship of physical activity, but you
26 mentioned AICR's recommendation to be more physically
27 active, and, in fact, it's part of our charge to consider in
28 the weight guidelines.

29 Do you know of any evidence outside of the

1 relationship of physical activity to obesity and then to
2 cancer, particularly breast cancer, do you know of any
3 direct evidence of physical activity and cancer?

4 DR. BYERS: Yeah, I think there is pretty good
5 evidence for colorectal cancer. Just what the mechanism --
6 presumably it's got something to do with gut mortality, but
7 I think it's pretty clear that there is an independent
8 protective effect of being physically active for colon
9 cancer that's independent of body weight.

10 For breast and prostate cancer, the other two
11 cancers where there has been thought to be a relationship, I
12 think it's either not there or very much weaker than colon
13 cancer.

14 CHAIRMAN GARZA: Dr. Dwyer, did you have your hand
15 up or not?

16 DR. DWYER: No.

17 CHAIRMAN GARZA: Any other questions?

18 Tim, there is one other one, and it wasn't in your
19 charge either but perhaps you ran across the whole issue of
20 selenium and prostate cancer.

21 How strong is that relationship, and obviously it
22 has some implications then for how one balances between
23 animal and non-animal food given the sources of selenium?
24 Any observations you want to share with the group?

25 DR. BYERS: Yeah, but let me stop with your
26 implications first, because really the selenium in animal
27 comes largely from the grain that it consumes, so that
28 varies depending on grain sources. So the ultimate source
29 really, or the source really is not the animal; it's just of

1 the vehicle.

2 I think the possible effect of selenium on cancer
3 is one of the most exciting things that's happened in the
4 last decade in cancer research, but it still is possible and
5 needs to be confirmed. I think if subsequent trials
6 indicates even half the benefit of selenium, as Larry
7 Clark's found in the secondary data points in his trial,
8 then I think that's the biggest finding since tobacco and
9 cancer.

10 So are we excited about it? I'm very much
11 enthusiastic about new trials that are going to be underway,
12 and I've got my fingers crossed.

13 CHAIRMAN GARZA: All right. I just want to add a
14 work on the selenium, a cautionary note, that in the Larry
15 Clark trial, the benefit was almost instantaneous, which
16 goes against what we think we know about how cancer works,
17 and there is some suggesting that the rates may have been
18 higher in the placebo group.

19 But another point of interest is that Finland,
20 which is the lowest selenium country based on their early
21 studies, decided to fortify and raise their selenium levels
22 quite dramatically 10 years ago, and there hasn't been one
23 iota of suggestion that their prostate cancer rates have
24 decreased.

25 So, yeah, I'm hopeful too, and it would be great
26 is the Larry Clark data were replicated, but I think we need
27 to be cautious.

28 DR. BYERS: Yeah, just a couple comments. One is
29 that the adverse effects of beta carotene in the two large

1 trials that indicated we could increase lung cancer risk by
2 20 percent or more with beta carotene also occurred much
3 sooner than people would have believed. So it's quite
4 possible that there are some late stage effects of nutrients
5 on cancer that we don't yet understand the biology of.

6 The other aspect of Finland is very interesting,
7 and that is that their lung cancer rates began to dip before
8 the rest of Europe, and so I think the jury is out on
9 selenium.

10 CHAIRMAN GARZA: Okay. If there are no other
11 questions or comments, thank you very much for an excellent
12 presentation. I have to echo Dr. Meir. That was possibly
13 the best 20-minute summary that I've heard. Thank you.

14 For those of you who missed the meeting yesterday,
15 we did not get to grain products, vegetables and fruits
16 despite heroic efforts on everybody's part, so we're going
17 to go back to that portion of yesterday's agenda and take it
18 up from there.

19 DR. DECKELBAUM: Okay, I'm Richard Deckelbaum, and
20 I guess what happened last night is that grain, fruits and
21 vegetables got displaced by going out and figuring whether
22 we should have one moderate or two moderate inputs.

23 So first slide, please. Can we lower that a bit
24 because most titles will be missing then. That's fine.

25 CHAIRMAN GARZA: While Dr. Deckelbaum is getting
26 ready, I want to welcome Dr. Shirley Watkins, who has just
27 joined us, under-secretary for the Food and Nutrition
28 Service. Thank you so much for coming. We're trying to get
29 her at the table and she refuses, but maybe publicly we can

1 coerce her. I don't know.

2 DR. DECKELBAUM: So the grains, vegetable and
3 fruits working group was consisted of myself, Alice
4 Lichtenstein and Meir Stampfer, but we really had very
5 substantial help and input from USDA and other staff,
6 including Elta Salton, Shanthy Bowman, Andrea Lindsey, and
7 Kathryn McMurry, Carol Davis, and they gave us a lot of
8 materials and lot of substantive advice in formulating some
9 of the things you are going to hear ago. And as well, I'd
10 like to thank Carol Suitor and Suzanne Murphy and Burt
11 Garza, who also took place in our meetings and had a role in
12 what you're about to see.

13 Next slide, please.

14 So the 1995 guideline here is at the top of this
15 slide, and the three major sections that were discussed in
16 the previous guidelines are listed here underneath. Our
17 charge was really to review the science base and add to it,
18 focusing on literature since the previous report. If
19 supported by new evidence, we were asked to make appropriate
20 revisions, and as well, we looked at modalities to suggest
21 approaches for better implementation of the content of the
22 grain, fruits and vegetables guidelines.

23 Next slide.

24 So these are the options that we mainly focused on
25 during our deliberations. We asked if there should be an
26 increased emphasis on whole grains in the guideline itself
27 and/or in the text. Should there be clearer definition of
28 different types of carbohydrates?

29 Are carbohydrates in potatoes as good as

1 carbohydrates in broccoli? And we also had discussions
2 related to the potential role of the glycemic index in
3 choosing carbohydrates, and Dr. Stampfer will be talking
4 more on this in a few minutes.

5 Should there be more emphasis on quality versus
6 the quantity of grains, vegetables and fruits ingested, and
7 that's related to what I just said? But this could also
8 include should we better point out grains, fruits and
9 vegetables that are rich in certain macra nutrients, say
10 fiber or micro nutrients, certain antioxidant vitamins?

11 Should we have an increased emphasis on not
12 ingestion, and, again, Dr. Stampfer will be addressing that?
13 And we also reviewed potential ways for clearer
14 implementation guidance for grains, fruits and vegetables,
15 and along these lines Dr. Lichtenstein will be giving a
16 short talk as to the question should the grain guideline be
17 separated from vegetable and fruits.

18 Next slide.

19 I'm not going to spend much time on this because
20 the good news is that since 1995 there's an increasing body
21 of literature showing beneficial effects of fruits and
22 vegetables in decreasing cancer, cardiovascular disease,
23 cataracts, diverticular disease and likely Type 2 diabetes,
24 and the references for these will be provided in our updated
25 report.

26 Next slide.

27 So I'd like to turn now to the question whether
28 there should be increased emphasis on whole grain products
29 and look at some evidence relating to coronary heart disease

1 risk, cancer risk and Type 2 diabetes and insulin
2 resistance.

3 Next slide. This is where slides didn't -- got
4 messed up.

5 This is a meta-analysis study of Dr. Jacobs and
6 his group, "Whole Grain Intake May Reduce the Risk of
7 Ischemia Heart Disease, Death and Post-Menopausal Women,"
8 the Iowa Women's Health Study. So I will be focusing on the
9 first few slide actually on coronary heart disease.

10 They studied almost 35,000 post-menopausal women.
11 The relative risk was about .6 confidence integrals shown
12 for the top versus lowest quintile of whole grain intake,
13 and this was not explained when adjustments were made for
14 fiber, Vitamin E and Folic, suggesting that whole grain
15 intake is protective for ischemic heart disease.

16 Next slide.

17 The next two slides have been borrowed from Dr.
18 Stampfer. This is unpublished data from the Nurses' Health
19 Study, looking at whole grain foods and the risk of coronary
20 heart disease in the Nurses' cohort, and looking at exposure
21 of at least five to six servings per week. You can see that
22 cereals, bran and brown rice, all markedly decrease the
23 relative risk of coronary heart disease in women, but I
24 don't know if this is good news or bad news, popcorn had no
25 effect.

26 (Laughter.)

27 Next slide.

28 What about men? These are two studies that
29 include men, Rimm paper published in JAMA is on the health

1 professionals study; Pietinen study where it was the ATB
2 study where the primary end point was cancer, but they
3 looked at coronary heart disease and found that there was
4 substantial decreases in risk associated with whole grain
5 intake. Now, this is in addition to the effects that whole
6 grains could have on improve lipoprotein profiles and
7 separate, and along those lines I'd like to point out that
8 the effects of carbohydrates, for example, and HDL
9 cholesterol may not be -- may not be applicable to all types
10 of carbohydrates.

11 We published a paper, Tom Stark and our group,
12 last June in the American Journal of Clinical Nutrition,
13 showing that in hypercholesterol anemic children when they
14 went on to fat low, cholesterol lowering diets that HDL
15 decreased only when simple sugar increased but not when
16 complex carbohydrate intake was increased.

17 But, in general, the papers also seem to indicate
18 that risk reduction is associated with higher levels of
19 whole grain intake, and they cannot entirely be explained by
20 adjustments for fiber intake.

21 Next slide.

22 This is another slide borrowed from Dr. Stampfer
23 which shows that the relative risk for coronary heart
24 disease is decreased in general across a number of studies
25 looking at fiber consumption and relative risk for coronary
26 heart disease. I think the important point here that it
27 looks like whole grains do have an important effect, but we
28 still can't dismiss fiber, as we just heard we might have to
29 do in terms of cancer, in terms of coronary heart disease

1 risk.

2 Next slide.

3 Now, what about whole grain intake and cancer, and
4 I thought I was showing this slide earlier but it came up
5 now? This is the study of -- another study of Jacobs, et
6 al., looking at meta-analysis of 40 case controls studies
7 between 1984 and 1997, looking at a variety of cancers, 20
8 in all, and colonic polyps, and, again, there is a lot of
9 data in this paper and I'd be interested in Dr. Byers'
10 comments on them.

11 But the pooled odds ratio, looking at the entire
12 cohort, was about 0.66 for high versus low whole grain
13 intake. And again, this was maintained in general through
14 most cancers. Of interest, breast and prostate had lower
15 correlations with whole grain intake in terms of reduced
16 risk after -- this was maintained after adjustments for
17 social-economic status, age, sex, BMI and other things
18 listed here and not listed on the slide.

19 Next slide.

20 An interesting paper published by Chanteoud in the
21 International Journal of Cancer looked at 10,000 cases and
22 8,000 control cases of hospitalized cases -- hospitalized
23 patients, mainly in Italy, with different kinds of cancer,
24 and they reported risk ratios, again, with high whole grain
25 food intake reduced by substantial amounts for GI tract
26 cancer, bladder and kidney, lymphomas and myelomas, but not
27 for breast cancer. And again, these were maintained after
28 adjustments for a number of potential other variables, other
29 confounders.

1 Next slide.

2 Briefly, I'm just going to show an overhead. Here
3 on whole grain intake in non-insulin dependent diabetes, two
4 papers by Salmeron and Group. The top one, "Diabetes are,"
5 and the bottom one -- sorry, the top one is "Diabetes Care
6 On Men." The bottom one is in JAMA on women, large cohorts
7 again, the Nurses' Health Study and The Health Professional
8 Study.

9 And the bottom line here is that glycemic index,
10 they suggested foods that have a high glycemic index are
11 associated with about a 1.4 to 1.5 increased risk of non-
12 insulin dependent diabetes, and, again, whole grains are
13 associated with a decreased risk, about .7 relative risk for
14 non-insulin dependent diabetes.

15 Next slide.

16 So what are some of the issues that could be
17 involved in considering adding whole to the guideline itself
18 or increasing its emphasis in the text going with the
19 guideline?

20 Well, there could be implications that grains that
21 are not whole are not part of a healthy diet. That's
22 actually related to something a little lower on the slide
23 that we don't want to label necessary good versus bad food
24 or should we.

25 Could there be a cost factor involved which might
26 affect certain classes, lower SES classes who might not be
27 able to afford some of the good whole grain products?

28 Will there be decreased intake of enriched and
29 fortified foods and how would this affect, especially micro

1 nutrient intake, and actually in some analysis we did we
2 really didn't see any effect on this that we could sort of
3 tease out relating to folate as well?

4 Would there be a decrease in food choices that
5 could affect certain segments of the population?

6 Might certain important nutrients actually have
7 decreased absorption? For example, might fitates in whole
8 grains affect iron and zinc absorption? And again, there is
9 really no evidence yet on this, but this is something that
10 certainly would have to be looked at.

11 I mentioned good versus bad foods, and again, is
12 the scientific evidence really strong enough to make these
13 changes because as we've seen in a number of examples of
14 where studies that came out, for example, in case control
15 fashion a number of years ago are not supported by larger
16 cohort studies or clinical intervention studies, so that we
17 still have to consider whether the -- for example, even the
18 papers I showed you are sufficient to allow us to make these
19 substantial changes.

20 Next slide, please.

21 Just a couple of words on our plans, our thoughts
22 towards better guidance provisions in the guidelines, and
23 actually if we can just -- we have to actually raise the
24 bottom of this slide. This is Box 9 from the current
25 recommendations which sort of tells us how to go about
26 getting the diet better with more grains, fruits and
27 vegetables.

28 At the bottom of the box you've got to go to Box
29 2, page 7 for what counts as a serving. So, in other words,

1 it's not a total user friendly, take one look, it's all
2 there type of box.

3 Next slide.

4 So we're going to be looking at as a working group
5 ways to improve the message, and this would be just one
6 example for Box 9 where here would be some of the messages
7 that we might want to include, and paralleling it right next
8 to it would be a "how to" box with different kinds of
9 messages. You know, pack X and X fruit in your purse or in
10 your handbag for lunch or your afternoon snack, et cetera.
11 So these are the kind of things we are considering.

12 I would like to ask Dr. Stampfer to come up and
13 talk about glycemic index and different kinds of
14 carbohydrates.

15 DR. STAMPFER: Okay, thanks. I've been given the
16 job to cover nuts and glycemic index in seven minutes. I'm
17 going to try to stick to that.

18 Nuts are basically recommended against, if you
19 take the dietary guidelines at their face value, because
20 nuts are a high fat food and it says choose foods low in
21 fat. And a lot of people have been doing that, and it's not
22 a good thing I want to tell you about that. Although nuts
23 are a very high fat food, most of the fatty acids are
24 unsaturated and it's a good source of protein and some other
25 good things.

26 Next slide, please.

27 Consequently, as you might expect, nuts have a
28 favorable impact on the lipid profile because of their
29 mostly unsaturated fat content, so with a high walnut diet

1 the LDL/HDL ratio was substantially reduced. This is a very
2 good predictor of risk of heart disease, diet supplement
3 with almonds also lowered LDL substantially, and this is
4 just what you'd expect from what we know about the impact of
5 high unsaturated fats on the end -- monounsaturated fats on
6 the LDL and HDL ratio.

7 Next slide, please.

8 Well, that's all well and good, but what about
9 clinical end points, and I think this is something we need
10 to keep coming back to and not just rely on the influence of
11 diet on intermediate markers, but we need to look at actual
12 disease outcomes, and there has been three published studies
13 so far looking at nuts. All three find substantially
14 reduced risks of coronary heart disease with nut
15 consumption. We're not talking about mega doses here; just
16 a handful of nuts a couple times a week was enough to reduce
17 risk in the range of 30 or so percent, a very big decrease.

18 So I think nuts should not have this astigmatism
19 attached to them, but to the contrary. Their consumption
20 should be promoted.

21 Next slide, please.

22 Okay, that's all I'm going to say about nuts. Now
23 onto glycemic index. It's a complicated concept, and I
24 don't know if I'm going to succeed in getting it across in
25 the short time I have, but I'll try.

26 The basic idea is very simple. Different foods
27 have a different propensity to raise blood sugar following
28 their ingestion, and the glycemic index is a way of
29 quantifying that. Typically, in the traditional sense

1 nutritionists have divided carbohydrate into simple and
2 complex. Simple being mono and diet sacarides, sugars, and
3 complex being everything else. But this is not a
4 physiologic distinction as the glycemic index is. The
5 glycemic index is really based on what happens to real
6 people who eat food, so that they're given various kinds of
7 foods and blood sugar is actually measured, and it goes up
8 sharply with foods that have a high glycemic index, and less
9 so for foods that have a flow glycemic index.

10 Next slide.

11 So this shows, for example, what happens if you
12 have the same caloric intake for three different kinds of
13 carbohydrates, glucose, amylose pectin or amylose based on
14 either the glycemic response -- let's see over here -- so
15 you can see how glucose, blood sugar shoots up very fast
16 with amylose pectin goes up but less so, and with amylose,
17 which is less readily broken down, its much flatter, and
18 consequently the same pattern emerges with insulin, and this
19 has physiologic effects. It's not a good thing for the
20 system to have your glucose and insulin shooting up and down
21 very sharply that way.

22 Next slide, please.

23 There is -- in muscle, this is in animal studies,
24 insulin sensitivity of muscle, glycogen synthesis is
25 impaired in amylose pectin fat rats. That's a type of
26 carbohydrate that has a higher glycemic index than amylose.

27 Next slide, please.

28 And post-meal lipogenesis, also another animal
29 study looking at, again, the different kinds of carbohydrate

1 here. The high glycemic index fed animals had a higher
2 capacity for lipogenesis after a meal. So these are all
3 adverse physiologic outcomes of high GI diet.

4 Next slide, please.

5 Now, it's important to distinguish between
6 glycemic index and glycemic load. The glycemic index is the
7 property of the food. It's the property of how the food can
8 raise blood sugar. Glycemic load is what that food -- is
9 basically taking into account the amount of carbohydrate
10 that is in there, so it's not just the quality of the
11 carbohydrate but the amount. So you can think of it as
12 glycemic index is sort of the nutrient composition of the
13 good whereas the glycemic load is the amount of that
14 nutrient that you get from eating a normal portion size.

15 Now, everything is calculated in terms of percent
16 of white bread. So you can see, for example, carrots have a
17 high glycemic index, 131 percent compared to white bread is
18 100 percent, but there is very little carbohydrate per
19 serving, so it only accounts in a typical diet to one
20 percent of the glycemic load whereas potatoes are similar to
21 white bread, they have more carbohydrate, of course, and
22 they account for eight percent of the glycemic load of a
23 typical diet, and obviously it's going to change, but you
24 can see those differences.

25 Next slide, please.

26 Well, again, what about clinical end points? Does
27 this really matter for real people?

28 Well, these are data from the Nurses' Health Study
29 looking at glycemic load and risk of coronary heart disease,

1 unpublished data from Dr. Sima Lu in our group. And you can
2 see that the high glycemic load in various statistical
3 models, either adjusting for fat or adjusting -- without
4 adjustment for fat, you can see in the best model, which
5 adjusts for fat intake, the highest level of glycemic load
6 is associated with about a doubling of risk of coronary
7 disease, highly statistically significant, and this is
8 taking into account all the other coronary risk factors.

9 Next slide, please.

10 Now, what is contributing to the glycemic load in
11 the Nurses' Health Study diet? The number one contributor
12 is potatoes. And so we looked specifically at potatoes and
13 after adjusting for all the coronary risk factors what one
14 finds in these data, again, is about a doubling in risk with
15 high intake of potatoes. It's not -- the confidence
16 intervals are broad and the trend is of borderline
17 statistical significance, but it's clear that I don't think
18 we can consider potatoes as a health food here.

19 Next slide, please.

20 Finally, you can ask why, if glycemic index and
21 glycemic load is so important, why don't we have an epidemic
22 of coronary disease in China, for example, where white rice
23 makes up a big part of the diet? And that's a fair
24 question.

25 And the answer is that the impact of a high
26 glycemic load diet is mainly among people who are already
27 marginally glucose intolerant; that is, overweight and
28 inactive, and those qualities are uncommon in China,
29 although they are getting more common, but in the U.S. they

1 are very common.

2 So these are data showing the relation between
3 glycemic load in relation to risk of coronary diseases by
4 body mass index. So among people who are lean, glycemic
5 load really doesn't matter very much. Unfortunately, most
6 Americans don't fit into this lean category. Most Americans
7 are here where high glycemic load will double the woman's
8 risk of coronary heart disease.

9 I think I've gone over. I'll stop here.

10 Oh, let me just say one more thing on popcorn.
11 Although it wasn't statistically significant, popcorn did
12 have the same trend as all the other whole grains.

13 DR. DECKELBAUM: Was that Cracker Jack?

14 (Laughter.)

15 DR. STAMPFER: No brand name endorsement.

16 DR. DECKELBAUM: It has corn syrup, and Dr.
17 Lichtenstein will now discuss the possibility or the
18 question to splitting the guideline.

19 DR. LICHTENSTEIN: Okay. Well, as indicated, I
20 would like to suggest that we consider splitting the grains
21 from the fruits and vegetables and having two separate
22 guidelines. One of the primary reasons I think we should
23 consider this is I think we should really think a lot about
24 what we are recommending people should do. We've spent a
25 lot of time in the guidelines recommending what people
26 should not do, and there's been word smiting over the years
27 as far as, you know, consumer diet low in something while
28 consider diet moderate in something, trying to make it sound
29 more positive. But I really think what we need to go is

1 give individuals more guidance on what they should do and
2 what makes a healthy diet.

3 I think if you read the literature on the
4 predictors of grain intake are different from the predictors
5 of fruit and vegetable intake, so this is one reason for
6 splitting them because people view them differently, and I
7 also think the barriers to grain intake are different than
8 the barriers to fruit and vegetable intake, and that's
9 taking into consideration cost, storage, preparation,
10 perishability so that -- and safety also, so that one needs
11 to give different guidance and advice to individuals with
12 respect to fruits and vegetables versus grains.

13 I tried to see if this issue had been addressed
14 directly because we are supposed to propose changes on the
15 basis of scientific -- a scientific basis for proposing
16 changes, but the question has never really been addressed
17 directly.

18 Now, there is an error. The first focus group
19 that I'm going to mention was actually in 1995.

20 But what I did is I looked at the focus groups,
21 and although that specific question had never been posed or
22 considered, we can get certain nuggets of information about
23 it, and when the fruit, vegetable and grain guideline were
24 considered one of the comments was that the suggestion that
25 increasing fruit and vegetables was challenging because of
26 cost, and this speaks to the issue of different barriers to
27 fruit and vegetable intake versus grain intake.

28 There was another focus group conducted in
29 September of 1988, and one of the comments there, again

1 since the question specifically was not posed, was "I like
2 to eat more fresh fruit and vegetables but I can only shop
3 once a week. In two or three days the stuff is no good.
4 The rest of the week is going to have to be canned or
5 frozen."

6 Well, clearly, we didn't get the message across
7 because canned and frozen fruits and vegetables are quite
8 acceptable, and it's not just that someone has to consume
9 fresh fruits and vegetables to get the nutrient value.

10 Another very telling comment was in a focus group
11 that was published in August of 1998, and in this case it
12 had to do with the whole guideline, and the guideline has
13 the word "You should consumer a diet that has plenty of
14 grains, fruits and vegetables." One of the comment was, or
15 on the term "plenty" because it was being equated actually
16 with the "Five-a-Day Program," which is another federal
17 program; that the comments were related to how much is
18 "plenty." Well, it's five and that someone actually
19 mentioned the Five-a-Day Program, so I think that there is a
20 program specifically that focuses on fruits and vegetables.
21 The "plenty" is not a quantitative term, although it's
22 certainly quantitative in the food pyramid which, by the
23 way, also distinguishes between grains, fruits and
24 vegetables. So I think separating the two would be quite
25 consistent with current programs that actually encourage
26 increased consumption of fruit, vegetables, and then grains.
27 Again, just to reiterate, I think the message
28 should really emphasize what people should be doing as
29 opposed to what they shouldn't be doing, and that the

1 message is if fruits and vegetables were separated from
2 grains within each of those categories, I think we could be
3 more focused and clearer in what guidance we're giving for
4 grains, what guidance we're giving for fruits and
5 vegetables.

6 Also, as mentioned, that Box 9 that's actually in
7 the current guidelines is quite big. There is a lot of
8 information, and I think that's where the difficulty lies in
9 actually distinguishing between how to give advice for those
10 different groups, so I think it's something that we should
11 consider.

12 CHAIRMAN GARZA: Thank you very much.

13 Are there any questions of any of the presenters?
14 Roland?

15 DR. WEINSIER: First of all, compliment the group,
16 all three of you, and all the assistance for doing such a
17 great job.

18 Meir, help me with the glycemic index and trying
19 to do something practice or make a practical, safe and
20 reasonable recommendation to the public based upon these
21 findings. If in fact the -- I don't know what you're
22 calling it on the right side of the slide. What is it, "The
23 relative glycemic impact of the diet which takes into
24 account the glycemic index as well as the glycemic load."
25 Potatoes are eight-fold greater impact on the diet than
26 carrots. And I'm trying to envision populations in the
27 world, whether it's China, or Papuans or other, you know,
28 potato/rice eating populations that subsist in these foods,
29 I guess, are healthy.

1 Their glycemic impact factor would be what, 100
2 percent? Ninety percent? You know, it would be
3 extraordinary.

4 So we'd have to argue then, based upon the date
5 you're representing, well, it doesn't have an impact on them
6 because perhaps of their normal weight, and it's only with
7 BMIs that are getting in the higher range with insulin
8 resistance that's having an impact. And I don't know what
9 the answer is, but I'm having trouble from a rational
10 standpoint trying to separate that we would do something
11 different from a dietary standpoint that prevents a disease,
12 a chronic disease, than we do in people who have the chronic
13 disease, i.e., if you're lean, we should have one diet
14 prescription; if you're getting above a certain BMI, we have
15 a different diet prescription.

16 Help me come up with a plan that would make sense.

17 DR. STAMPFER: Okay.

18 DR. WEINSIER: A guideline that makes sense.

19 DR. STAMPFER: It will take a minute or two.

20 First, just let me explain that slide with the
21 eight percent and the one percent. That slide represented
22 different contributors to the total glycemic load in the
23 Nurses' Health Study diet. So of the total glycemic load,
24 eight percent was contributed by potatoes. That's what that
25 eight percent means. It doesn't mean that potatoes are
26 eight times as bad as carrots or something. It just means
27 that the way the diet is distributed of the total glycemic
28 load of the diet, eight percent was contributed by potatoes,
29 which was the number one contributor.

1 Now, the question you raised about populations
2 that seem to do fine, and certainly have very low rates of
3 coronary disease despite a high glycemic load diet, I
4 believe the reason for that is that they have -- do not have
5 by and large the levels of insulin resistance that we have
6 by virtue of physical activity, because muscle decreases
7 insulin resistance and lean body mass, so that the adverse
8 effects of the high glycemic load diet are manifest where
9 there starts to -- starts to be insulin resistance.

10 Now, I wouldn't characterize that as a disease
11 state because if we did, you know, three-quarters of the
12 population in the U.S. would be characterized as diseased.
13 Well, maybe they are. But to the extent that we as a
14 country are fat and slothful in our physical activity
15 patterns, there is a lot of insulin resistance and this is
16 being exacerbated by the high glycemic load diet.

17 Now, how to implement that is another issue
18 because it's kind of complicated to get across in a dietary
19 guidelines, and also I think -- although I think this is an
20 exciting area of research, I don't think the findings are
21 completely proven or conclusive, so we need to decide, you
22 know, if this is ready for prime time.

23 My take on it is that this lends strong support to
24 what our group has been trying to get across, which is an
25 emphasis on whole grains and minimally processed foods, and
26 I think it also lends support to taking potatoes out of the
27 vegetable group and maybe thinking of it as a starchy food
28 group where it might be more appropriate. So those would be
29 a couple ways to implement it.

1 CHAIRMAN GARZA: Related to that, this has come up
2 in previous committees, or at least in one previous
3 committee that I was one, and we, the previous group felt
4 that the concept was difficult to deal with because it was
5 the total diet that contributed the glycemic load --
6 determined the glycemic load, and it was the diet glycemic
7 index that we should be concerned with, and it was very
8 difficult to sort out the dietary pattern from an imbalanced
9 pattern, and the best analogy that I can recall were papers
10 that were published about 10 years ago, warning us that, in
11 fact, children that had very low fat diet were stunted, not
12 realizing that in fact the way they were achieving the low
13 fat intake were by having very low micro nutrient intakes
14 because of the types of foods they were consuming was --
15 again, it was isolating one factor rather than looking at
16 the total diet.

17 The ADA, the work of the Diabetes Association
18 looked at the glycemic index and they found it difficult to
19 deal with.

20 How in the studies that you've looked at do you
21 look at pattern and say, well, maybe it's the pattern we
22 have to be concerned about, to say, well, you know, you
23 don't get to choose one guideline over another, you've got
24 to take them all? And so that if you just take potatoes
25 without variety or you just take this without the other,
26 that in fact you can run into the sorts of problems that
27 you've uncovered.

28 Is that something we need to be concerned about or
29 do you really feel that, gee, we need to focus in on

1 potatoes and other starchy vegetables because they are the
2 culprit and not the pattern?

3 DR. STAMPFER: No, I think -- I think you make a
4 good point. And in terms of the pattern, obviously this is
5 important, but in a sense our guidelines are supposed to be
6 defining a pattern in a way, not just -- not just responding
7 to a pattern.

8 I think the details of getting across the glycemic
9 index concept may be too difficult, but in broad brush
10 strokes I think it's actually not difficult, and that would
11 be an emphasis on whole grain, minimally processed grains;
12 get away from this concept that just because white bread is
13 low fat that therefore it's healthy.

14 And I think the potato issue is just that right
15 now, according to the guidelines, it's considered a
16 vegetable, and if you have a large McDonald's french fries,
17 you've got four out of your a day vegetables according to
18 the guidelines, and I don't think this is right, and I think
19 we ought to consider -- we oughtn't to consider potatoes
20 along with broccoli and carrots and other things that we
21 think of as vegetables.

22 CHAIRMAN GARZA: Okay. Scott and then Johanna.

23 DR. GRUNDY: Ask a follow-up about the glycemic
24 question. It seems like there might be two issues here.

25 One is the total carbohydrate load in the diet,
26 which if you have a very high percentage of carbohydrate in
27 the diet, then the problem, I think, with the American
28 population that you point out, which tends to be sedentary,
29 creates a metabolic stress on the insulin metabolism and so

1 forth. And then the second is that the type of carbohydrate
2 can accentuate that.

3 Is that what you're saying?

4 DR. STAMPFER: Yes.

5 CHAIRMAN GARZA: Johanna.

6 DR. DWYER: Meir, I'm very much interested in the
7 glycemic load concept, but I must admit to ignorance on much
8 of it.

9 First of all, how many foods have glycemic indices
10 experimentally determined? Are there a lot or a few?

11 DR. STAMPFER: A lot. Yeah, there is --

12 DR. DWYER: How many is a lot?

13 DR. STAMPFER: Hundreds.

14 DR. DWYER: Hundreds.

15 And how does the glycemic index of an individual
16 food affect the glycemic index of meals. I thought years
17 ago that it changed depending on the total meal for example,
18 if you mixed all the foods together in a meal.

19 DR. STAMPFER: Yes, that's an important point, and
20 it's one that's still somewhat controversial because some of
21 the initial studies hadn't done the exact correct
22 calculations for looking at the peak and area of glucose.
23 But basically there have been about a dozen studies that
24 have looked at that now. And what one finds is that the
25 glycemic load of a mixed meal is the weighted average of the
26 glycemic load of its -- of the component parts. And, in
27 fact, the correlation between the glycemic load of a mixed
28 meal and the calculated glycemic load based on the component
29 parts is about .987, something like that, so it's very high,

1 so you can predict with a good deal of accuracy what the
2 glycemic load of a mixed meal is if you know what the
3 components are, and it does change, absolutely.

4 DR. DWYER: Just one other -- we just put together
5 an issue, a journal on this topic, and it's very popular in
6 Australia, for example. Australia doesn't have food labels
7 like we do so, you know, it's harder to find out these
8 things. I just wonder if in the future if we're going to
9 consider this it might be possible to get a representative
10 of the Diabetes Association or some other group of
11 endocrinologist who deal with this every day.

12 CHAIRMAN GARZA: I've got three people, Richard,
13 Ellis and Rachel, so I want to assure everyone that I will
14 get to the three of you in just a minute.

15 Richard.

16 DR. DECKELBAUM: I think, you know, in our
17 discussions also on the sugar working group this came up.
18 And, you know, one of the questions we have to look at, we
19 have to look at this also in terms of the whole guidelines
20 because it's -- let's say it's 100 percent right. Let's say
21 it's 100. It's still -- it's very controversial, and there
22 are a lot of people out there who don't -- you know, who
23 were not quite keen on glycemic index, so it may be a
24 gradual process. And one thing I think we'd have to
25 consider with the whole group is -- you know, if you get
26 sort of a big noise about one aspect of the new guidelines,
27 how would that affect the whole report, and so that's
28 something we have to weigh.

29 And I think when we deliberate this afternoon,

1 we'll be bringing this in to, you know, where it should be,
2 if it should be, but where should it be and in what manner
3 because I think we have to make sure that we look at the
4 whole and not, you know, have some kind of topic that may
5 bring up a major controversy which will affect the whole
6 report.

7 We spent a lot of time on this, but I did bring up
8 some other questions, so I'd prefer, if it's okay, not to
9 discuss glycemic index anymore. We can bring it up again
10 tomorrow when we report, but there were some other issues
11 that we looked at as I reviewed, and that included nuts, and
12 that included separation of grains from vegetable and
13 fruits, and I'd like to get some comments from the committee
14 before we break into our working groups this afternoon.

15 DR. JOHNSON: I'll just follow up quickly as part
16 of the sugar group. I did have conversations with Dr.
17 Xavier Pesuniet, Dr. Gerald Reven and Dr. Denny Beer about
18 the glycemic index, and I have all of my notes of those
19 calls with me so I can share some of their opinions.

20 DR. LICHTENSTEIN: I was going to comment on the
21 glycemic index, but what I'll do is first ask my question
22 about nuts -- free speech. But just with nuts, are peanuts
23 included with the nut group?

24 DR. STAMPFER: Yes.

25 DR. LICHTENSTEIN: Okay. Okay, peanuts are a
26 legume and then nuts, most of the other nuts grow on trees.

27 (Laughter.)

28 DR. LICHTENSTEIN: Okay. So is it -- I guess what
29 I'm getting at, is it the nuts themselves or is it the fatty

1 acid pattern that's common to those foods? And then if it's
2 peanuts, is there anything unique that distinguishes peanuts
3 from other legumes? So that are we really talking about
4 nuts in this conglomerate of these two -- I don't know if
5 they're called species or whatever, rely on the food
6 scientists, or is it the fatty acid pattern or some other
7 pattern, the protein pattern, amino acid pattern that's
8 associated with those foods?

9 DR. STAMPFER: Well, if you look at the
10 composition of peanuts, it looks a lot like nuts, and so the
11 common parlance of nuts, peanuts being considered nuts
12 actually makes more sense than the laneon classification of
13 where they all came from. I don't think we should be hung
14 up on the --

15 DR. JOHNSON: Which components of the nuts? I
16 guess, what aspects of the nuts?

17 DR. STAMPFER: In terms of the protein and fatty
18 acid composition of peanuts. They look like other nuts and
19 peanuts, in our study we separated out peanuts from other
20 nuts, and they are both have the similar effect. And you
21 might say, well, what about peanut butter? And peanut
22 butter, if it's just made from peanuts, presumably has the
23 same effect. But a lot of peanut butter has trans added to
24 it, trans fatty acids to keep the fats from separating, so
25 that probably detracts from the benefits.

26 DR. JOHNSON: But I guess that's -- I'm getting at
27 something a little bit different. Is it specifically that
28 people should increase their nut consumption or should they
29 increase their consumption of a diet that's consistent with

1 the fatty acid profile of the nuts as far as advice?

2 DR. STAMPFER: Well, I think -- I don't know. I
3 mean, these are the observations that people who eat nuts
4 have a lower risk of coronary disease, and they also have a
5 better lipid profile. You could get that lipid profile by
6 feeding them oils instead of the nuts.

7 But I think the main message is a simple one, that
8 we should remove the stigma from nuts. Instead of
9 considering them bad, because they are a high fat food, we
10 should consider them according to what their health effects
11 really are.

12 CHAIRMAN GARZA: We have at this rate -- I'll just
13 let the committee know -- we will be leaving tomorrow at
14 about eight p.m.

15 (Simultaneous conversation.)

16 CHAIRMAN GARZA: And so that I will caution you to
17 pay attention to Dr. Deckelbaum's request as to what the
18 group is going to need guidance from you. He mentioned two
19 or three points, so make sure that your questions are
20 targeted to that so that we can be of assistance to the
21 working group.

22 DR. DECKELBAUM: Well, let me ask it in a
23 different way. Is anyone against including nuts, you know,
24 somewhere --

25 DR. DWYER: I have a question.

26 CHAIRMAN GARZA: Let's make sure that of the ones
27 are targeted.

28 DR. DWYER: The question is, you know, I've heard
29 about a lot of single groups, some of which apparently risks

1 enormously in these analyses, some of which decrease risk by
2 large amounts. Risk go down to .6, .4, whatever. How much
3 of this is confounded?

4 DR. STAMPFER: Oh, these are -- these are
5 adjustments for --

6 DR. DWYER: I know they are, but I'm still asking
7 the question.

8 DR. STAMPFER: I think -- I mean, we measure diet,
9 we try to assess diet, we assess coronary risk factor, we
10 adjust as well -- you know, is there some residual
11 confounded? Yeah, probably there is. Is it all correct?
12 Well, it's a guess. These analysis I presented are after
13 multiple adjustments. Obviously, you can't fully adjust --

14 CHAIRMAN GARZA: Meir, can you get closer to the
15 microphone because people cannot --

16 DR. STAMPFER: The question is are these findings
17 due to confounding, and obviously that's our bread and
18 butter. We pay a lot of attention to confounding and try to
19 avoid it as much as we can recognizing that there is
20 residual confounding which could explain part of it. But it
21 seems very unlikely that it can explain these effects to a
22 very great extent, either the adverse or the beneficial
23 ones.

24 CHAIRMAN GARZA: All right, we are going to do
25 everything. Right now every single committee member has
26 their hand up, so I'm just going to go around the table.

27 Scott?

28 DR. GRUNDY: I want to address your question about
29 the separation. I know that's on your list, and I think

1 it's a very good idea to do that, and I think it's a very
2 fundamental idea for this whole guidelines because in the
3 past the linkage of those two together has been part of the
4 whole idea of a high carbohydrate diet, and to separate
5 those is -- is conceptually a very good idea, and it
6 refocuses on the role of carbohydrate as a separate issue
7 from fruits and vegetables, so I would strongly support it.

8 CHAIRMAN GARZA: Dr. Murphy.

9 DR. MURPHY: Since I'll only get one turn,
10 probably I'm going to make a couple of comments.

11 First, on the nuts issue, I think certainly nuts
12 are a nutritious food, and I have no problem encouraging
13 consumers to eat more nuts. I'm not sure I think they're a
14 fruit or a vegetable or a grain, and I would prefer, if we
15 can, to see the nut issue addressed as a protein food, and
16 in the context then of variety or maybe in our introduction
17 that is now going to be as long as the original report, but
18 I don't -- I don't think it's necessary that it be in with
19 fruit, vegetable and grains.

20 A second comment I'd like to address, I think, is
21 whether "whole" should be in the grain guideline, and whether
22 it's separate or whether it's combined. I think we need to
23 be careful about discouraging consumption of non-whole grain
24 products for many of the reasons that Richard has already
25 summarized. But I do think it would be good to focus more
26 on variety. If we're going to take it out as a separate
27 guide, it should be emphasized more in these guideline or
28 guidelines. And certainly if we could just get consumers to
29 do 50/50 whole grain/non-whole grain, we'd be many times

1 better off than we are right now.

2 So my personal preference would be to see some
3 focus, some additional focus on specificity, particularly
4 with whole grains. Could we even say try to make half your
5 grains whole grains? That would be my preference.

6 And I would also like to see potatoes on the
7 fruits and vegetable because the pyramid is based on certain
8 calculations that assume that all your vegetables are not
9 potatoes, and we don't want people to eat all of any one
10 fruit or any one vegetable. And so at least in the text we
11 say things like eat dark green or colored vegetables
12 frequently or more or whatever. Maybe we need to be more
13 specific about that, at least once a day or whatever. So I
14 would encourage the group to think about both variety and
15 specificity within these guidelines.

16 CHAIRMAN GARZA: Rachel?

17 DR. JOHNSON: No.

18 Roland?

19 DR. WEINSIER: Just a quick comment. First of
20 all, I agree with Alice. I think she has proposed a
21 reasonable addition to the guidelines in terms of splitting
22 fruits and vegetables from the grains. Conceptually, I
23 think it's a good idea. In terms of adding the number of
24 servings or whatever, I'm a little uncomfortable.

25 On the second issue very briefly, Meir suggests
26 that perhaps potatoes, which seems to be the standout in
27 terms of the vegetable group, be considered as part of the
28 grain, perhaps the starch group, and one of the compelling
29 reasons picks up on what Suzanne is saying, and that is that

1 I think the figure yesterday was about 25 percent of the
2 vegetable intake comes as potatoes, and I think the group
3 should strongly consider putting it in the grain/starch
4 group with consideration from people such as Suzanne,
5 whether it in fact is more like a starch grain or is it more
6 like a vegetable from a nutritional standpoint. So I'm just
7 raising this for consideration. I think it's a good point
8 to consider of people such as Suzanne, you know, think that
9 it is more comparable to the grain group in terms of
10 nutritional content than the vegetable group.

11 CHAIRMAN GARZA: Shirika?

12 DR. KUMANYIKA: I think separating the fruit and
13 vegetable guideline is a good idea. I wanted to emphasize
14 the need to encourage fruit consumption because the data
15 suggests that that's much more of a problem, vegetable
16 consumption, I think, even if you subtract the potatoes.

17 The other issue that comes up, I think, most
18 because of these extreme guideline is the range of servings.
19 I'm now convinced, based on totally anecdotal evidence, that
20 most people don't understand the servings as they appear in
21 the pyramid, and it's very easy to elicit a conversation
22 with a consumer who thinks that they should try to get 11
23 servings of grains, even if they're appropriate at the 1600
24 calorie level.

25 And the way the information is put in the book
26 aggravates it in the same way that you have to go hunting.
27 You have to really hunt for the information that that range
28 means different calorie levels because I went looking for
29 it, and I almost thought it wasn't in here for a minute, and

1 then I found it. But it's very submerged. So however we do
2 the servings, I think we should pick up that issue of what
3 this number means for people who are eating different -- one
4 base range and then say something else later.

5 And I would go for moving the potatoes. I think
6 we have a tendency to use the guidelines to reenforce
7 traditional wisdom rather than really tell the public things
8 that make sense based on the way we look at the data, and I
9 think moving potatoes would be a good sign.

10 CHAIRMAN GARZA: Richard?

11 DR. DECKELBAUM: Well, I think we would like some
12 input from USDA. First of all, are we allowed to move
13 potatoes?

14 (Laughter.)

15 I mean, I can just see -- I don't know --

16 (Laughter.)

17 (Simultaneous conversation.)

18 DR. DECKELBAUM: No, seriously, but that's a -- I
19 think some of these things -- some of these things are
20 history.

21 CHAIRMAN GARZA: You may not like the answer.

22 DR. DECKELBAUM: The other point, I think the
23 working group would like some input just to remind us on the
24 history of why grains, vegetables and fruits are together.
25 And I think, you know, this may have come up at previous
26 meetings. And why was a decision made not to separate them.
27 We need to know, we need to have that kind of information.

28 CHAIRMAN GARZA: Let me, before turning it over to
29 USDA, let me try once again to remind the group the pyramid

1 is not our responsibility. You can make recommendations
2 certainly. I mean, you can move potatoes, add t-bone steak,
3 but that's what it is; it's a recommendation.

4 DR. DECKELBAUM: Put steak with milk.

5 (Laughter.)

6 CHAIRMAN GARZA: You can do anything you wish --
7 only if you're concerned about --

8 (Laughter.)

9 CHAIRMAN GARZA: And I guess you'd move shell fish
10 as a stand-alone group anyway.

11 (Laughter.)

12 CHAIRMAN GARZA: Let me ask for the history and
13 then we'll turn to Johanna on why grains, fruits and
14 vegetables were put in the same -- that antedates my
15 involvement so I don't know whether anyone whose memory
16 goes --

17 DR. KENNEDY: Can I before I answer that question
18 talk about -- I think there is a fairly straightforward
19 reason why potatoes are where they are, and it's based on
20 the -- I think, Richard, your question or comment -- the
21 nutrient profile of potatoes fits more in the vegetable
22 group than it does in the grain group.

23 Now, having said that, Shirika's issue, and I
24 think it's one that as we in both departments think about
25 how we're actually going to promote the guidelines once the
26 next edition is released, I think there are serious issues
27 related to how consumers see the guidelines.

28 I mean, an example: We all know that botanically
29 tomatoes are fruit, yet we put them in the vegetable group.

1 And why do we do that? The overriding reason, I think, we
2 do it is because that's the way most consumers see tomatoes.
3 They see them as vegetables rather than fruits.

4 But from the point of view of Dr. Garza's comment,
5 well taken, that the guidelines as they will emerge guide
6 the one part of any revision on the food guide pyramid that
7 would take place, but they are only one part of it. The
8 other two parts I keep coming back to are what will emerge
9 from the new DRI because that clearly is the second key
10 building block of the pyramid, and the third part is
11 American's latest consumption patterns, which will be based
12 on the '94 - '96.

13 So get back to comments made yesterday, Alice's
14 point about what do you do with calcium fortified orange
15 juice. Well, to the extent, in proportion to how it's
16 showing up in consumption patterns, it gets fed back in our
17 algorithm into the composites that are used to look at here
18 is where we are, here is where we need to be as far as
19 having an adequate diet. So it is very complicated.

20 I would suggest maybe if this plays to some of the
21 issues which are being discussed, that maybe at the next
22 Dietary Guidelines Advisory Committee we set aside a period
23 of time where someone goes through what actually has to be
24 done to modify the food guide pyramid because it is a very
25 tedious, sometimes frustrating, but an awful lot of what the
26 staff say to me is grunt work going into the revisions that
27 are necessary for the food guide pyramid is not, as people
28 who were involved in the '95 guidelines, it's not two or
29 three people sitting down one day and pulling something out

1 the air. There is a lot more thought that went into it.

2 CHAIRMAN GARZA: Johanna.

3 DR. DWYER: I wanted to support provisionally the
4 notion of thinking about two guidelines for fruits and
5 vegetables and for grains, and express my reservations as a
6 person of Irish descent at a time -- the potato suggestion.
7 I'm not sure it isn't beyond our scope, and I'd like to hear
8 a lot -- a lot more about the food composition, the reasons
9 why it was put there originally before precipitous action is
10 taken.

11 CHAIRMAN GARZA: Well, remember --

12 DR. DWYER: I just don't have a --

13 CHAIRMAN GARZA: -- there is no precipitous action
14 that will be taken at today's meeting. We are still in the
15 information gathering stages, and all of these are just
16 suggestions.

17 Linda and then Alice.

18 MS. MEYERS: I'm responding to your, or trying to
19 respond to your comment about historically what was the
20 reason behind keeping them together, the fruits and
21 vegetables and grains together, and I'll bring in the '80,
22 '85, '90, the wording, so you can see that tomorrow, because
23 I think that gives some idea of how it's changed.

24 I'm blanking on what the 1980 said, whether it was
25 the avoid times, but clearly it wasn't an avoid starch, but
26 it was a starch and fiber emphasis at that time and I
27 recall, so that was -- it was in the context of this was the
28 first time you were making -- the government was making
29 recommendations related at all to chronic disease

1 prevention, so there was clearly attention to fats and
2 lowering fats and lowering sodium and someone, sugars, I
3 guess, and keeping carbohydrates and starch up, so
4 everything was just sort of lumped there. But I'd be happy
5 to bring those in so you can see it, unless Katherine may
6 have them here.

7 DR. JOHNSON: Yeah, the 1980 and 1985 guidelines
8 were "to eat foods with adequate starch and fiber," the
9 focus was on that. The 1990 changed to "choose a diet with
10 plenty of vegetables, fruits and grain products."

11 CHAIRMAN GARZA: Okay.

12 UNDER-SECRETARY WATKINS: I was not going to come
13 to the table because I thought it was more fun sitting back
14 listening to the comments, until you talked about separating
15 potatoes out.

16 (Laughter.)

17 But I thought you better get to the table quick.

18 I think, Dr. Deckelbaum, you asked the question
19 what kind of fire storm would this raise, and I think it
20 would be a -- one that the Forest Service couldn't handle.

21 (Laughter.)

22 DR. DECKELBAUM: Why?

23 UNDER-SECRETARY WATKINS: I think the people on
24 the other side of the house in the producing community, just
25 knowing as much as I know about what they would say, I think
26 you'd have tremendous opposition to moving potatoes from a
27 vegetable/fruit category and putting it into some other
28 category. I think you'd have -- if you think about how
29 you're going to move the dietary guidelines, if that

1 question is raised, and if anybody gets any inkling that
2 you're talking about moving it, I think you're going to have
3 a lot of controversy about the dietary guidelines.

4 And whatever you do on that issue, you would
5 certainly want to make certain, if you talk about moving it,
6 that it truly is science-based and that you have some real
7 rationale for moving it.

8 The other issue that I will mention very quickly
9 is how you connect the dietary guidelines and the food guide
10 pyramid, and there seems to have been a disconnect. People
11 really don't know. If you ask the question, do you know
12 what the dietary guides are, they really don't. Do you know
13 what the food guide pyramid is, yes. We need to connect the
14 two, and what we're hoping is that when the dietary
15 guidelines are approved that the food guide pyramid's
16 revision will be right on the heels of that. I really would
17 like to see them come out almost simultaneously. Then
18 people start to connect the two. Then we can do something
19 about the changes in diet of people.

20 But when they are two separate entities, and
21 people are not connecting them, I think we have a real
22 problem in this country, and we need to address that. So
23 we'd like to look at how can we move the two simultaneously,
24 and I know the staff is just about dying when we talking
25 about that, but I think that's kind of the way it needs to
26 move.

27 CHAIRMAN GARZA: Remember we're talking about
28 three different steps. One is whether, to the degree that
29 we want to emphasize whole grains in any guideline, to the

1 degree that we want to separate the two, the issue of
2 potatoes is an extraneous one because that can be embedded
3 in the text as to how we convey either of those two
4 guidelines, all right. But we don't have a separate potato
5 guideline.

6 (Laughter.)

7 DR. DECKELBAUM: Could we get --

8 UNDER-SECRETARY WATKINS: I'm going to pass.

9 DR. DECKELBAUM: In terms of separation of the
10 guidelines, is that something that you would think would --
11 would be -- would there be -- do you envision opposition
12 from certain groups there?

13 UNDER-SECRETARY WATKINS: I don't think so. I
14 think anything you can do to make it easier for people to
15 understand the dietary guidelines is going to be acceptable.
16 I think anything that helps make it easier and helps us to
17 get the message out, I think, is going to be critical.

18 CHAIRMAN GARZA: All right, on that positive note
19 we have five minutes for our break.

20 (Laughter.)

21 (Whereupon, a recess was taken.)

22 CHAIRMAN GARZA: We are now approximately an hour
23 and a half late, which makes any excessive, compulsive
24 individual very nervous, and I'm assuming we have at least
25 eight or nine around the table that fit that category.

26 VOICE: You could say 100 percent safely.

27 CHAIRMAN GARZA: So we should have a lot of
28 stressed individuals.

29 I'm going to challenge Dr. Johnson to get us

1 through this one very, very efficiently.

2 DR. JOHNSON: No problem.

3 CHAIRMAN GARZA: And the committee to be yet more
4 insightful and inciseful in your comments.

5 VOICE: Incise?

6 CHAIRMAN GARZA: Concise.

7 (Laughter.)

8 VOICE: And decisive perhaps.

9 DR. JOHNSON: Are we ready? Ready to roll? Okay,
10 we're ready to roll with sugar.

11 First, I've chaired the subcommittee on the sugar
12 guideline, and I'd like to thank Dr. Lichtenstein and Dr.
13 Deckelbaum for their assistance and input, as well as the
14 USDA staff, Dr. Garza and I'm very sorry that I left Shanthy
15 Bowman off this slide because she was tremendously helpful
16 in some work that we did that you'll see in a minute.

17 May I have the next slide, please?

18 The 1995 guideline said, "Choose a diet moderate
19 in sugars," and the text elaborated by saying, "Sugar should
20 be used in moderation by most healthy people and sparingly
21 by people with low calorie needs."

22 Next slide, please.

23 the first thing the subcommittee did was grappled
24 with the definition of what is a sugar, and according to the
25 World Health Organization's 1995 report, or 1997, I'm sorry,
26 report on carbohydrates in human nutrition, they say that
27 "Sugars are conventionally described as the
28 monoendysaccharides."

29 Next slide, please.

1 Unfortunately, this broad definition becomes
2 problematic in the context of the dietary guidelines because
3 it includes sugars like fructose, which are naturally
4 present in high amounts in fruit, and lactose, which is
5 present in dairy products. In fact, in the ILSI report done
6 by Dr. Geiger, consumers reported being confused by what the
7 guideline means by sugars, and they reported being
8 uncomfortable with having one guideline that limits sugars
9 and another in the same list of guidelines encouraging them
10 to eat fruit which contain sugars.

11 Next slide, please.

12 Some of this dilemma, I think, led to the
13 introduction of a number of terms which have been since
14 developed to help further classify sugars. For example, in
15 the U.K., the Department of Health uses the term "intrinsic"
16 and "extrinsic" sugars to differentiate between those sugars
17 which occur within the cell walls of plants and those which
18 are added to foods.

19 Next slide, please.

20 The USDA has begun using the term "added sugars"
21 when analyzing the nutrient intake of Americans,
22 particularly with CSFII surveys. And added sugars have been
23 defined by USDA as "all sugars used as ingredients in
24 processed and prepared foods, such as breads, cakes,
25 candies, soft drinks, jam and ice cream, as well as sugars
26 eaten separately or added to foods at the table," and there
27 is a list here specifically of which sugars are included in
28 that definition of added sugars.

29 To me, the definition is quite clear cut and

1 straightforward, and it has been well defined. It's
2 important to note that added sugars do not include naturally
3 occurring sugars, such as the lactose in milk or the
4 fructose in fruit.

5 Next slide, please.

6 Next, I'd like to get into consumption or trying
7 to answer the question exactly how much sugar is America
8 eating. The Economic Research Service of USDA collects food
9 supply data for caloric sweeteners which is comprised
10 primarily of sucrose and corn sweeteners, including high
11 fructose corn syrup, and total consumption has risen
12 steadily since 1970, as you can see here. In 1997,
13 Americans consumed on average 154 pounds of caloric
14 sweeteners compared to the 122 pounds per person in 1970.

15 Next slide.

16 Using the USDA definition which I just gave you,
17 added sugar intake varied with age and gender in the U.S.
18 population. This chart shows the number of teaspoons of
19 added sugar consumed by participants in the '94, '95, '96
20 CSFII surveys. Adolescent males had the highest intakes at
21 35 teaspoons of added sugar per day, and older females had
22 the lowest intakes at 12 teaspoons per day.

23 Next slide.

24 Added sugar intakes ranged from 12 percent of
25 total calories in females 51 and above, to 20 percent of
26 total calories in adolescents, and this was true for both
27 males and females between the ages of 12 and 18.

28 Next slide.

29 Now I wanted to move into looking at sources of

1 added sugar. Clearly the most important source of added
2 sugar in American's diets is regular calorie soft drinks,
3 which accounts for one-third of all added sugar intake in
4 the CSFII. Sugars and sweets were second in importance at
5 16 percent of added sugars and sweetened grains were third,
6 contributing 13 percent of added sugars, regular calorie
7 fruit aids and drinks were also important sources of added
8 sugars, and together these four food categories were the
9 source of three-fourths of all added sugar intake.

10 Next slide.

11 Okay, now, the next question is, okay, how does
12 sugar intake relate to diet quality or the nutrient
13 composition of the diet.

14 There were a couple of earlier studies which I
15 show here that examine total sugar intake and nutrient
16 adequacy, and the conclusion from these studies in both
17 children and adults were that high amounts of total sugar do
18 not necessarily lead to a poorer quality diet in comparison
19 with consumers with low sugar intakes. It's important to
20 realize that these studies included natural occurring sugar,
21 such as fructose and lactose which are present in foods with
22 generally high nutrient densities.

23 For example, in one study dairy foods contributed
24 31 percent of the total sugar intake in children, and fruits
25 contributed 17 percent of the total sugar intake for all
26 ages.

27 Next slide.

28 With the new CSFII database, there is now public
29 access to data on the added sugar content of foods, and this

1 allows for investigations into the impact of added sugar
2 intake on nutrient quality. And with Dr. Bowman's
3 assistance, we conducted analyses for the association of
4 added sugar intake and diet quality in the '94, '95, '96
5 CSFII surveys. We statistically adjusted for age, sex and
6 total energy intake to look at the association between added
7 sugar intake and the total unsaturated fat, protein fiber,
8 the essential vitamins and minerals, and we also looked at
9 food groups.

10 And as you can see on this slide, after
11 statistically controlling for age, sex and total caloric
12 intake, added sugar intake was negatively associated with
13 intakes of total unsaturated fat, protein fiber, the
14 vitamins listed there, A, E, C, riboflavin, niacin, B 6
15 volute, B 13 and the minerals, calcium, phosphorus, irons,
16 zinc and magnesium. It was also negatively associated with
17 the number of servings of grains, fruits, vegetables, meats
18 and dairy products.

19 And Meir and I were talking just before this,
20 clearly what this shows is that when you add added sugar to
21 the diet, which is not accompanied by any nutrients, any
22 other nutrient will be negatively associated because you're
23 simply adding empty calories, and so the impact will be
24 negative on really any other nutrient, which is different
25 than the analyses that you get when you look at total sugar
26 intake, which is included -- which accompanies food that
27 have some nutrient density.

28 Okay, I did want to point out one last thing about
29 that slide, which was -- sorry -- that note the total

1 unsaturated fat intakes are inversely related to sugar
2 intake, and this is true both with total and added sugar
3 intake. This has been referred to as the fat sugar see-saw
4 such that as sugar goes up, fat goes down. But it's also
5 important to notice that high consumers of added sugars were
6 also more likely to have low intakes of shortfall or problem
7 nutrients such as fiber, Vitamins A, C, folate, calcium,
8 iron and zinc. So we have that interesting disparity there.

9 Okay, next slide, please.

10 I wanted to move into looking at beverage patterns
11 among U.S. children because they've changed remarkably over
12 the past decade, and I'm showing some data here from a paper
13 by Morton and Guthrie that in the Family Economics and
14 Nutrition Review just late in '98.

15 I know you can't see this well but it's just
16 important to see the different bar graphs, and this shows
17 what's happening in terms of dairy product intake; that
18 basically low fat milk is remaining stable, whole milk
19 consumption is going down, skim milk and other dairy product
20 intake is going up slightly.

21 But it's important to note that overall milk
22 consumption did decline in the period between '89 and '91,
23 from 422 grams per day down to 396 grams a day in the '94,
24 '95 surveys, so milk consumption is going down, and there
25 has been some change in the type of milk that is consumed as
26 well.

27 Next slide.

28 At the same time that milk consumption was
29 declining major changes occurred in other beverage patterns.

1 The largest increase occurred in the soft drink category,
2 which increased from 198 grams per day up to 279 grams per
3 day in the '94, '95 surveys. Male adolescents increased
4 their consumption of soft drinks from a mean intake of 352
5 grams in '89 - '91, to 580 grams, which is almost 20 ounces
6 a day of soda in '94-95, and this just shows the change and
7 this is tea and breakfast drinks; soft drinks, you can see
8 the big jump. This is fruit, aids and non --

9 VOICE: It looks like "other."

10 DR. JOHNSON: Thank you, Joanne. Other drinks.
11 The author is over here so I knew she would know.

12 And on that note, I wanted to share with you an
13 article which Dr. Lichtenstein very kindly brought to me.
14 This is from the Boston Globe, March 1. It says, "Here is
15 the so-called problem, the kids in the Colorado Spring
16 schools just aren't drinking enough Coke, or so says John
17 Bushy, an area superintendent, for 13 schools who signs his
18 correspondence 'The Coke Dude.' The Colorado District was
19 hard up for money for extras like band competitions and
20 debates, so in 1997, they signed a 10-year contract in which
21 it would get eight to 11 million dollars from Coca-Cola in
22 return for giving the soft drink giant exclusive rights to
23 sell Coke and other beverages in school vending machines.
24 Sales of Coke products have been so sluggish that Bushy
25 wrote to school officials in September, and I quote 'We need
26 to all work together to get next year's volume up to 70,000
27 cases,'" and the article goes on. But this is the
28 situation that's occurred in some school districts in our
29 country with regards to soda and access by your young

1 people.

2 Next slide, please.

3 Why are we concerned about this change in beverage
4 patterns in U.S. Children? Calcium is concerned a problem
5 nutrient among most age and sex groups in the U.S.
6 Particularly problematic are adolescent and adult women, the
7 majority of whom do not meet current calcium
8 recommendations.

9 There has been research, some research by Gunthur,
10 who was with USDA, established that carbonated beverages
11 tend to displace milk in the diets of teenagers with
12 negative implications for diet quality. This displacement
13 effect has also been shown in adults. Joanne Guthrie found
14 adult women whose diets failed to meet calcium
15 recommendations, drank significantly more regular calorie
16 sodas than those with diets meeting recommendations.

17 And as we've discussed yesterday, the DRIs did
18 recently increase calcium recommendations over and above the
19 1980 RDA. So in my view or in the subcommittee's view, this
20 ongoing trend for calcium-rich beverages to be displaced by
21 beverages high in added sugars is a concern.

22 Next slide.

23 There is little evidence suggesting diets high in
24 total sugar promote weight gain when consumed in amounts
25 that do not exceed energy requirements. There is some
26 evidence, however, that soda consumption is a major factor
27 in the increased energy intakes of children and adults
28 between the '89, '94 and '94-95 USDA surveys. And, in
29 addition, a meta-analysis by Dr. Rick Matters at Purdue

1 suggested that beverages high in carbohydrates have a low
2 society effect, leading to a poor regulation of energy
3 intake and subsequent weight gain.

4 Next slide.

5 In terms of sugar and diabetes, we looked at the
6 Nurses' Health Study report which has already been talked
7 about today, so I won't elaborate on that. Basically, at
8 this time the sugar subcommittee felt that there was a
9 paucity of evidence making it difficult to determine diets
10 high in sugar are linked with the etiology or causality of
11 non-insulin dependent diabetes. There are many papers on
12 the use of glycemic index for the treatment of diabetes but
13 a real scarcity of papers on the actual etiology.

14 I did want to point out there is a paper in this
15 month's issue of Pediatrics, which I will get and circulate
16 to the committee. It was done in Susan Roberts lab in
17 Tufts, and she demonstrated that when teenage boys consumed
18 a lunch with a high glycemic index, they consumed nearly
19 twice as much food afterwards in comparison with a low
20 glycemic index lunch. And they suggested that meals with a
21 high glycemic index set off a chain of actions that caused
22 overeating and potentially could lead to subsequent obesity,
23 so that's a new paper that I think we'll want to consider in
24 our deliberations.

25 Next slide.

26 Again, I won't spend a lot of time on this. Dr.
27 Byers mentioned this earlier, and I mentioned it in my
28 comments that the World Cancer Research Fund is recommending
29 limited consumption of refined sugars for cancer prevention.

1 Next slide.

2 Very quickly, because I mentioned this in
3 September when we got together, there has been a meta-
4 analysis of 23 studies over a 12-year period, leading to the
5 conclusion that there is little evidence that sugar has any
6 significant influence on either behavior or cognitive
7 performance in children. So this idea of sugar and
8 hyperactivity in children has pretty much been put to rest,
9 at least scientifically, with these data.

10 Next slide, please.

11 Clearly, there is a role for dietary sugars in the
12 development of dental carries and between milk consumption
13 of sugar remains a risk factor for occurrence of dental
14 carries, and the recommendation now is that we focus on
15 fluoridation, adequate oral hygiene and not just on sucrose
16 intake alone.

17 Next slide.

18 Wanted to show you some data on low calorie
19 sweeteners. The USDA Economic Research Service collected
20 food supply data on low calorie sweeteners from 1970 to
21 1992. There are no data available after '92, primarily
22 because low calorie sweeteners are used as constituents in
23 other products like soft drinks and food manufacturers
24 consider this proprietary information and it's difficult to
25 get. But between 1970 and '92, consumption increased from
26 an average of five pounds per person to 24 pounds per person
27 per year, so there has been a dramatic jump in the use of
28 sugar substitutes.

29 Next slide.

1 When we did a literature study for sugar
2 substitutes, we find this one study in George Blackburn's
3 study done in George Blackburn's lab that showed some
4 evidence that multidisciplinary weight controlled programs
5 that included Asparte, a sugar substitute, enhanced or
6 facilitated long-term weight maintenance, but we could only
7 find that one study so the evidence, again, is fairly
8 sparse.

9 Next slide.

10 So, in review, I'd just to review these key points
11 that I've raised. There is consumer confusion about what we
12 mean when we say to eat diet moderate in sugar, and it
13 particularly seems to be problematic as it relates to the
14 fruit group. Since '975, USDA has defined, created a
15 definition of "added sugar" which now allows us to do
16 analyses of food consumption data of the USDA database which
17 look at added sugar and its impact on diet quality. Sugar
18 intake is clearly increasing. A third of all added sugars
19 now comes from soft drinks. I showed you some new data that
20 we had on sugar and diet quality, the sugar/fat see-saw
21 which I talked about, and the fact that added sugar intake
22 was negatively associated with a number of those problem or
23 scarcity nutrients in the food supply.

24 Next slide.

25 And I also reviewed the evidence that we've looked
26 at with sugar and weight, diabetes, cancer, behavior, dental
27 carries and sugar substitutes. So thanks very much.

28 I think in terms of the committee and what it
29 would be nice to have some comments about are what we're

1 really grappling with is this question of added sugars and
2 whether or not the guideline needs to reflect the nature of
3 some of the data that I've showed.

4 Thanks.

5 CHAIRMAN GARZA: Alice?

6 DR. LICHTENSTEIN: I think you did a nice job of
7 summarizing the group's work.

8 I think that -- I agree, I think that there needs
9 to be a mechanism for distinguishing sugar that comes from
10 fruit and milk from other kinds of sugar, and added sugar
11 really sounded like a way of doing that.

12 However, what I think we really need to know is
13 what the public's perception is of the word "added sugar,"
14 if it's just sort of sprinkling it on some breakfast cereal,
15 and it seems like the major contributor in soft drinks, and
16 I wonder how that's actually perceived terminology was
17 because that may not be perceived as added sugar, and
18 whether there are any suggestions on alternate terminology
19 that would really capture that issue.

20 DR. JOHNSON: Right.

21 CHAIRMAN GARZA: You want to respond now?

22 DR. JOHNSON: Well, I think that's a good point,
23 and I don't have the answer. I know in the focus group
24 study they said they're confused how the general public
25 would perceive the term "added sugar."

26 DR. LICHTENSTEIN: I just think we need to get the
27 information.

28 DR. JOHNSON: I think that's a good question,
29 yeah.

1 DR. LICHTENSTEIN: Yeah.

2 DR. JOHNSON: I think I'll sit down if that's all
3 right.

4 CHAIRMAN GARZA: Meir.

5 DR. STAMPFER: I think it's clear that the added
6 sugar is having a big impact on diet even though we can't
7 pin much in the way of specific diseases to this, but its
8 adverse effect in -- main adverse effect is that it's
9 displacing foods that do provide nutrients. So I think this
10 might make a good model case to consider as one of the sort
11 of second tier guidelines, if we got to that proposal that
12 several people had made of distinguishing sort of top tier
13 and second tier guidelines, that this, I think, should
14 remain as a guideline, and perhaps the text could be shrunk
15 a little bit, and it could go into the second tier where
16 maybe some other guidelines might go.

17 CHAIRMAN GARZA: Any other? Johanna?

18 DR. DWYER: Just a -- thank you for an interesting
19 presentation.

20 One thing it seems we need a little more work on
21 is cariogenecity, and it seemed to me in a brief review of
22 the literature a couple of months ago that it might be
23 helpful to think of all of these variables, these various
24 diseases as which ones seem to be most associated with
25 whatever this thing is, extrinsic or whatever you want to
26 call it, and what isn't.

27 Certainly with cariogenecity there are two issues.
28 One is something to do with the composition of food with
29 respect to sugars. Another is the carbohydrate, the cooking

1 of the carbohydrate so that the starch is also associated
2 with cariogenicity. And then there are all of these other
3 things about when you eat it, what you do afterward and so
4 forth. Do you have a toothbrush afterward?

5 CHAIRMAN GARZA: Johanna, can you speak closer to
6 the mike?

7 DR. DWYER: It would seem to me it would be
8 helpful if we could just array them as we ponder this issue.

9 The other thing, Rachel, was I wasn't sure what
10 you were -- the group was suggesting. Is it change the
11 existing guidelines to choose a diet moderate in added
12 sugars?

13 DR. JOHNSON: That's one option that we've
14 considered.

15 DR. DWYER: And what are the others?

16 DR. JOHNSON: We're looking for help. That was
17 our primary option that we considered, was whether or not we
18 wanted to use that term "added sugar" in the guideline
19 itself.

20 I mean, I suppose another option is do we need a
21 sugar guideline.

22 CHAIRMAN GARZA: Dr. Kumanyika?

23 DR. KUMANYIKA: I think that the presentation is
24 very convincing that a guideline is needed on something like
25 food and beverages with added sugars, which is different
26 from what it says now, which is "moderate in sugar." So you
27 get into the "avoid" issues. And if you said then you're
28 back to the limit, foods and beverage with added sugar, but
29 something in that spirit is much clearer this round than

1 I've remembered in the past because it's very clear that
2 it's a displacement issue and it's not that you're trying to
3 link sugar in the diet itself to the health problems, but
4 it's part of the pattern, that it's not -- that it's
5 replacing things that are needed, so maybe that word could
6 give someone an inspiration for how to --

7 DR. JOHNSON: Say that again, Shirika? Food is?

8 DR. KUMANYIKA: Foods and beverages with added
9 sugars, because then it doesn't matter who adds them.
10 People know it's been added.

11 CHAIRMAN GARZA: Any other comments or
12 suggestions?

13 Dr. Dwyer?

14 DR. DWYER: Yeah, I still have concerns about the
15 "added" business, and I guess the first thing is something
16 that Dr. Lichtenstein brought up, which is what does that
17 mean. Does that mean to most people sugar from the sugar
18 bowl or does it mean corn syrup? It strikes me that maybe
19 there is more focus group information we haven't seen, but
20 I'd really like to see that.

21 And the second things is I'm having trouble
22 remembering all the things you said and which ones are
23 related to "total." For instance, cariogenicity, I know
24 from our own work that vegetarian children who eat a lot of
25 raisins get just as high carries as kids who were non-
26 vegetarians who ate a lot of added sugar. So that it would
27 help me to array those things and think of them before I
28 made a decision.

29 CHAIRMAN GARZA: Dr. Deckelbaum and then Dr.

1 Kumanyika.

2 DR. DECKELBAUM: I guess added sugars was a major
3 part of our discussions and how to define it. And I guess
4 in the simplest way, because it's added sugars by food
5 producers or its added sugars in the home. But I think that
6 it's where you add simple sugars, as a first step add simple
7 sugars to food, to sort of natural food either in home or
8 industrially. So I guess if you were a soft drink
9 manufacturer and you put it in the mix, that would be an
10 added sugar. Similarly at home by adding it to tea or
11 coffee or whatever would be an added sugar.

12 We didn't really discuss too much about corn syrup
13 and corn syrup solids, but, again, those added sugars, and
14 we had some discussion on that yesterday, are in large part
15 small glucose polymers. Corn syrup, I think the mean sort
16 of size of a glucose polymer is about 15 glucose molecules
17 together and they're not very sweet, so they're not added --
18 they're not added --

19 DR. DWYER: I take issue --

20 DR. DECKELBAUM: They're not added for sweetness.

21 DR. DWYER: Okay. I take issue with the
22 importance, I take issue with that statement, but I also
23 take issue with the question that they are not important. I
24 think they are very important and we need to see some
25 breakout data because it strikes me that a lot of --

26 DR. DECKELBAUM: It's not important.

27 DR. DWYER: A lot of the corn syrup solids,
28 sweeteners, I think a lot of what's added to the soft drinks
29 that Dr. Rachel mentioned are corn syrup, it's not -- isn't

1 it?

2 DR. JOHNSON: Well, remember that soda is the
3 number one source.

4 DR. DWYER: So they're not added to the make up --
5 (Laughter.)

6 CHAIRMAN GARZA: Dr. Kumanyika, on that.

7 DR. KUMANYIKA: I just wanted to pose the question
8 of if the goal of this guideline would shift to being foods
9 and beverages with added sugar or sweeteners or whatever,
10 then it might not be the place to address some of the issues
11 of total sugar or total carbohydrates. It's just -- I mean,
12 we think about some carbohydrate issues as part of another
13 guideline. If this one could be clearer, focusing on this
14 added sugar displacement problem.

15 CHAIRMAN GARZA: Carole, is there either plans
16 within the department to do some focus groups --

17 MS. DAVIS: Yes.

18 CHAIRMAN GARZA: -- before, that one could explore
19 at least the meaning to consumers of added sweeteners, added
20 sugars, a variety of various messages such as the one that
21 Shirika suggested?

22 MS. DAVIS: Yes, we have plans -- I don't know if
23 this is on or not. We have plans to do that. it's going to
24 be very limited, and we're using this to get other things
25 that you want to -- to have us study, and we just hope the
26 timing will be right. We're in the process now of going
27 through all of our clearances that we have to do.

28 CHAIRMAN GARZA: Because what the group might want
29 to do is to focus its attention on the rationale for

1 modifying the current guideline either as a first or second
2 tier, and then coming to some judgment later on, based on
3 that evidence and the added input of the focus group, as to
4 what would be the best wording, and then making a final
5 recommendation based on the basis of both types of evidence.
6 Is that --

7 DR. LICHTENSTEIN: I think that's a very good
8 idea. I think when the subcommittee was deliberating, you
9 know, we came up with the word "added," and we all -- we're
10 just abnormal, but it all seemed real clear to us what we
11 were, you know, talking about, and it wasn't until I
12 actually heard the presentation and then relooked at the
13 contribution of sugar that I realized that it probably
14 wouldn't be perceived as added sugar, and I also think we
15 need to find out more about corn syrup because if you taste
16 corn syrup it's sweet. If the mean polymer size is 15, one
17 wouldn't predict it to be --

18 CHAIRMAN GARZA: That's a smaller size though.
19 It's not --

20 DR. LICHTENSTEIN: Right. One wouldn't predict
21 thought that it would be sweet because even the
22 monosaccharides would be glucose, which have less
23 sweetening, relative sweetening than fructose. I just think
24 we need to find out, but that's something that's sort of a
25 factual thing. But it seems in a lot of the food labels
26 that you look at, a lot of it is corn syrup and not sucrose.

27 CHAIRMAN GARZA: Okay. Can we move on to the next
28 one?

29 DR. DWYER: It seems to me it depends on what the

1 meaning of "is" is.

2 (Laughter.)

3 CHAIRMAN GARZA: That was Dr. Dwyer. Can you tell
4 me why?

5 (Laughter.)

6 CHAIRMAN GARZA: It's gotten to be an internal
7 joke among the committee.

8 All right, let's move on then.

9 Kathryn?

10 MS. MCCURRY: i'm sorry. I just wanted to remind
11 the committee that if you're taking about distinctions
12 between added versus intrinsic sugars, and thinking about
13 guidance to the consumers, virtually every processed food
14 label is "added" in terms of total grams of sugars per
15 serving, and that would account for all forms of sugar that
16 are added, although I believe there are distinctions in
17 terms of -- I think it monoendysaccharides that are labeled,
18 although there is some --

19 CHAIRMAN GARZA: You're saying that the term
20 "added sugar" is on the food label?

21 MS. MCMURRY: No. Total sugars.

22 CHAIRMAN GARZA: Total sugars.

23 MS. MCMURRY: Total sugars is what's on the label,
24 although there is --

25 CHAIRMAN GARZA: We'll get somebody to clarify.

26 All right then let's move on then to another
27 noncontroversial guideline. Dr. Kumanyika is going to
28 provide us with untold wisdom in about 30 minutes. In 30
29 minutes we will have this guideline resolved.

1 DR. KUMANYIKA: I actually think this is going to
2 be short. What time is lunch? You think you can get two
3 more in before lunch to catch up?

4 CHAIRMAN GARZA: At the present time there is no
5 lunch.

6 DR. KUMANYIKA: Okay.

7 CHAIRMAN GARZA: But we'll see what we can do.

8 DR. KUMANYIKA: All right. Okay.

9 DR. DECKELBAUM: It's the hour and a half that we
10 went over.

11 DR. KUMANYIKA: The sodium subcommittee consists
12 of myself and Drs. Dwyer and Stampfer, and Joan Lyon, who is
13 giving us staff support and very helpful in keeping us on
14 track.

15 What I'm going to do is just highlight what we've
16 been doing and some of the issues that we think are
17 important for sodium this time. If we leave that one for
18 awhile, the key issues in sodium are whether to keep the
19 guideline at all, and then if we decide to keep it, what
20 should it say. And the reason I say whether to it at all is
21 because it has been suggested, for example, yesterday that
22 we drop the guideline, so I think the committee needs to
23 consider that since the challenge is put before us of
24 whether this guideline should continue.

25 What we did after the last meeting was to go
26 through and identify all of the issues that had anything to
27 do with either the validity of the guideline or the reasons
28 for supporting the guideline, the evidence. And we had the
29 sort of unusual situation of having the National Hot Line of

1 Blood Institute convene a conference on this topic between
2 the two meetings. So rather than -- and I certainly am not
3 going to try to summarize the proceedings of the conference,
4 because I think we needed to have a two-day conference
5 where, fortunately, our entire subcommittee was present for
6 the whole time, so we've heard a review of the evidence on
7 the topic. I'm going to go through what was covered at the
8 conference first. Then as Drs. Dwyer and Stampfer to give
9 their impressions of whether -- the question I posed to them
10 is whether anything that they heard in the presentations at
11 the conference alters their understanding of the supporting
12 evidence.

13 Before I do that, I want to read off a list of
14 questions that I raised about this guideline because there
15 is a sense that this case may not be strong or may not be
16 existent. So one of the questions is if the cases for
17 sodium reduction as a guideline is not strong, why is it not
18 strong? And here is the list. I don't have this on the
19 side.

20 One is, is it not strong compared to that for
21 other guidelines, which would mean it's a second tier type
22 of guideline or something? Is it not strong compared to the
23 case for other dietary factors that might influence blood
24 pressure, which is a different issue? And some of the
25 arguments that are made are that it's not strong because by
26 itself it won't do as much as other factors and therefore we
27 don't need it.

28 Thirdly, is it not strong simply because the
29 methodology to address the issue is limited, which means

1 that people think it's there but because urinary sodium, 24-
2 hour urinary sodium is hard to collect and dietary doesn't
3 get sodium? It just not strong because the type of evidence
4 we have is by its very nature inconclusive. Another reason
5 it might not be strong is because the studies that would
6 tell you if the case is good haven't been done yet, and I'm
7 going to mention a little bit later one of the studies
8 that's currently in the field. Or is it not strong because
9 the evidence is actually equivocal and, you know, the case
10 isn't there?

11 And, finally, is it not a strong case because
12 there's a lot of noise in the system, which I take to mean
13 to mean that it is actually a strong case, but there are --
14 people put noise in the system by trying to cite odd and
15 invalid evidence to confuse whether there is a strong case
16 or not.

17 So why don't we put up the next overhead.

18 I think this actually summarizes the sense that we
19 had at one of our conference calls, but this is still open
20 to discussion; that the guidelines says there is a role for
21 sodium reduction in the general population, and that there
22 is a defensible case for that based on the evidence.

23 At the NHLB conference, which was chaired by Drs.
24 Martha Hill and Aram Chobanian, we had an overview of the
25 relationship between sodium and blood pressure by Dr. Paul
26 Whelton; differences in the blood pressure responsiveness to
27 sodium intake, which was the salt-sensitivity issue, by Dr.
28 Myron Weinberger, and that wa discussed -- there were
29 discussants: Drs. Margo Denke, John Flack and Steven Hunt;

1 and Dr. Frank Sacks talked about the interactions between
2 sodium, potassium, magnesium and calcium, and gave a little
3 DASH advocacy at the end of his presentation. He's one of
4 the main players in the DASH study and we had that discussed
5 by Drs. Ted Kotchen, David McCaron and Laura Svetkey.

6 As you might know if you know this literature from
7 some of the names, we had almost all of the antagonists and
8 protagonists there except that, as Dr. Lenfant mentioned i
9 his opening remarks, Dr. Mickey Alderman chose to be out of
10 the country during the days that we had the conference.
11 That's the way that Dr. Lenfant put it. And so he was not
12 there. Someone else presented some of his data that he's
13 published.

14 We went to sodium and blood pressure in the young.
15 Dr. Ron Prineas talked about the effects of neonatal sodium
16 intake. Dr. Bonita Falkner, Sodium and blood pressure in
17 children, which was discussed by Dr. Gerry Berenson,
18 Clarence Grim and Julie Ingelfinger.

19 We looked at the clinical trials and studies. Dr.
20 Graudal presented his meta-analysis of trials of sodium
21 reduction. Richard Grimm talked about subpopulations by
22 age, race and gender. Janice Douglas, prevalence of sodium
23 sensitivity in postmenopausal women, and I talked about
24 sodium reduction and quality of life issues, and we had
25 discussants including Larry Appel, David Freedman, Diana
26 Petitti, James Robins and Michael Stoto. And by this time
27 it was really a very lively meeting with, I think, people
28 who hadn't all been in the same room together to discuss
29 these issues actually having a chance to hear each other's

1 arguments and agree with them or rebut them at that time.

2 Paul Elliott presented by INTERSALT data, and then
3 Dr. Chris Sempos talked about the cohort studies that have
4 some long-term data and mortality data, so he presented
5 Alderman's Work Site Cohort Study and the NHANES I analysis
6 that Dr. Alderman has published, and the Scottish Heart
7 Health Study data, so those were the three -- three of the
8 studies that have relationship, some relationship between
9 sodium and mortality.

10 And then Dr. Jerome Cohen presented the mortality
11 data from the MRFIT-Follow-up. So that's a fourth source of
12 mortality data; and we had Dr. Nancy Cook, Kesteloot, Graham
13 MacGregor and Louis Tobian to discuss that.

14 The role of sodium in non-cardiovascular
15 conditions was a topic on the second day. Dr. Dan Jones
16 addressed that. We talked about sodium in left ventricular
17 mass, Richard Devereaux, and Jay Cohen, Ed Fruhlich and Lew
18 Kuller discussed that.

19 Dr. Suzanne Oparil was at the -- gave a
20 presentation on the renin-angiotensin system. The
21 sympathetic nervous system was discussed by Allyn Mark, and
22 then Plasma insulin, cholesterol and coagulation factors.
23 So this was really an exhaustive discussion not designed for
24 the Dietary Guidelines Committee, but certainly more than we
25 ever wanted to know about the details of the topic.

26 The basic research in the area was discussed and
27 the future studies, clinic and epidemiological research and
28 the question was posed as a discussion topic of whether
29 there should be a randomized clinical trial on sodium

1 reduction and blood pressure with morbidity or mortality as
2 an outcome.

3 At the end we had a presentation by Dr. Michael
4 McGinnis on dietary guidance public policy, and Drs. Dwyer
5 Stampfer both were discussants, Dr. Bill Harlan and Marion
6 Nestle all talked about some of the policy considerations.

7 In the hallways, some of the people who were
8 present actually revealed that they thought it was a
9 consensus conference, and they thought we were going to take
10 a vote at the end of the conference, and some of the NHLBI
11 people, and Dr. Hill, certainly made it clear that the
12 purpose of this meeting was not to have a consensus, but it
13 was just to really describe the evidence and have people
14 have a chance to hear the arguments and hear the arguments
15 about the arguments so that we could make up our minds.

16 So I think I'll stop there and just ask for
17 whatever comments that the other two subcommittee members
18 from the DJAC want to make, and then I'll go into the talk
19 about what we would have in the guideline if there will
20 still be a guideline.

21 Do you want to come up here or use your mike
22 there? As long as people can hear you.

23 DR. DWYER: I thought you gave a good summary
24 there. Just a couple of observations.

25 The first was the level of heat was considerable,
26 the level of vipe was also considerable in this conference,
27 but I thought that the data were very well reviewed. Some
28 impressions I had was that one of the problems is the data
29 aren't as strong as some of the other things we've been

1 talking about, like saturated fat. Nevertheless, I think
2 what your side says, Dr. Kumanyika, is correct; there is a
3 role. The problem is not exaggerating on either end of the
4 debate.

5 That slide from DASH is interesting, and certainly
6 it will be of great interest to follow the community-based
7 intervention, but a feeding study with the facts is not the
8 same as what we're after, which is more what happens if you
9 give dietary advice or recommendations to federal officials
10 or other officials. Nevertheless, it does seem to be safe.
11 Any concerns I had on that were dispelled by what I heard.

12 The question is how effective it is and how big an
13 effect, what people can do, can make.

14 DR. KUMANYIKA: Thank you.

15 Meir?

16 DR. STAMPFER: Those pretty much were my
17 sentiments after going to the meeting. I think first on the
18 safety issue, we can basically completely dismiss any
19 important concerns on all of safety of low salt diet. That
20 turned out not to be a credible issue.

21 I think the evidence is actually pretty good for
22 modest effect, and I think the evidence for big effect is
23 weak. So my take on it is that I agree there is a role for
24 sodium reduction in the general population, and where to
25 rank it in the hierarchy of advice is open to question, you
26 know. If we want to tell Americans some number of things
27 that they can do in their diet to improve their health, I
28 think sodium fits in there, but it's not in the top tier of
29 recommendations.

1 So my bottom line take would be to keep the
2 guideline, perhaps shrink the text, and like with sugar, to
3 put it in the second tier. But I wouldn't weaken it in the
4 sense of relaxing the recommendation at all. I think the
5 evidence is good for a modest but important effect on a
6 population level.

7 DR. KUMANYIKA: Okay, thank you.

8 So if there will be a sense that there is no
9 scientific evidence that's been put forth since 1995 that
10 warrants dropping this guideline, then the question is what
11 should we say in the supporting evidence, and is there
12 something we can put in the text that would make it clearer
13 to people.

14 Put up the next.

15 So we went through the points that are made in the
16 current guideline, and have just summarized the things that
17 the committee has discussed and will be working on according
18 to those points.

19 These three bullets, "Sodium and salt are found
20 mainly in processed food, processed and prepared foods" is
21 currently one of the lead-off points in the bulletin, and
22 will probably remain in the bulletin, and one question will
23 be would we suggest that it be in that section? Is that
24 really the main point? And this is, again, this is foods
25 with added salt. It's less likely to add salt to beverages
26 although there are some liquid foods that have salt added.
27 So this would be one to retain and maybe elaborate.

28 The next is "How much sodium or salt should the
29 average adult consume?" This is one where the 1995

1 committee has been criticized for being vague. What 's
2 there now is a statement, "Most Americans consume more salt
3 than needed," and then in the advice for today, it says that
4 the food label says 2400 as a daily value.

5 So one question for you is should we then have
6 this section on how much sold, or sodium or salt should the
7 average adult consume, and then address head on things like
8 where we get -- where the recommendation would come from,
9 like what requirements are; point out that the average adult
10 includes a lot of people who, like the hypertension, more
11 than 50 percent of adults who are trying to reduce their
12 sodium intake have been so advised; comment on children,
13 lower limits.

14 The other thing that we've wondered is whether we
15 should come and explicitly on the type of people who would
16 be accepted from any recommendation we would make about the
17 amount of sodium. Do we mention conditions, salt-losing
18 conditions or something where people would know that they're
19 not in the general healthy population, because some of the
20 criticism that's come about adverse effects really talks
21 about populations that are not healthy, that have some
22 condition where you would not restrict their salt, where you
23 might even want to give them salt, and that's mixed in in a
24 way that I think frightens a lot of people, or certainly
25 confuses them. So we could help consumers perhaps by saying
26 there are certain people from whom sodium reduction is not
27 recommended and say who they are.

28 In terms of how much sodium Americans eat, I was -
29 - on our table this morning is a report from the USDA, "Away

1 From Home Foods." Some of you have it. The committee has
2 it. And there are three tables on sodium that I thought
3 were interesting, so this is too small for you to see the
4 details. I'll tell you what the bylines are for these
5 graphs.

6 I thought this was interesting. I hadn't seen
7 data put together this way before, and I would like
8 ultimately to know from someone who's been involved in the
9 analysis how this information was derived.

10 The first one is that American sodium intake
11 remains high above the recommended level, and it shows that
12 the -- over time that sodium intake is -- this is in
13 milligrams per 1,000 calories, that it's been remaining
14 high. It hasn't gone down. The food away from the home
15 actually has a little blip there. I just had looked at this
16 this morning during the meeting so I'm not that familiar
17 with it. And if the author is here, you will be free to
18 comment if the Chair would permit it. So that's all food
19 and food at home.

20 And then this benchmark on sodium density is
21 showing that it's going down because calorie intake in these
22 data sets has gone up, and so the sodium intake being
23 relatively flat by this estimation is from less per 1,000
24 calories. That's a little bit confusing, I mean, because
25 sodium intake sometimes tracks very well, correlated about
26 .6 with calories, so I'm not sure where that comes from.

27 But this Figure 10, Restaurant of foods contain
28 much more sodium than other away from home foods," this is
29 interesting, certainly something that consumers are not able

1 to figure out themselves how much sodium is in restaurant
2 foods. And this graph suggests that it's going down since
3 about 1991, but it's still higher than foods from other
4 sources. This is new information that might give us a
5 handle on how to advise consumers.

6 And the third is showing the percent of people
7 meeting recommendation. I'm assuming this is data that
8 includes discretionary salt, but if not, then it would be
9 probably an over-estimation of the people meeting the
10 recommendation. So it gives the average intake over time
11 and shows that -- and it's gone from 41 percent meeting new
12 recommendation down to 34.

13 And I've seen NHANES data put forth differently
14 that implied that sodium intake is going up, and I haven't
15 been sure whether that's because of a better probe being in
16 the more recent dietary assessments, or if we actually feel
17 confident that sodium intake is going up and the fewer
18 people are meeting the recommendations.

19 That would certainly address one of the issues
20 related to whether we need a guideline, because in the
21 absence of a guideline, or if the guideline is downplayed,
22 you may not stay level; you may actually have sodium intake
23 increase and what is the health effect of that.

24 Okay, I also had just -- some of our discussion,
25 we talked about behaviors and sodium-related behaviors. We
26 have this "Advice for Today" section, and you could replace
27 that or add a section of new habits that can reduce sodium
28 intake in the interest of how and maybe positive behaviors
29 that have the effect of reducing sodium intake instead of

1 the avoid.

2 I also wondered when I looked at this and listened
3 to the discussion yesterday if anyone has ever tried to
4 standardize the rest of the bulletin, because the "Advice
5 for Today" seems to be the only section that appears in all
6 of the guidelines at the end. But when somebody was talking
7 about the alcohol and they said, well, you made the
8 statement about historical use in alcohol but you didn't
9 make it for anything else, and so I just wondered if anybody
10 had ever thought about having the same categories as you go
11 through the guidelines, and one would be what behaviors will
12 get you there and another might be what's the history behind
13 this or whatever.

14 But we could focus on habits because there are
15 types of habits that people in some of the clinical trials
16 have adopted to help them reduce their sodium intake, and
17 that could be made more prominent so it's more of a "how-to"
18 guideline as to necessarily a blood pressure guideline,
19 which is the way it reads to some people.

20 Let's put the next one up.

21 The next key point in this guideline has it
22 appears now is that sodium is associated with high blood
23 pressure, and some of the criticism that have come up relate
24 to the fact that it is not the only factor related to blood
25 pressure and maybe is not the main factor related to high
26 blood pressure in the population. So one possible way of
27 addressing that would be to talk about the two things that
28 are shown here: to mention that it's an important factor
29 associated with high blood pressure, but not imply that it's

1 the only factor; and then describe where sodium intake fits
2 in to the overall eating pattern, which would be a place
3 where you could mention something like a DASH diet and
4 cross-reference to other fruits and vegetables.

5 Text like that appears there now, but it's framed
6 as other factors also affect blood pressure, and one way to
7 think about it is, you know, where does sodium fit in with
8 other dietary factors, which is not quite the same thing,
9 but it would allow you to put the same information there.

10 I wanted to comment on the DASH II study because I
11 think that the ultimate question about the short-term effect
12 of the DASH eating pattern on blood pressure and its
13 relationship to sodium reduction will be answered in this
14 study and we might even in the supporting evidence describe
15 the study even though the results will not be available. So
16 I'm just going to review the design of that which Joan got
17 from the worldwide web and it's available there for those of
18 you who don't have a copy.

19 That study is contrasting the DASH dietary
20 pattern, high fruit and vegetable, dairy dietary pattern
21 with the control pattern, and then people will be crossed
22 over on three levels of sodium intake so DASH 50, 100 and
23 150 per day.

24 So as I understand the design, there will be four
25 centers and each one will have 100 -- there has, there has.
26 I think the actually finished data collection in November,
27 and will be able to look at 50, 100 and 150 without the DASH
28 diet and also be able to look at the effects of 50, 100 or
29 150 with the DASH diet using people as they are on controls,

1 and the cross-over design with like a two-week washout in
2 between.

3 They began that in August 1997. Recruitment,
4 began the experimental diets in January of 1998, and
5 eventually, perhaps for the 2005 dietary guidelines
6 committee, would be able to address the issue of dietary
7 pattern of sodium in blood pressure in a more coherent
8 fashion.

9 I probably mentioned that to say that until that's
10 type of study is done there won't be any way to tell much
11 about additive benefits of DASH and sodium intake because
12 the DASH held sodium intake constant at a moderate level of
13 three grams per day.

14 There is also a study that is planning -- planning
15 stages now which is called "Permier." It's an investigator-
16 initiated study, it will test the DASH diet with weight
17 reduction and sodium reduction in a free living population,
18 and that might answer the test of what you could expect if
19 you put everything actually in the dietary guidelines
20 together, except with the DASH pattern being a little bit
21 more aggressive on fruits and vegetables than the
22 traditional pattern, so some of this is just to say that we
23 don't know much more about the role of sodium and its
24 ultimate benefits than we did in 1995, but we can describe
25 it a little bit differently, and we may know down the line.

26 May I have the last slide? It's about consistency
27 with other recommendations.

28 Reduction of sodium or salt is recommended in the
29 Surgeon General's report on nutrition and health, the

1 National Research Council, the Report on Diet and Health,
2 and that is like six grams of salt per day. The Surgeon
3 General's report doesn't have a numeric goal or didn't have
4 a numeric goal. Then the daily value is 2400 both for the
5 2000 and 2500 kilo-calorie intakes. The level of sodium
6 reduction hasn't been -- maximum sodium intake hasn't been
7 changed by calorie level.

8 The requirement is listed as 500 milligrams of
9 sodium per day in the 10th Edition of the RDA as a safe
10 minimum intake. As some of you will remember, the 9th
11 Edition had a range for adults of 1100 to 3300 milligrams
12 per day of sodium but that was not repeated in the 10th
13 Edition of the RDA.

14 The healthy people 2010 objectives are moving
15 towards a total sodium objective, but I don't know what it
16 is yet. It may not have been formulated. The 2000
17 objectives, some of the text that the committee has says
18 that the objectives were met, which is an error, but the
19 2000 objectives were behavioral objectives that had to do
20 with the addition of salt, preparing foods without adding
21 salt, buying low salt foods and avoiding adding salt at the
22 table.

23 There is, I think, no data on the food preparation
24 goal. The goal was 65 percent of people preparing foods
25 without added salt. But the use of salt at the table, the
26 goal is 80 percent rarely or never using, and the 1996 data
27 shows 62 percent rarely or never using salt at the table,
28 which also emphasizes that it's going to be food sodium.
29 And then adults who regularly purchased foods with reduced

1 salt, the goal was 40 percent, and the 1995 data say 19
2 percent.

3 But I think that there is a move to change because
4 those behaviors by themselves may not add up to a low sodium
5 intake because they only deal with discretionary additions
6 or buying certain products, so if there is a new healthy
7 people 2010 guideline that's quantitative, we might want to
8 see when that's going to be available, and if we can align
9 ourselves with that.

10 That's all I wanted to say about this -- well, one
11 more thing. We've looked at adverse affects and other
12 issues and have reviewed this iodine issue. Dr. Dwyer had
13 some comments about that in one of our conference calls and
14 mentioned that the HLBI conference, and since then we've
15 obtained a report from a CDC conference on the iodine issue.
16 And right now I think that it means we might want to make
17 sure we describe the role of iodized salt and current levels
18 and the fact that they're going to be monitored in the
19 population more clearly in the supporting evidence and in
20 advice to consumers, and I don't know if you want to say
21 anymore about what you think we should say because, I mean,
22 the concern is that if iodized salt is an important source
23 of iodine, we need to take that into account when we are
24 recommending limited use of discretionary salt.

25 DR. DWYER: I think on that iodine issue, that
26 there are two issues. One is that it appears from the work
27 of Powell, I think it is, et al., and we can show you the
28 slide tomorrow if you wish, that there is a sizeable
29 proportion of people who don't seem to be getting enough in

1 terms of their urinary iodide, and maybe some of that has to
2 do with the characteristics of these particular people and
3 their use of various foods and salting and so forth.

4 Then there is another group of people who probably
5 get more than they need or too much, and so it's like the
6 three bears: too much, not enough, just right. And what
7 we'd want to do is as we ponder these issues is to think
8 more about how we can help on that.

9 The other issue is the second issue that is a
10 hangover from the '95 guideline discussions, and I don't
11 think we have really discussed this in depth yet, Shirika,
12 but the question of quantifying sodium or salt guidelines
13 means that we think the evidence is very great that there is
14 reasons to quantify, doesn't it?

15 And the only recommendations, I think, in '95 that
16 are quantified now are saturated fat and total fat.

17 DR. KUMANYIKA: I don't know. I think we need to
18 hear comments on that. The best reason for quantifying, at
19 least with the range or some limit, to me is that consumers
20 won't be able to use the guideline without that. But
21 whether or not that makes it easy to quantify it and be
22 correct about it is a different story.

23 DR. DWYER: But how can consumers use it even if
24 we quantify it?

25 DR. KUMANYIKA: Well, I mean, I'm thinking about
26 like the old cholesterol guideline that was 300 because it
27 was half of what people were consuming. Do you know what I'm
28 saying?

29 If people -- if we decided that people should eat

1 less than their -- without saying that this is their
2 physical threshold that will have an effect on your blood
3 pressure, but just that there is a greater relationship.

4 CHAIRMAN GARZA: Shirika, was there anything that
5 any of you might have learned that would bring greater
6 specificity to the recommendation in terms of who might
7 benefit? Or at the NHLBI conference are there
8 characteristics of the population that would help us provide
9 guidance beyond saying, well, if you're human, you know, and
10 regardless of whether you're 90 years old or five years old?

11 As we look at other guidelines, we're trying to
12 make them wherever we can as specific as possible. Any new
13 data that we should be paying attention to along those
14 lines?

15 DR. KUMANYIKA: I think if my hearing of the
16 conference was that the only basis for doing that would be a
17 lifestyle basis in terms of how has lifestyles or eating
18 patterns that are most likely to put them at risk of eating
19 a lot of sodium to people who have -- I mean, family history
20 type issues. But the salt sensitivity discussion -- well,
21 some of the discussions were very disappointing because they
22 really didn't tell you anything more than you knew before,
23 even though we had hoped to come to a new level of
24 discussion. I didn't hear anything new about salt
25 sensitivity. Many people think it is not a dichotomy and
26 that it's confusing to make it sound like a dichotomy.

27 But we have a list of specific issues that we've
28 done a literature review on but we haven't, you know, read
29 through everything and discussed it. I don't know if we're

1 going to get anything new or not of that, but that's one of
2 the issues that we have and we've posed some questions in
3 our outline that we might be able to turn into special
4 analyses of data or modeling of how many people -- you know,
5 how far would people have to reduce their sodium to move the
6 blood pressure distribution into a better range? How many
7 people in certain studies have actually lowered their blood
8 pressure if they reduce their sodium intake? Just different
9 ways of looking at data that haven't been exactly done in
10 published reports that we might be able to get a better
11 handle on the likelihood of the average person responding.

12 But at the conference, I think it was a population
13 argument, so it's not really responder/non-responder type of
14 argument, and Johanna may want to comment.

15 DR. STAMPFER: I agree.

16 DR. DWYER: The question I've got though is I
17 don't think -- I think the RDAs for sodium, for all those
18 electrolytes, are they coming up soon? Are they going to be
19 reviewed soon?

20 CHAIRMAN GARZA: They are going to be reviewed. I
21 don't anticipate reviewing -- is Alice, is Alice on the
22 floor here?

23 The last is that there is at least a year and a
24 half, two years away. I don't see them coming up before
25 then.

26 DR. DWYER: From starting.

27 CHAIRMAN GARZA: From starting, that's right,
28 unless the federal group can bring us more current
29 information.

1 MS. SUTOR: Part of that -- part of that depends
2 though, doesn't it, on whether the DRI committee sees that
3 it would be more important or more useful to do the
4 electrolytes as their next group rather than the macro
5 nutrients.

6 CHAIRMAN GARZA: And there is so much pressure now
7 to do macro nutrients that I doubt if that's going to be
8 reordered.

9 Rachel, and then Scott.

10 DR. JOHNSON: I just wanted to comment on the
11 quantification of the sodium guideline. It seems that there
12 is this driving need for people to have a number. And if I
13 can remember correctly, there really never was a number, and
14 then diet and health came out, what, in the mid eighties or
15 late eighties, and they said six grams of salt, which we all
16 immediately translated to 2400 milligrams of sodium and that
17 sort of became the magic number and then it was on the food
18 labels, and everyone uses that as the cutoff in diet quality
19 research to say what percent of the population meets t.

20 So I think, you know, that seems to be the number
21 that's sort of out there. So whether or not we concur with
22 that or not, I think in the absence of the dietary
23 guidelines giving a number, there will be a number that's
24 used as a cutoff for the population.

25 CHAIRMAN GARZA: Scott.

26 DR. GRUNDY: The current terminology is moderate,
27 and I think -- I think that might have a moderating effect
28 on the population. And, you know, one of the questions is
29 going from moderate to low, which seems like it has a

1 questionable effect, but it's -- you know, it's also a
2 little unrealistic.

3 What about if you totally eliminated any comment
4 about sodium and let's say the levels doubled in the
5 population because for whatever reason people no longer paid
6 any attention to it, and you were taking in 12 grams a day
7 or 13 or 14 grams like some populations? Is there any
8 evidence that that would be harmful?

9 DR. KUMANYIKA: I think there is -- I mean, we
10 would have to follow the population, but the presumption is,
11 from the evidence associating from salt data and those kind
12 of comparisons, and from the ability to reduce the incidence
13 of hypertension and lowering sodium in people with high
14 normal blood pressure, the assumption is that more people
15 would be at a high sodium intake and then more of those who
16 are predisposed would convert over into hypertension at an
17 earlier age and so forth.

18 So the assumption is that it goes both ways, that
19 you could say in the similar way that the weight of the
20 population increased. One of the problems in the blood
21 pressure literature is that blood pressure in
22 pharmacologically controlled in the churn data, and that we
23 did a paper trying to model the impact to see if the
24 incidence of high blood pressure is actually going up, and
25 that we just suppressing it with medication, and the only
26 way we could do that was to impute the people who were on
27 meds, to put them in the high part of the distribution and
28 then look at it churn over time.

29 But that's one of the problems, people -- because

1 the treatment -- well, see, and for blood pressure about 25
2 percent of people who know they have blood pressure and
3 taking medications have blood pressure in the desirable
4 range, so that's another like 50 to 70 percent of the adult
5 middle age population who might have a harder time
6 controlling their blood pressure if they were to be
7 consuming more sodium.

8 This is such a passive issue from the point of
9 view of consumers because you saw the discretionary data.
10 It's not that convincing. Now, this is something consumers
11 are doing, but this guideline is always a little bit
12 circuitous because the message is really to the say catering
13 industry or some of the people who feel that consumers want
14 this amount of sodium and therefore put it in their food,
15 not because consumers want it, and I don't know what you do
16 with that, but at least this is a platform for working with
17 industry on how to keep it at a moderate level.

18 CHAIRMAN GARZA: Alice?

19 DR. LICHTENSTEIN: I have two comments about the
20 guideline itself.

21 First, right now it says "Choose a diet matureate
22 in salt and sodium." And I'm wondering, I didn't look at
23 the consumer focus group data on that, but what is the
24 perception, you can't do one without the other as far as
25 salt and sodium, and, you know, historically were they
26 always both -- those both terms included and then is that
27 confusing or is that actually helpful?

28 DR. KUMANYIKA: My impression is that
29 historically, when it was sodium, that nobody had any idea

1 what that meant. So you have to say "salt" because people
2 don't --

3 DR. LICHTENSTEIN: Right, but then is it necessary
4 to say "sodium" or is it confusing just as far as the
5 message goes?

6 I mean, people don't eat sodium. They do eat
7 salt, and salt is listed on the ingredient label, not
8 sodium. So I think that's something that should be
9 considered.

10 DR. KUMANYIKA: Sodium. On the ingredient list?

11 DR. LICHTENSTEIN: Yes. But on the nutrient
12 label, I guess, it's different.

13 DR. KUMANYIKA: Unless it's another sodium
14 compound.

15 DR. LICHTENSTEIN: Right, yeah.

16 The other point is right now the way the guide --
17 the commentary to the guideline is written it says,
18 "Consuming more fruits and vegetables also increase
19 potassium intakes which may help to reduce blood
20 pressure." That sort of comes out of the blue, and then
21 there is a box that talks about some good sources of
22 potassium.

23 You didn't mention potassium, but I'm wondering,
24 given that we have a limited amount of space in the booklet,
25 is that particularly useful to the consumer with respect to
26 the guideline?

27 CHAIRMAN GARZA: Is that a rhetorical question or
28 is that what you wish us to say?

29 DR. LICHTENSTEIN: I guess I'm really interested

1 because, you know, being either a normal or abnormal
2 consumer, I'm a little confused.

3 DR. JOHNSON: Maybe when we promote fruits and
4 vegetables, and as the effect of the DASH diet, you know,
5 you could say something about blood pressure in the fruit
6 and vegetable part. That's where your potassium --

7 DR. LICHTENSTEIN: But we need information. I
8 mean, I didn't hear anything about potassium. Do you think
9 that potassium should be here and then the box should be
10 devoted to potassium-rich foods as it's written?

11 DR. KUMANYIKA: Yeah, I can, and maybe we didn't
12 have anybody we thought would be the best to invite this
13 time to talk about some of these issues, but from this
14 discussion maybe we will come up with the right person for
15 next time.

16 The blood pressure literature makes it hard to
17 distinguish between whether it's sodium itself or the sodium
18 potassium ratio, but potassium doesn't come out as standing
19 on its own as a risk factor, and potassium supplementation
20 seems to -- they do more in certain populations if potassium
21 intake is low; like African-Americans have low potassium
22 intake, and there have been couple studies showing that
23 supplementation will help, also the same with calcium.

24 So this has been the holding place, just like
25 calcium is mentioned here and there and other reasons, this
26 has been a place for mentioning factors that don't stand
27 alone, and that's why I was thinking that maybe we would
28 change some wording about sodium in the diet, and the
29 committee hasn't had a chance to discuss this, but there may

1 be a better way to do it because it does sound silly if you
2 don't know why it's there, and you don't know the
3 sodium/potassium ratio literature, it looks like --

4 DR. LICHTENSTEIN: No, no, no. I know some of the
5 literature and I know there have been reports that there is
6 a relationship. I'm trying to put myself in the shoes of
7 just a regular old consumer, and it's sort of buried in a
8 sentence. I'm looking at a guideline that's referring to
9 salt and sodium, and what I see as a visual in a box which I
10 might just focus on is potassium, and I'm just wondering how
11 much of an impact that actually has because it's not part of
12 the guideline, which was maybe to, you know, read it, so
13 that's why. There may be a good reason for it but I think
14 it's a good question.

15 CHAIRMAN GARZA: Richard and then Roland.

16 DR. DECKELBAUM: Actually, just to -- the
17 potassium box is the first box you see in the guideline.

18 DR. LICHTENSTEIN: Yes.

19 DR. DECKELBAUM: I wasn't going to bring that. I
20 was going to suggest that quantifying, I guess that would be
21 a number.

22 (Laughter.)

23 CHAIRMAN GARZA: I hope that's not a reflection of
24 our glycemic index now.

25 (Laughter.)

26 DR. DECKELBAUM: But from what I understand, and
27 correct me, that the dietary guidelines have been food-based
28 and not number-based, and use numbers when other may other
29 organizations provided them, much like it's used in the

1 current guideline. I think quantifying, just, you know,
2 putting it in as a number is just going to give a lot of
3 area for controversy because you're going to have to spend a
4 lot of time finding that exact number and, you know, maybe
5 the -- so I would let the DRI committee or other committees
6 worry about it, but we don't -- I don't think we have to
7 worry about a number in our verdicts.

8 CHAIRMAN GARZA: Okay, Roland.

9 DR. WEINSIER: As it comes across now, it seems
10 that sodium is punitive primarily for risk of hypertension,
11 and I wonder if your committee has or would want to consider
12 including here the relationship of sodium intake to calcium
13 loss and bone health.

14 I think that there is an accumulating body of
15 literature -- did I miss it?

16 DR. KUMANYIKA: It's in the guideline, yeah, in a
17 sort of catch-all category.

18 CHAIRMAN GARZA: Okay, I'm going to take one or
19 two questions. Let me tell you what the plan is before
20 everyone decides what they need to say needs to be said now.
21 We're going to take the next guideline before we break for
22 lunch, and we're going to be back by 1:15.

23 (Laughter.)

24 Otherwise, we're not going to get through what we
25 need to do this afternoon, to make sure that the groups meet
26 in the afternoon and start wrestling with these issues,
27 because we have to come back tomorrow and deal with outlines
28 and begin to make your work more concrete.

29 So as long as everybody is aware of what you will

1 be doing to your lunch break, then we can proceed.

2 VOICE: Can we get sandwiches or something brought
3 in?

4 CHAIRMAN GARZA: And work through lunch?

5 VOICE: Yeah.

6 CHAIRMAN GARZA: Yes, that would be a great --
7 because when we can just break for 10 minutes or five
8 minutes and have lunch brought in.

9 Is everyone agreeable to do that?

10 (Chorus of ayes.)

11 VOICE: We heard you, we heard you.

12 CHAIRMAN GARZA: All right, good. Let's just plan
13 to do that.

14 VOICE: And it's still snowing out.

15 (Simultaneous conversation.)

16 CHAIRMAN GARZA: All right, let me ask or someone
17 in the staff if they can bring in a menu or Shanthy
18 something, then we'll just pass the menu around.

19 VOICE: Give us two choices, give a variety.

20 DR. LICHTENSTEIN: I would just say one thing
21 about if the subcommittee is considering a number, just
22 remember that the guidelines are for individuals two and
23 above, so with the fat it's 20 percent of calories, but
24 little people may -- it may be more appropriate for them to
25 have higher or lower total amounts of sodium than big
26 people.

27 CHAIRMAN GARZA: Any other comments?

28 Linda?

29 MS. MEYERS: The salt and sodium, you're quite

1 correct that the -- when salt was added, it was because
2 nobody understood sodium. Sodium wasn't deleted only
3 because it was on the food label, and I think probably it
4 had an hour's discussion, so it wasn't based on a lot of
5 analysis about what people would understand.

6 DR. LICHTENSTEIN: But do we have -- did any of
7 the focus groups deal with that?

8 MS. MEYERS: I can find out. I don't know the
9 answer to that.

10 CHAIRMAN GARZA: Carole?

11 DR. LICHTENSTEIN: Connie is nodding yes.

12 CHAIRMAN GARZA: Connie, can you comment a bit
13 more than just yes?

14 VOICE: The majority of the consumers do not know
15 the difference between sodium and sodium chloride. They
16 don't understand why they are put there. Some, very sadly,
17 consumers --

18 CHAIRMAN GARZA: Okay.

19 DR. LICHTENSTEIN: I would like to ask Constance
20 about added sugar as long as we have her up.

21 Do you know if there is consumer data on their
22 perception of added versus the term "sugar".

23 VOICE: We did not in our, our study ask that.

24 CHAIRMAN GARZA: Okay. There are a number of
25 others that have questions and I may have stifled them. All
26 right, caloric restriction may be a very adequate strategy
27 for the next guideline and we're -- so Dr. Weinsier here,
28 would you like to go on then and over on to the next one?

29 DR. WEINSIER: Since the attention span is --

1 CHAIRMAN GARZA: You need to either -- there
2 should be either a --

3 DR. WEINSIER: Is that on now? Yeah.

4 Okay, I think I can shorten this considerably.
5 Some of the issues that were raised, before I get into this
6 category, and I say "raised" by some people that spoke
7 yesterday, letters that we've gotten, comments made within
8 and outside of our committee, basically focusing on four
9 areas of concern or suggestion. One was the title; second
10 was the etiology of obesity, and should we address this in
11 this guideline; third, the methods, methods for assessment
12 of body fat and methods for assessment of body composition
13 or body fat distribution; and, fourth, emphasis on physical
14 activity.

15 Basically, the working group consist of Rachel
16 Johnson, Shirika, Joan, Kathryn, myself. The general
17 considerations in our working model was to look at new
18 evidence 1995, focus on general concepts to be emphasized,
19 recommendations should be appropriate for the population at
20 large rather than too prescriptive, and this is a though one
21 for us, and then information should obviously be practical
22 for dealing with the public at large.

23 In terms of the first issue and concerns that were
24 expressed, the title has raised a number of questions. this
25 is one possibility, but others have been suggested. The
26 previous title was "Balance the food you eat with physical
27 activity, maintain or improve your weight." Apparently the
28 word "improve" in focus groups has given people pause and
29 concern because apparently the definition of "improve" is

1 not clear. Does that mean gain weight? Or other issues
2 have been raised, but it was not clear.

3 A second issue that's come up, and I'm not sure
4 it's addressed in this proposed title, is the word "Balance
5 the food you eat with physical activity." The implication
6 for some has been, well, I'm heavy, but I'm balancing the
7 food I eat with my activity, so I'm doing what I should do
8 when it should be "imbalanced" at that point. So I'm not
9 sure that this deals with it, but we're open to suggestions.

10 One that has been proposed is more focused, simply
11 "Achieve a healthy body weight." The word "healthy," our
12 committee seem to fee fairly comfortable as a word we might
13 want to have repeatedly within the guideline.

14 In terms of areas of new evidence, actually I was
15 going to go into some detail on this, but I'm going to skip
16 probably a fairly large number of overheads and discussion
17 and simply come back and give you a feeling here of one way
18 we might want to deal with this.

19 But the other things we need to look on, I think
20 there are new data that we need to consider in terms of
21 methodologies for measuring percent body fat or body fat, as
22 well as distribution. We need to look at the relationship
23 of fatness; the fat pattern medical risk, we have new
24 information here; the guidelines, as before, who should
25 lose weight probably should be retained. In terms of weight
26 loss goal, we may want to rethink this a little bit in terms
27 of weight loss approach, eating pattern and physical
28 activity may just need some tweaking and not major changes.

29 With regard to the first, that is, the etiology,

1 just allow me to skip way ahead, and if you really feel
2 strongly, I'll come back and discuss these. I think it's a
3 very important area, but it may not be necessary for our
4 discussing right now.

5 The reason they came up, at least as I understand
6 that it came up is that there is concern that the public has
7 a feeling that with so much emphasis on the genetics of
8 obesity certainly in recent years, since '95 when the OB
9 gene was discovered, that there is a perception that it is
10 now really out of our control for many of us. It's in our
11 genes or it's not in our genes. If it is, then it's not a
12 modifiable factor. This was raised -- this concern was
13 raised to me by Dr. Goldman, the editor of the Annals of
14 Internal Medicine, just several weeks ago when he expressed
15 concern that of his 100,000 readership, he feels strongly
16 that a vast majority of them -- these are medical folks --
17 feel that obesity is determined by genes and therefore it's
18 not modifiable.

19 So jumping past all the data on one side and the
20 other, one thing we might consider on the basis of my
21 understanding of the literature is that there are non-
22 modifiable genetic influences. I mean, this is absolutely a
23 given. I think the data is absolutely solid that there are
24 genetic influences and there are certainly variations of
25 energy requirements, which you can say are inherent, perhaps
26 genetically determined, plus we have the changing
27 environment and these pose challenges for weight control.

28 But it still appears that for the general
29 population weight changes are determined, underline the word

1 "determined," not by our genes or environment, but the
2 causative or cause/effect relationship is based upon the
3 behaviors. And so I think we have the flexibility and
4 should try to encourage the readership of the guidelines
5 that it may be difficult, but we still have modifiable
6 behaviors that are still under our control, so you may want
7 to come back and discuss that, make suggestions to our
8 committee, but that's the approach that we're currently
9 taking.

10 Methods to assess body fatness, we do have some
11 new guidelines. The BMI seems to be useful for categorizing
12 degrees of obesity and the association of health risk.
13 Proposed recommendations based upon the WHO and the NIH
14 reports, the NHLBR report suggest these guidelines for
15 normal. They used the term "normal." We suggest the
16 possibility of substituting "healthy weight" as the BMI,
17 these numbers proposed overweight, the number here proposed,
18 and obesity greater than 30 kilograms squared.

19 It's also been suggested the NHLBR guidelines,
20 that the same BMI cutoff for obesity is justified for all
21 gender and race groups.

22 So our suggestive revision is, without showing you
23 a picture of it, but the last, the '95 guidelines based on
24 weight for height, it was like several graded areas that you
25 could trace your weight for your height. One consideration
26 is to perhaps use the BMI, the Body Mass Index, rather than
27 weight for height based upon the newer reports.

28 With regard to health risk and the other
29 parameter, assessment of body fat pattern, the BMI appears

1 to be associative of increasing health risk in a graded
2 fashion, so we have to be careful that when we talk about
3 BMIs and cutoffs that the interpretation is not that if my
4 BMI is 24.9, I am perfectly healthy. If mine is 25.1, I've
5 got real problems; that it is a graded association.

6 Waist circumference appears to be an independent
7 risk factor, at least up to actually a BMI of 35, for
8 various disease, including cardiovascular disease, diabetes,
9 hypertension. So there is good reason to consider in the
10 guidelines giving the public information about how to assess
11 waist circumference as well as BMI.

12 Since body fat pattern is determined in part by
13 modifiable risk factors, such as exercise reported in this
14 study in '95, waist circumference is to some extent
15 modifiable, so this has to be taken into account. If it's
16 not modifiable, maybe we shouldn't push this on the public.
17 But on the basis that it may well be modifiable, then we
18 should consider focusing on waist circumference as another
19 measure.

20 In contrast to BMI, waist circumference cutoffs
21 differ between genders, so this does have to be taken into
22 account. So in our revision we may want to consider using
23 the waist circumference -- previously it was the weight to
24 height ratio -- excuse me, waist to hip ratio -- consider
25 using waist circumference as a reference replacing the WHR.

26 And we might want to consider a table such as this
27 or a box such as this. It's based upon the NHLBI clinical
28 guidelines report. We might want to substitute the word
29 "healthy" for "normal" BMI. We may want to consider

1 rounding off a number here to make it consistent, but these
2 are things that need to be discussed. But basically the
3 point is that to figure out -- to estimate your weight
4 status and risk of disease, to give some guidance in terms
5 of what your BMI is, how to measure it, and where the waist
6 circumference fits in to figuring out your relative risk of
7 disease.

8 And in terms of controlling weight, who should
9 lose weight, it's important that, as in the previous
10 guideline, that we emphasize that not everyone, you know,
11 has to lose weight. And the BMI is greater than or equal to
12 25, that in itself may not be an indication that medically
13 the person needs to lose weight. However, if the BMI is
14 greater than 25, the data do suggest that we consider
15 looking for a BC-related considerations. As well, if the
16 waist circumference is greater than 88 in women or greater
17 than 102 in men, we look for obesity-related conditions.

18 Our suggested revision maybe include a reference
19 table about who should lose weight and try to emphasize the
20 distinctions of who should and who should not lose weight,
21 and I won't read all of this now, but this is an example of
22 what a box might look like in terms of who should lose
23 weight. The main point, regardless of the wording, is that
24 you may not need to lose weight even if you are in the
25 overweight category, i.e., a BMI greater than 25, or between
26 25 and 29.9, or even if your waist circumference has
27 increased, i.e., greater than 35 for women or 40 inches for
28 men, however, risk factors should be assessed and then
29 perhaps have some guidelines for who should consider losing

1 weight based upon BMI, waist circumference, number of risk
2 factors.

3 Now, in terms of how to go about it, i.e., what
4 are some reasonable guidelines for the weight control or
5 achieving a healthy weight, the previous guidelines
6 emphasized the five to 10 percent weight loss. By weight
7 loss of five to 10 percent can reduce but may not eliminate
8 co-morbid conditions.

9 One thing to consider and this is, in part, based
10 upon the NHLBI report, but also more recent, the CLEM
11 report, looking at the registry of individuals who have lost
12 significant amounts of weight and kept it off for an average
13 of five years, is that medical risk is assumed to be
14 maximally improved by achieving a normal body weight. So if
15 we're talking about maximal improvement, then the guideline
16 should be achieve a normal body weight. Thus, the ultimate
17 goal might be to achieve what we're calling a healthy or
18 normal body weight rather than leaving the impression that
19 five to 10 percent takes care of it. It's a good initial
20 goal, but we need to think about the wording and consider
21 whether we want to emphasize normal weight because it's a
22 goal that most people will probably not achieve.

23 Does that mean it should be taken out of the
24 guidelines or do we want to leave it in? Weight reduction
25 can improve health, the ability to function and quality of
26 life for overweight or obese individuals at any age, so age
27 is a consideration to think about in this guideline,
28 certainly for the elderly population or the older adult
29 population.

1 The suggested revision, five to 10 percent, is an
2 appropriate initial goal, ultimate goal, a healthy body
3 weight is something we may want to consider.

4 In terms of dietary pattern for weight control,
5 there are some data that have been published since 1995 that
6 we probably want to consider. They come out -- they come
7 from the same group. Bell and Barbara Rolls are both at
8 Penn State. But these studies are meticulously conducted,
9 at least in my view, and are compelling in terms of short-
10 term control of energy intake. Anyway, the data suggests
11 that energy intake is determined more by the volume and the
12 weight of the food consumed rather than the fat content.
13 these studies, as I say, were very carefully conducted.
14 Lower energy intake occurs spontaneously with use of the
15 lower energy dense food. Reducing dietary fat alone without
16 reducing calories is not sufficient for weight loss.

17 So just taking account of some of this data, most
18 of it new, some of it not so new, we may want to consider
19 the possibility of giving guidelines that relate to the
20 foods at the base of the pyramid; that is, the whole grain
21 foods, the plant foods, vegetables and fruits, which are
22 going to be relatively low in energy density, and at least
23 in the short term, seem to be a primary determinant of
24 energy intake.

25 Above findings are consistent with emphasis on
26 intake of foods at the bottom of the pyramid, as I say,
27 which are low in energy density and fat content.

28 Suggested revisions: The 1995 guidelines are
29 primarily focused, at least in my view, on negative advice.

1 There were six of seven on one box given, six of seven at
2 the advice statements were negative in the sense that
3 they're saying "eat less....", "eat smaller...", "eat
4 without...", it's all things that we should not be doing,
5 and I'd like to think through, if it's okay with the
6 subcommittee and the entire committee, on ways to emphasize
7 positive statements, perhaps by putting the emphasis on the
8 majority of intake being derived from minimally processed
9 whole grains and cereals, vegetables and fruits at the base
10 of the pyramid, enabling advice for weight control which is
11 positive as well as appropriate for overall health. So it's
12 a consideration I'd like for us to remember.

13 And, finally, with regard to physical activity,
14 physical activity benefits only modestly the weight loss.
15 So in terms of inducing weight loss, I think it's pretty
16 well documented it's not going to contribute a great deal.
17 It does, however, improve cardiovascular fitness and it
18 certainly appears to improve weight loss maintenance, may
19 have a specific effect on decreasing abdominal and inter-
20 abdominal fat.

21 Individual's success with long-term weight
22 control, according to the Weight Loss Registry, have
23 physical activity levels that are about three times that of
24 the American College of Sports Medicine Recommendation. So
25 those individuals who seem to be successful in achieving a
26 large amount of weight loss and maintaining it for a long
27 period of time seem to be more active than would be
28 recommended by the American College of Sports Medicine.
29 In older adults, preserving strength is particularly

1 important to reduce risk of falls and fractures.

2 So the bottom line, in terms of suggestive
3 revisions we might want to consider, physical activity is an
4 important part of a weight control program, being highly,
5 and I highlighted the word "highly," highly active favors
6 long-term success in overall health. We need to be thinking
7 about where and how to emphasize "highly," not to mislead
8 people that just becoming a little more active may
9 healthier, may be more helpful, but, again it's like
10 achieving a healthy weight versus a five to 10 percent
11 intake. We try to set a goal a little bit higher which may
12 not be realistic for most.

13 So that's basically the comments that I have based
14 on the input from the committee. But, Shirika, Rachel,
15 please jump in.

16 DR. JOHNSON: I just wanted to add that we did
17 talk about children as well, and I think it's something we
18 definitely need to include with obesity increasing at the
19 rate that it is among American's children. And I know we
20 talked about some behavioral things, primarily based on
21 Leanne Burges' work on what happens when you coerce or force
22 children to eat, and the negative impact that can have over
23 the long term. So I'm hoping that we're going to be able to
24 put some behavior tips for parents and caregivers related to
25 children and really emphasize physical activity in regard to
26 the pediatric population.

27 CHAIRMAN GARZA: Other comments or questions?
28 Meir?

29 DR. STAMPFER: Yeah, I thought that was a really

1 thoughtful and measured set of recommendations. I just had
2 a few comments.

3 One was, in terms of the diet composition that
4 promotes or doesn't promote weight loss, those studies, I
5 agree, they're careful, but they are very short term. They
6 are really based mostly on meals in terms of the energy
7 density, and I think you can gain weight on just about
8 anything that has calories, as long as you don't balance
9 them, and I don't think the evidence for energy-dense foods
10 as especially promoting weight gain or being against weight
11 loss is very compelling.

12 My second point I think may be more important, the
13 notion about should you try to lose weight when you're over
14 a BMI of 25. I think our emphasis should be on prevention
15 of these conditions, like blood pressure, adverse lipids and
16 diabetes, and so I don't think we should tell people to wait
17 until they have an adverse condition and then try to lose
18 weight. It's much harder to lose weight than it is to
19 maintain weight. So I think we should stress prevention,
20 not waiting until these things.

21 I think just about anybody over 25, unless they
22 are in a very unusual body builder type with a big muscle
23 mass, is probably going to need to lose weight, and I think
24 we should stress that there are exceptions, but most of
25 those people by far should be losing weight, and they'll
26 improve their lipids and their glucose tolerance and their
27 blood pressure even before they reach a critical level where
28 they need treatment.

29 Likewise, even people who are within, sort of at

1 24.8, some of those people ought to be losing weight too,
2 depending on their lean body mass and so on, and I think
3 there should be more emphasis on weight gain during --
4 trying to avoid weight gain during adult life. if you've
5 gained weight from say age 20, on the average you're
6 overweight, and the -- for most people the weight that
7 you've gained is adipose.

8 So those are my two main comments.

9 CHAIRMAN GARZA: Alice?

10 DR. LICHTENSTEIN: I have a comment on the wording
11 of the guideline. It seems like the important point is to
12 maintain or improve your weight. I'm wondering, there is a
13 lot in that guideline, and, again, going back to the focus
14 groups and what we know about perception, balance of food
15 you eat with physical activity, as pointed out in the
16 beginning, is a -- I mean, it can be interpreted a lot of
17 different ways. I'm wondering if we consider just the --
18 you know, consider the wording, and that perhaps if the
19 basic, the most important message is to maintain or improve
20 your weight, that's what the guideline should say, and then
21 the text should be more explicit regarding how to do it.

22 CHAIRMAN GARZA: Johanna?

23 DR. DWYER: Just two, two points.

24 The first is it would be helpful, I think, to
25 include instead, if we're worried about space, a little
26 formula to calculate BMI from inches and pounds, because
27 it's clear that we're not going that track. It's also clear
28 that most people do not know how to calculate this, and so
29 it becomes a magic, yet another magic medicalized thing that

1 we tell people.

2 The second thing is is the issue of the BMI drift,
3 and the problem, certainly the same BMI for all gender and
4 age groups may be appropriate for adults, but it's
5 certainly, I don't think you would want to be including
6 children in that BMI, would you?

7 Perhaps you would, but I don't think so from what
8 I know serum cholesterol values at the 85th and 95th
9 percentile are different and the believe the VMIs are too.
10 So I think you need to reconsider that for kids over the age
11 of two but under the age of 19.

12 I also would like to endorse Meir's suggestions
13 with respect to the prevention. In terms of the treatment,
14 we really do have to realize that where we are with this
15 after 50 years is still with cure rates that are about the
16 same as cancer of the stomach, right? Ten percent cure
17 rates at five years. So we need to be, I think, modest in
18 our promises to people about what we can do.

19 CHAIRMAN GARZA: Richard?

20 DR. DECKELBAUM: Just one point, I think, that
21 will help in terms of the genetic contribution of the
22 argument. I don't think the gene pool in the United States
23 has changed that much between NHANES I and NHANES III.
24 Obesity is doubling and tripling in some populations and
25 that's certainly a major argument, that there has been some
26 other change besides genes.

27 I think if we look where we're going with the
28 different risk factors and chronic diseases, this is the one
29 where we have the greatest failure. I think I'm right,

1 Paul, in that statement. And therefore, with the knowledge
2 of coma morbidities and even obesity as an independent
3 factor, it's the one that I really think as a group we must
4 focus on in a very integrated fashion throughout the
5 guidelines because it represents the greatest failure of,
6 you know, risk factor control that's actually happening now
7 in the United States.

8 And in doing so we have to have, I think, very
9 careful integration throughout the related guidelines, and
10 somewhere, either in the introduction or a variety or
11 wherever it's going to be, in terms of, you know, pulling
12 all the data together that we're hearing, we're hearing some
13 of it. You know, if the science base is out there that kids
14 who have a lot of sugar at lunch are going to eat more later
15 on in the afternoon with their snacks and during the day, we
16 really must gather that kind of evidence wherever we can,
17 and we -- even -- we've really got to, I think, take this
18 factor in terms of appetite control, because this is what
19 we're really talking about, it's appetite control, and one
20 of the factors that contribute to appetite control, and
21 we've had very little of that at this meeting, but I think
22 it's something I think we should be considering in our
23 deliberations, and perhaps we can get someone to help us on
24 that because we really -- there are people who know a lot
25 about appetite control, but we have, except for very little,
26 we've had very little discussion on it, but that's a major
27 factor in the number of calories is how do you control your
28 appetite.

29 CHAIRMAN GARZA: Do you know of any data that

1 relates -- measures appetite control to clinical outcomes?

2 I know the theory, but is it -- getting back to
3 Meir's question, I mean, do we have any data that would give
4 us any sense that this is related?

5 DR. DECKELBAUM: I don't know. Again, this isn't
6 my major field. There may be some things out there, but if
7 you can just take -- take the big increase in soft drink
8 consumption, which does have sort of simple sugars in it,
9 and if there is some data on simple sugar consumption and
10 appetite later during the day, it's something we should be
11 considering even if -- even if the direct link isn't --
12 hasn't been made yet. Certainly if you look at cholesterol
13 and heart -- cholesterol levels and heart disease, the
14 initial links were made before the data was in, showing that
15 lowering cholesterol, in fact, lower risk of heart disease.

16 CHAIRMAN GARZA: But the point I thought Meir was
17 making is that the data are in there, at least people have
18 looked in terms of looking for diet composition. I mean,
19 they've looked for fat, they've looked for carbohydrates and
20 not been able to link it because, in fact the idea is that
21 you somehow compensate the following day. Is that --

22 DR. STAMPFER: My read of the long-term data is
23 that diet composition isn't a terribly important predictor
24 of weight gain or weight loss. But there are data on
25 satiety and how you feel after a meal.

26 Now, linking that to obesity is logical, but I'm
27 not aware of the data that's done that.

28 CHAIRMAN GARZA: is it because of a lack of
29 then -- do you compensate the following day, and that's the

1 dynamic that we don't have a good handle on.

2 DR. GRUNDY: I wanted to say something about this
3 eating out of the base of the pyramid. Let's say the
4 pyramid, you know, if all of our food came in the form of
5 little pyramids like Hershey's Kisses, and that's the way
6 you said it, we'd just take a bite out of the bottom of
7 those and throw away the top. And I think what I think we
8 ought to do is to eat the whole thing, eat the whole
9 pyramids, but just eat less of them. It wouldn't be eating
10 the total diet, so I have a problem with that concept.

11 DR. WEINSIER: I may have misstated. I did not
12 intend to say that the foods selected should be limited to
13 the base of the pyramid. I didn't intend to say that. I
14 was trying to imply --

15 DR. GRUNDY: I mean, the pyramid should be
16 structured so that you eat the whole pyramid, right? That's
17 what I'm saying?

18 DR. WEINSIER: Well, I look at it as a guideline
19 to food selection. So if an individual is going through a
20 smorgasbord line and they have to make choices, that in the
21 back of their mind they've got this image of a pyramid
22 where, I guess, most of the food on my plate should come
23 from the grains, the fruits and vegetables, and then I have
24 the option to choose some additional foods from the protein
25 group, from the dairy group. So you've got some reference
26 point at what to put on your plate as you're going through
27 the cafeteria line.

28 Does that make sense?

29 DR. GRUNDY: I have a feeling that in your case

1 you'd never get to the top of the pyramid when you went
2 through that line the way you describe it. I'm not saying
3 that's bad, but I think our pyramid ought to be constructed
4 in a way that most people eat the whole pyramid.

5 DR. WEINSIER: Well, that's what I said, I didn't
6 mean to intend --

7 DR. GRUNDY: Yeah.

8 DR. WEINSIER: -- to exclude any food because I
9 don't practice that when I treat patient.

10 CHAIRMAN GARZA: Before we continue with a
11 discussion, let me tell you what's being passed around. You
12 have the menu. Please put your initials by whatever you
13 want. Then I want to reassure the transcriber that we are
14 going to take a 20-minute break at the end of this
15 discussion and that will give everybody a chance, give them
16 a chance to rest up a bit and you a chance to stand up and
17 stretch. But I want these filled out, otherwise, you may
18 not get your food in a timely manner. So do that as we
19 speak, okay?

20 Johanna, then Suzanne.

21 DR. DWYER: Richard, just back to your intriguing
22 remarks. I was trying to remember, and perhaps someone in
23 the audience or at the table knows of the study. This was a
24 study that we done at your institution in the early 1970s by
25 a wonderful investigator who died prematurely by the name of
26 Kathy Porocoust, and she did this study with Tadbee in
27 Italy, and it was one of the few, as I remember, long-term
28 studies of regulation of food intake over months rather than
29 weeks.

1 Barbara Roll's study, as Dr. Stampfer has pointed
2 out, are acute studies. They are not as long as that.

3 And as I remember, the results were not very
4 compelling. The results were compelling but the effects
5 were very small. But I may be wrong and it's in your
6 library some place.

7 CHAIRMAN GARZA: There are the Labinsky studies as
8 well for three months, and there are some longer term
9 studies.

10 DR. JOHNSON: I just had a couple of points. I
11 wanted to follow up on Johanna and Meir's plea for an
12 emphasis on prevention. I agree with that, and I think what
13 I would like to see us really emphasize is physical activity
14 and some behavioral tips that are commonly used in
15 behavioral weight control programs, and with some particular
16 emphasis on portion sizes.

17 I think we also need to be careful about the
18 definition of "healthy," because if you recall when the NIH
19 guidelines came out they were broadly misinterpreted in the
20 media to say that anyone with a BMI over 25 needed to lose
21 weight, and that's not what they say. They say a BMI
22 between 25 and 29, with two additional risk factors. There
23 is going to be a lot of debate about what is healthy if we
24 come out and define that.

25 I mean, if we're going to define over 25 as not
26 healthy, we need to be on very firm footing, I think.

27 Shirika, you were on that NIH panel, weren't
28 you --

29 DR. KUMANYIKA: Um-hmm.

1 DR. JOHNSON: -- for the weight guidelines, so
2 maybe you could help us out there.

3 CHAIRMAN GARZA: Hold on, let me ask -- let me ask
4 Suzanne because she's been trying to get in a comment.

5 DR. MURPHY: If it's directly related to what
6 Rachel just said, go ahead.

7 I too want to chime in about physical activity. I
8 think that is the biggest purpose of this guideline, and all
9 the weight discussion in the world should take second place
10 to focusing on physical activity.

11 I also wanted to ask maybe somebody who knows more
12 about the chart than I do that's in Figure 3, I had always
13 thought that was calculated based on a BMI of 25, 30, et
14 cetera. Is that true?

15 And in that case, isn't that much easier for
16 consumers than giving them a formula? Why wouldn't you just
17 keep Figure 3 the way it is?

18 MS. MEYERS: It was actually calculated with a
19 simple Exell spreadsheet.

20 CHAIRMAN GARZA: If you track back, it comes out
21 to the same BMIs, is that what you're saying, Linda?

22 DR. KUMANYIKA: What did you think it was based
23 on? I notice when you were saying, I had the feeling that
24 you thought this was based on something?

25 DR. MURPHY: He said height for weight.

26 DR. WEINSIER: Yeah, weight for height, but not
27 weight of height squared.

28 DR. MURPHY: Okay.

29 DR. LICHTENSTEIN: That last two guidelines have

1 basically taken BMI cutoffs and converted them to weight for
2 eight squared for the table.

3 DR. MURPHY: I think anything we can do to
4 simplify these things for the readers is great. And along
5 that line, there is also a statement about if their waist is
6 bigger than your hip, you need to worry. I like that sort
7 of easily implemented approach. It may not be theoretically
8 quite a correct as giving them the centimeters, but it sure
9 is a lot easier for people to understand.

10 DR. STAMPFER: I just wanted to respond to Rachel.
11 I think, you know, for a cutoff of 25, I think that's
12 basically what the current WHO cutoff is as defining a
13 desirable weight. And I think there is just plenty of data
14 to support that. I don't think we're on thin ice at all to
15 use a 25 cutoff. There is just lots of data out there for
16 diabetes and blood pressure and other risk factors,
17 mortality. I think we're quite comfortable on that.

18 DR. KUMANYIKA: I didn't hear all of your comment.
19 You asked me -- what were you suggesting instead?

20 DR. JOHNSON: Well, when the NIH guidelines came
21 out, my understanding of the interpretation is they don't
22 say everyone with a BMI over 25 needs to lose weight; that
23 between 25 and 29, they say with two additional risk
24 factors.

25 Am I not interpreting that correctly?

26 DR. KUMANYIKA: NO, it -- but it says that they
27 are overweight, and it says that that's a window of concern
28 and that that's a -- should be trying not to gain anymore
29 weight. Those are clinical guidelines meant for people to

1 really talk to individuals, and what's factored in there is
2 that the person may not want to lose weight. And so I think
3 that I agree basically with the 25 mention there because it
4 is first level of concern about weight. It's just that what
5 a clinician does with that person may vary depending on
6 their --

7 DR. JOHNSON: See, you're comfortable with saying
8 that everything over 25 is not a healthy weight. If you say
9 anything under 25 is healthy, the implication is that
10 anything over 25 is not healthy.

11 DR. KUMANYIKA: Well one of the DJAC report from
12 1995 also had a list of BMI range, which was based on BMI
13 25, and all that made it into the final booklet was the
14 chart, but this chart is based on a BMI of 25, which was
15 somewhat controversial at the time for people who had
16 noticed it, and then it showed the gradation, and probably
17 not on -- I'm not sure now, Linda probably knows where the
18 shading changes, if it changes at 32 or 30.

19 But I think the point was that, yeah, once you're
20 over 25, you should pay more attention to your weight than
21 below, than below 25, so I would change it from 25, but also
22 change the 1995 dietary guidelines.

23 CHAIRMAN GARZA: Johanna.

24 DR. DWYER: Just two things.

25 One is i didn't hear anything with aging, so it's
26 going to be flat for aging.

27 And secondly, I don't think we -- something needs
28 to be said about the appetite suppressing drugs. One is on
29 the market, one was withdrawn from the market, and a third

1 is hoping again to go on the market. It seems to me to
2 simply ignore it, you can't.

3 CHAIRMAN GARZA: All right. On that rather
4 controversial note.

5 DR. LICHTENSTEIN: Well, I would point out those
6 are prescription drugs.

7 DR. DWYER: Right.

8 DR. LICHTENSTEIN: So it's not that it -- so there
9 are other issues.

10 Just on the comment that Rachel made. Desirable,
11 healthy, what's the word? Because if it's a desirable
12 weight, then it doesn't sound so bad if you're above or
13 below 25.

14 DR. JOHNSON: Well, I just know we will get
15 arguments. I mean, if you want controversy, we'll get
16 arguments from people who say that if your blood pressure is
17 normal and you don't have diabetes, then all these other
18 things, and your BMI is 27, are you unhealthy.

19 CHAIRMAN GARZA: Scott.

20 DR. GRUNDY: I think we have to be quite careful
21 about that BMI of 25. You know, when that NHLBI guideline
22 came out, we got a tremendous amount of flack about that
23 from people who were saying that a lot of healthy, young men
24 have BMIs above 25 and they're not overweight and are not
25 obese, and that is very true.

26 And my argument in defense of that was that was
27 like you said, Shirika, a clinical guideline, and it's one
28 in which physicians can look at the patient and say
29 something about it.

1 Now, it's true that probably 90 percent of people
2 whose BMI is over 25 are overweight, but there has to be
3 some caveat in there that there will be some people who are
4 not over -- are not obese or have too much total body fat at
5 a BMI of 25.

6 CHAIRMAN GARZA: Shirika?

7 DR. KUMANYIKA: I'm going to try to look at the
8 aging literature again and see if we know how the wording
9 should be for older people. It's not -- it's not easy to
10 figure out what wording there is. We weren't thinking about
11 changing the guidelines, but we want to have language there.

12 I think, even though I've been participating in
13 getting things to this point, I still have a feeling that
14 this guideline is missing something that consumers need, so
15 I just wanted to say that. I had actually mentioned to
16 Roland before he presented that we might be so bold as to
17 consider a separate guideline for physical activity and
18 allow this one to be more of a "how do you eat to manage
19 your weight," because as I look through what we have in the
20 old version, and probably the new one, it's just like even
21 the box that you wanted to fix about what you could eat, if
22 you look at Box 6, it really doesn't say you have to eat
23 less food except in the most subtle -- I mean, it just
24 doesn't say that. And I think that this issue of portion
25 sizes that someone mentioned, to become much more specific
26 in terms of the fact that the size of a container is not the
27 size of a portion. It would really help consumers
28 understand where all the extra food is coming from, and to
29 mention that weight has gone up in the population, and try

1 to get that prevention message a little stronger.

2 And this one, we've talked about this, it still
3 feels more clinical than it could be for the purpose of a
4 dietary guidelines, and the question is what should be
5 added. And I think, Mary, your comment about making the
6 prevention message stronger would be a place to start, and I
7 think also talking more about what to eat, how to eat as
8 opposed to only the losing weight, which is a weight control
9 guideline, but it minimizes -- it minimizes food.

10 CHAIRMAN GARZA: The only other issue that I have
11 not heard anyone raise, and probably would want to include
12 in the secretary's report, is if there is any new data on
13 the consequences of losing and gaining weight, because that
14 was one of the -- I think if I think back on the two, two
15 principal drivers that ended up with the wording that in
16 fact many have criticized as being awkward was the point
17 that Johanna raised that in fact the success rate of losing
18 weight and keeping it off looks so low that if we focused on
19 achieving a healthy weight, that we were going to be asking
20 half of the population, given the percent that's overweight,
21 to constantly be losing and gaining and losing and gaining
22 because we know that so few gain -- or rather, lose and keep
23 it off.

24 So we probably would have to update that database
25 to say has yo-yo dieting --

26 DR. WEINSIER: But this was addressed fairly
27 recently. Van, what was the date of or NIDDK report on
28 weight cycling? But it was within the last two years that
29 there's basically a data --

1 CHAIRMAN GARZA: No, I was saying included in
2 our -- I'm not saying it hasn't been reviewed.

3 VOICE: There is no other -- there is no newer
4 data out.

5 CHAIRMAN GARZA: Right, it's since '95.

6 VOICE: Right.

7 DR. WEINSIER: Yeah.

8 CHAIRMAN GARZA: What we need to do is include
9 that consensus document or the evidence that led to that.
10 That's the one piece that I saw was missing. There might be
11 others.

12 DR. LICHTENSTEIN: How does that compare,
13 losing/gaining, losing/gaining, how does that compare to
14 gaining/gaining/gaining? Is there any evidence -- no, with
15 all seriousness, is there any --

16 DR. WEINSIER: No, that's never an option. It is
17 either maintained -- that was how the issue of maintaining
18 your weight got there.

19 DR. LICHTENSTEIN: Oh, I understand that, but I
20 guess what I'm concerned about is that in taking the people
21 that gain/lose, gain/lose, what would happen if they were
22 advised not to do that?

23 CHAIRMAN GARZA: The guideline was crafted in a
24 way that would say, look, you have to focus peoples'
25 attention, focus their attention on at least maintaining
26 their weight and not gaining any more weight rather than
27 focusing their attention on losing weight -- this is a bad
28 pun because we know that's a losing proposition.

29 DR. GRUNDY: I would like to raise a point that

1 Shirika brought up and expand on it, and ask whether it's
2 possible to associate physical activity here in these
3 guidelines from body weight?

4 There are so many other advantages of physical
5 activity that go beyond body weight, and, in fact, you know,
6 you can eat in a minute what you can run off in three hours.
7 So, you know, there has -- it's not the solution to the
8 obesity problem, but it is a solution to a lot of other
9 problems that we have. I mean, I don't know whether this is
10 out of our purview or not to get into putting that in as a
11 separate thing, but to highlight it as a separate valuable
12 thing for health, you know, I think you could make a strong
13 argument for that.

14 Is that going to be something we could do?

15 CHAIRMAN GARZA: Well, we could consider it.

16 DR. GRUNDY: Yeah.

17 CHAIRMAN GARZA: I mean, the issue of whether or
18 not divorcing it from the diet fall under purview is
19 something that we need to think about carefully.

20 DR. GRUNDY: It would be divorced from the diet
21 entirely if you made it a separate item, but that it would
22 have benefit on a lot of other risk factors that relate to
23 diet beyond body weight, that's what I'm saying.

24 CHAIRMAN GARZA: I have to agree with -- I mean, I
25 think this is -- physical activity is a major issue that we
26 can't afford to overlook, and how we deal with it, either
27 with a separate guideline or strengthening the present
28 guideline, I mean, is something the group can certainly come
29 back. Either choice is available to us.

1 DR. JOHNSON: I had a discussion with Allison
2 Yates about the macro nutrients in the DRIs which I thought
3 was very interesting that may shed some light on this, and
4 she was saying that when you look at the DRI for energy, we
5 know from doubly labeled water energy expenditure data that
6 expenditures are lower than current recommendations.

7 The problem is if you get the recommendations down
8 to low, you can't easily meet micro nutrient needs, so there
9 may be sort of a paradigm shift where we need to look at
10 what do we need to get our activity levels up to in order to
11 be physically active enough that we can balance that with an
12 energy intake that we can reasonably meet our nutrient needs
13 at, so I think that just adds strength to the argument to
14 really emphasize activity.

15 CHAIRMAN GARZA: Alice and then Meir.

16 DR. LICHTENSTEIN: Just to get back to the point
17 about emphasis on maintaining, I think it should also be on
18 prevention in children; that there may be even a separate
19 section on that because that seems to be where some of the
20 problem is starting.

21 CHAIRMAN GARZA: We will give you the last word
22 and then we'll break. I'm sure the transcribers will be
23 very grateful for that.

24 DR. STAMPFER: Just to second the thoughts on the
25 physical activity. Scott and I were talking in the hall and
26 he was pointing out that caloric intake in China is
27 substantially higher than it is here even though their
28 typical BMIs are 20 or 21 due to physical activity.

29 And I think I agree with you, Scott, that it's not

1 the solution to losing weight, but I think it is either the
2 solution or it's necessary but not sufficient for
3 maintaining weight loss, and those few 10 percent that do
4 succeed several studies have shown that it's with physical
5 activity.

6 So I think it's pretty intrinsically bound in --

7 DR. GRUNDY: I'm not denigrating it's role in that
8 regard, but I think it has so many other damages beyond
9 weight control that it might deserve a separate identity.

10 DR. STAMPFER: Thereby linking it solely on weight
11 control.

12 CHAIRMAN GARZA: All right, well, then, let's try
13 to convene at 1:20. And if your sandwich has arrived, you
14 can start eating it before then. But if not, we will eat
15 after we convene.

16 (Whereupon, at 12:55 p.m., the meeting was
17 recessed, to reconvene at 1:20 p.m., this same day, Tuesday,
18 March 9, 1999.)

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A F T E R N O O N S E S S I O N

(1:35 p.m.)

1
2
3 DR. GRUNDY: Okay, what I would like to do while
4 you're eating your low fat meal is to just say a few words
5 briefly about the summary of our recommendations, and then
6 we can open this up for discussion. I would also like to
7 thank Kathryn McMurray, who helped so much and helped
8 putting this together. And after I make a few comments,
9 then Alice and Richard will also say something, and Meir
10 also wanted to comment on trans fatty acids.

11 If you look in quantitative terms, look at the
12 numbers, I don't think the recommendations that we come up
13 with are going to be a lot different than they were before,
14 but there clearly is going to be some difference in emphasis
15 if we follow the recommendations that we've outlined here,
16 and that's what I would like to go through with you briefly.

17 And if you have that before you what our summary
18 is, you can see that the priority of the recommendations are
19 saturated fat and dietary cholesterol and trans fat, and
20 then percent of energy from fat.

21 In the previous guidelines, it started out to
22 chose a diet low in fat, then saturated fat and cholesterol,
23 but we feel that if you take a look at the scientific
24 evidence, we are stronger on saturated fat than percent of
25 energy from fat or a diet low in fat, if that implies a low
26 percentage of fat.

27 So we think the guidelines should emphasize
28 saturated fat for chronic disease, coronary heart disease
29 and stroke, and this link is primarily through the effect of

1 saturated fats to raise cholesterol levels and promote
2 arteriosclerosis, and there is a broad base of evidence to
3 support that recommendation, which I think most of you are
4 familiar with. And we felt that dietary quantities of
5 saturated fat should be less than 10 percent of calories.

6 Now, dietary cholesterol intakes, currently the
7 recommendations have been on the books for a long time for
8 most groups in this country have been for dietary
9 cholesterol intakes of less than 300 milligrams per day. As
10 we read the data in this area, we think that still is a
11 reasonable recommendation.

12 We heard yesterday some evidence that dietary
13 cholesterol may have less effect than we may have thought in
14 the past, and I think it is quite clear that the effects of
15 dietary cholesterol in humans is not as great as it is in
16 animals on raising the plasma cholesterol level. But, in
17 our view, to some extent the quantitative effect depends on
18 the way you analyze the data, and which studies that you
19 take for analysis.

20 There have been a large number of studies in this
21 field that are not terribly well controlled, and then are
22 some tightly controlled metabolic studies. And if you rely
23 more on the carefully done metabolic studies, you will see a
24 somewhat greater increment in cholesterol levels in plasma
25 than what Don McNamara showed yesterday, but clearly the
26 effect is not great. But if you add it all up, say going
27 between 300 and 500 milligrams of cholesterol like it used
28 to be, in our view that would raise cholesterol levels by
29 about six to eight milligram per DL, which, I think, is not

1 trivial.

2 It also might be noted that we had a decline in
3 cholesterol levels in the population in the past 20 years or
4 so, and that has paralleled a reduction in dietary
5 cholesterol intake. In fact, the decrease in dietary
6 cholesterol may be the major factor, not the only factor,
7 but perhaps the major factor contributing to the decline in
8 cholesterol levels in the population. We can't be sure of
9 that but the data are consistent with the metabolic studies.

10 Now, we also recommended that trans fats should be
11 mentioned in the text under the saturated fats section.
12 Now, there might be one view, and maybe Meir would espouse
13 that view that trans fats should be listed as a separate
14 category separate from saturated fat. You know, that's
15 somewhat controversial and can be discussed. I think it was
16 the view of our group that the trans fat probably has about
17 the same effect as saturated fat, but the total intake is
18 quite a bit less, and therefore it might be contained under
19 the recommendation for saturated fat and sort of lumped
20 together as saturated fat and trans fat in some way which
21 we'll have to discuss. This is not to minimize the
22 importance of trans fat because it clearly raises
23 cholesterol levels in the same way as saturated fat.

24 Now, we thought that total energy intake should be
25 discussed in terms of the -- total fat intake in terms of
26 total energy intake, including carbohydrates. Now, this is,
27 in our view, would be a major shift, a paradigm shift for
28 the guidelines. But it seemed to us from reviewing the
29 literature that a case cannot be made for a very low fat

1 diet or a low fat diet being a major factor responsible for
2 obesity or for control of obesity in the population, and it
3 may be a more balanced recommendation to include those
4 together and to emphasize for the public the need to curtail
5 intake of total calories, whatever their source, both to
6 prevent the development of obesity and to reduce excess body
7 weight, and I'm sure that will be an interesting point for
8 discussion.

9 For unsaturated fatty acids, we really didn't
10 differentiate between the monounsaturated and the polyunsaturated.
11 The more recent data may give a new lease on life to polyunsaturates,
12 although probably in practical terms polyunsaturates may be
13 pretty much at the maximum intake available -- that is
14 possible now. But in any case, we put those together for
15 simplicity and called them unsaturated fatty acids, and we
16 recognize that they don't lower serum cholesterol levels and
17 they should be limited to maintain appropriate energy
18 levels. But I think at the same time they do represent an
19 important source of calories and one that should not
20 necessarily be targeted for reduction preferentially.

21 Among the omega 3 fatty acids are complicated and
22 we didn't get into those too much, and maybe it's not
23 necessary in these guidelines to emphasize those
24 particularly. Their intakes currently are low and the
25 appropriate intake is not clear.

26 We also thought it was important to emphasize
27 foods rather than fat per se as sources of particular fatty
28 acids. The discussion of nuts this morning was a good
29 example of that. These are rich in unsaturated fatty acids,

1 and it was pointed out how they may be a useful component of
2 the diet, and that's one way that we might emphasize foods
3 rather than particular fatty acids.

4 I think those -- well, perhaps one other
5 recommendation. We thought they should focus on individuals
6 and not populations, and that's the aim of the guidelines is
7 for individuals, and that's been a subject of some confusion
8 in the past.

9 There also needs to be some consideration for
10 children. There was emphasis given in the previous
11 guideline, I believe, for children under age two with regard
12 to dietary fat, but I believe Richard wants to make a case
13 for perhaps cutting that out of this particular guidelines
14 and making a more general statement for children.

15 Now, perhaps Alice might want to say something and
16 then Richard, and then we could open, and Meir, and then
17 have a discussion.

18 DR. LICHTENSTEIN: My comments actually are going
19 to be limited to omega 3 fatty acids and fish that I think
20 there may be some reason to include a recommendation that
21 fish be consumed, even on a weekly basis, something like
22 that, because I think there is epidemiological evidence to
23 support a relationship between decreased incidence of
24 cardiovascular disease and fish consumption.

25 I think there is some clinical data to support it,
26 that levels of omega 3 fatty acids in red blood cells
27 correlate inversely with risk of sudden death after a heart
28 attack, and that there is some work on a more basic level
29 suggesting there omega 3 fatty acids may be involved with

1 arrhythmias, heart arrhythmias.

2 I also think a recommendation like that is not
3 particularly radical and also would be consistent with some
4 of the other recommendations because recommending something
5 like fish would most likely displace other types of foods
6 that will be high in saturated fats. So do something for
7 consideration. I think that the amount of evidence has
8 increased between 1995 and now with regard to that point.

9 Otherwise, I totally concur.

10 DR. GRUNDY: Richard.

11 DR. DECKELBAUM: Just a short comment on children.
12 I think in the previous guidelines it was written that
13 children between the ages of two and five should gradually
14 adopt a diet to what would meet 30 percent whole fat and 10
15 percent saturated fat.

16 I think that, you know, in terms of
17 implementation, we're spending a lot of time on
18 implementation, it's hard to give guidelines for gradual
19 implementation, and I think the good news is that there is
20 certainly new data since the previous guidelines have been
21 published showing the safety of diets that are moderate in
22 fat, 30 percent or even lower, and there is the DISK
23 studies, a number of papers which are in older children, but
24 something that's ongoing right now is the -- which is
25 outside of the United States, but it's quite an exciting
26 study. It's the STRIP study, and I can't remember exactly
27 what it stands for. Meir, do you want to -- but in this
28 study, this is children in Finland, a relatively large
29 cohort. I think about 2,000 who were randomized after

1 leaning to a low fat diet, which is about 28 - 29 percent,
2 about 28 percent total fat, and low in saturated fat as well
3 by nutritional guidance compared to the normal control, some
4 other kind of guidance.

5 And now the initial STRIP cohorts are about five
6 and six years old, and they've done a lot of work on not
7 only lipid levels with the experimental group with the low
8 fat group, but quite important data has been accumulated to
9 show that there is absolutely no adverse effects on
10 cognitive or other development signs, so that these are kids
11 who were getting 27 to 29 percent fat beginning at the age
12 of, depending on when weaning is, four to seven months, and
13 there is no adverse effects and outcome.

14 And I know that they have some unpublished data
15 which is actually quite exciting with regards to how the
16 prevalence of obesity in five - six year olds, depending on
17 what group they were in. I don't have the data, but you can
18 guess.

19 So I would just encourage that you pretty much go
20 with the concept that children over the age of two can
21 pretty much follow the rest of the guideline for fat, and I
22 don't think there needs to be any qualifiers for children
23 over the age of two.

24 Under the age of two, I'm not sure that's within
25 the charge of the USDA, so that I think we would just stay
26 away from it and not include it as part of our mission.

27 DR. GRUNDY: Okay, Meir.

28 DR. STAMPFER: It's part of the USDA but it's not
29 part of this committee.

1 CHAIRMAN GARZA: While Meir is going to get up to
2 the table, I can brief you on at least the way that language
3 that Richard was referring to came into the guideline was
4 the sense that it was not safe, and there were some of us on
5 the committee that argued that, in fact, couldn't understand
6 the lack of safety given the normal weaning pattern. So the
7 data are not very surprising, but it was in the end a
8 compromise among those on the committee who felt that
9 extending those fat guidelines to the age of two was not a
10 problem, and those who felt very strongly than, in fact,
11 that might have been -- I think they were concerned we would
12 be stumping children and also causing cognitive problems.

13 DR. DECKELBAUM: I just want to add one thing and
14 this comes up repeatedly in terms of kids and children over
15 the age of two. I don't think there is anyone who promotes
16 these moderate fat intakes who says to achieve this that
17 dairy products should be excluded, so that these guidelines
18 do not mean decrease in the intake of dairy products in
19 children. What they do mean though is encouragement of low
20 fat dairy products.

21 DR. LICHTENSTEIN: Can I talk about children just
22 --

23 CHAIRMAN GARZA: Well, go ahead and then we will
24 go to Meir.

25 DR. LICHTENSTEIN: Okay. That I think also that
26 Richard's proposal is very reasonable because now that the
27 school lunch program, so that when the kids hit school at
28 age five, they're adhering to the recommendations of less
29 than 30 percent, less than 10 percent saturated fats. So

1 there is no phase-in at that point.

2 DR. STAMPFER: Yes, I just wanted to say a couple
3 of words about trans. Trans is unique because not only does
4 it raise LDL but it lowers HDL, and we haven't talked much
5 about HDL, and I guess there is some degree of controversy
6 over how much causality to attribute to HDL, but it's
7 certainly a very strong predictor. The higher HDL is
8 associated with lower risk of heart disease. So something
9 in the diet that does both bad things, raising LDL and
10 lowering HDL, we have to be very cautious.

11 This is a summary by Alberto Escharia, and it's a
12 figure from a paper that's under review that I will
13 distribute to the people around the table and ask that you
14 don't pass it around because it's not published. But this
15 summarizes all the studies that have compared saturated fat
16 with trans on the impact on the LDL to HDL ratio, and what
17 one finds is that trans is about twice as bad as saturated
18 fat is in terms of this ratio. Both saturated and trans
19 raise LDL, but because trans also lowers HDL, it has a much
20 worse impact.

21 Now, the second point is that the increase in risk
22 associated with trans is actually higher than what you would
23 predict just based on the lipid changes in the epidemiologic
24 studies. For example, this is from the nurses, and
25 admittedly this is just one study, but the bottom bar here
26 projects what the change in risk would be if the two percent
27 of energy from trans were replaced with two percent of
28 energy from unsaturated fat, and it basically cuts the risk
29 approximately in half, which is far more than you would

1 predict based on the lipid levels. And this makes sense
2 biologically because trans can interfere with the metabolism
3 of essential fatty acids.

4 Two final points: One is that the natural
5 replacement of trans is polyunsaturated fats, which are
6 beneficial, and the clinical trials that have shown a
7 benefit of lowering saturated fat by replacing with polies
8 have shown benefit in contrast to the clinical trials that
9 just reduce fat.

10 And the final point is that this easy to do
11 because a lot of the trans that we get comes from
12 manufactured product, baked goods, and fast food, fried
13 food, and there are replacements that are available at
14 higher cost but not astronomically higher cost. Trans is
15 basically pretty much being phased out in Europe. We can do
16 it here in the U.S., so that consumers can reap a dietary
17 benefit with very little effort on their part.

18 And also just to make a second to what Alice was
19 saying about the omega 3's. I think that's another point
20 that we should consider, whether the data is strong enough
21 to emphasize. I think that they are strong enough to
22 mention.

23 DR. GRUNDY: What kind of a recommendation would
24 you make for omega 3, that they are strong enough? I didn't
25 quite understand what you were saying there.

26 DR. STAMPFER: Whether we should distinguish that
27 from just unsaturated fats is all.

28 DR. GRUNDY: You think we should?

29 DR. STAMPFER: I think we should talk about it.

1 DR. GRUNDY: Okay. Are you going to lead the --
2 yes.

3 DR. LICHTENSTEIN: I would like to say something
4 about the trans fatty acid issue.

5 I don't agree that we should put a lot of emphasis
6 on trans fatty acids for a couple of reasons. Although the
7 epidemiological data does so, an association with a greater
8 risk with trans fatty acids as opposed to saturated fatty
9 acids, the clinical data doesn't necessarily support that
10 because there have been two very extensive studies on trans
11 fatty acids and blood clotting, and they both turned out to
12 be negative. There have been two studies published on trans
13 fatty acids and acceptability of LDL toxidation. However,
14 we feel that should be taken into consideration and those
15 are negative.

16 So although there is some -- or no effect. And
17 although there is some basic work suggesting an effect of
18 trans fatty acids on essential fatty acid metabolism, there
19 has been no biochemical abnormality or change to date that's
20 actually been associated with it, but I guess my concern is
21 that right now it's been estimated that the trans fatty acid
22 intake in the U.S. is about 2.2 percent energy intake
23 whereas the saturates is about 12 to 14, but we're not near
24 where the goal of 10 percent was set years ago.

25 And I think that there is not too much room for
26 play as far as discretion goes in trans fatty acid intake of
27 individuals. I think the food supply is going more towards
28 lower in trans fatty acids, and I certainly agree that the
29 impact of trans fatty acids on LDL cholesterol is similar to

1 saturated fatty acids, whereas saturated fatty acids do
2 raise HDL. I think trans may not quite lower them but have
3 no effect so that the ratio is a little bit worse, but I
4 think when you look at the relative portion of intake in the
5 U.S. diet and what the impact of the message has, I would
6 hate to see a lot of focus on trans and lose the focus on
7 sats. whereas I think we can make the most or the biggest
8 impact if we could get saturated fat intake lowered, the
9 biggest impact on risk for cardiovascular disease.

10 CHAIRMAN GARZA: Any other questions or comments
11 from the group?

12 Go ahead.

13 DR. DWYER: I thank Scott for his presentation. I
14 share, with less knowledge from direct experimental work,
15 Alice's concerns about trans, over-emphasizing it and not
16 just sort of lumping it under saturated.

17 What I wasn't clear about, however, Scott, and I
18 wanted a little more from the committee is it seems awfully
19 heavily weighted to cardiovascular coronary heart disease
20 end points. And on the total issue, it would seem to me
21 there might be reason to focus on other issues as well as
22 those.

23 So I find the second page of the possible way to
24 go a little coy. If there is a way to -- one of the biggest
25 randomized trials in the world is now being carried out with
26 American postmenopausal women on dietary fat, total fat. It
27 might be worth us at least reviewing the reasons for putting
28 that particular intervention into the -- into the study, the
29 Women's Health Initiative. I realize that we heard one

1 presentation this morning that suggested, well, maybe 10
2 years later they wouldn't have done it or he wouldn't have
3 done it. But these recommendations are extant both from the
4 National Academy of Sciences for low fat and also they are
5 now in play in a very large clinical trial, and wondered if
6 perhaps those concerns might be better reflected by a little
7 broader approach.

8 DR. GRUNDY: I think we're certainly aware of the
9 issue, and, you know, maybe we didn't highlight it
10 adequately for the whole, total fat discussion. I think
11 that the evidence that was presented today was pretty much
12 the way our committee read the evidence; that certainly when
13 we're talking about 30 percent fat in the diet, that is a
14 moderate to relatively low fat intake, and in a way it was
15 where we put the emphasis.

16 One of our concerns, I guess, had to do more with
17 the obesity issue than it did with the cancer issue, which,
18 you know, I think is a little problematic. But the obesity
19 area, it seems like that maybe our emphasis on low
20 percentage of at may have backfired on us, and that perhaps
21 we would be wiser to go forth with a message that was across
22 the board calories rather than just emphasizing fat. if we
23 stick with the 30 percent fat recommendation, that ought to
24 be adequate to lead to a good body weight if people would
25 pay attention to their diet and control all the components
26 in the diet. So I guess that we thought the obesity thing
27 was perhaps a little more in the fore in this regard than
28 the cancer issue.

29 Now, also, from what we have heard and seen from

1 the literature about the cancer issue is that saturate fat
2 seemed to rise to the top among the different components of
3 the fat story that led to the cancer. So there again by
4 reducing saturated fat the evidence seemed to point more
5 towards saturated fat than it did total fat, so that was
6 another reason, I guess, but maybe we didn't articulate that
7 adequately. Maybe you don't even disagree with that
8 conclusion either but --

9 DR. DWYER: No, I don't disagree with putting
10 saturated first. I think that's good. What I do disagree
11 with a lot is mentioning trans fat five times and mentioning
12 total fat once.

13 DR. GRUNDY: Uh-huh.

14 DR. DWYER: And only in the context of coronary
15 heart disease.

16 DR. GRUNDY: Okay. Well, I think that's a valid
17 point, that we have to make an appropriate argument for our
18 position considering all the different factors.

19 Now, you still may not agree with the conclusion
20 even if we -- even if we did it in a logical fashion like
21 you outline. I mean, I guess -- I'm sure that's going to be
22 a point of discussion, and I expect that, and I think we
23 should discuss that.

24 Go ahead, yeah.

25 DR. STAMPFER: Maybe Johanna -- maybe you could
26 elaborate a little. What adverse health effect is reliably
27 attributed to higher total fat that's not specifically
28 related to animal fat or saturated fat?

29 What adverse outcome is there for say more mono or

1 poly where there is credible data?

2 DR. DWYER: Well, I guess what I'd suggest is that
3 I'm concerned a little about your -- and perhaps you have
4 data that you could show us that would alleviate those
5 concerns.

6 DR. STAMPFER: You mean --

7 DR. DWYER: Yes. I'm not sure it's a subtle
8 issue. You know, I think that it's a legitimate debate
9 about whether it's fine to go to 35 or 40. You know, we're
10 really right back where we were in 1971 and '2, when they
11 were formulating the Mr. Fipp study when the decision was
12 made to go with a higher fat level instead of going down.

13 DR. GRUNDY: Well, I don't think we're going to 35
14 or 40. I mean, I think we have to --

15 DR. DWYER: Well, you give one mention of 30.

16 DR. GRUNDY: Yeah, I think we have to make that
17 clear that 30 is -- I mean, we're not changing -- that's why
18 I said at the very beginning -- we're not changing the
19 numbers; we're just changing the emphasis more to -- and
20 hope by changing the emphasis as the message gets out there,
21 that the final result will be a more balanced result.

22 CHAIRMAN GARZA: Alice, did you want to say
23 something?

24 DR. LICHTENSTEIN: No, I was going to --

25 CHAIRMAN GARZA: Rachel.

26 DR. JOHNSON: There is some data in children,
27 referring to what Johanna said, in a paper by Manose that's
28 in Pediatrics that analyzed USDA's survey data when the
29 children reached a high fat level, 35 - 40 percent, it does

1 affect adequacy. Only in the context of the U.S. diet when
2 you see somebody with 35 - 40 percent diet, it's not from
3 olive oil generally in the U.S. diet. So I think in the
4 context of as the guidelines fit into our culture there
5 could be adequacy problems at fat intake levels, getting
6 upwards of 35 and 40.

7 CHAIRMAN GARZA: Meir.

8 DR. STAMPFER: One other quick question. There
9 seems to be a contradiction in the text where for
10 unsaturated fat you say "must be limited only to maintain
11 appropriate levels of total energy intake."

12 How do you reconcile that with the -- do you
13 intend to maintain a 30 percent from calories limit for fat,
14 and if so, one of those two statements don't go together?

15 DR. GRUNDY: Okay. You know, I guess the answer
16 to that -- I think that's a good question, and if I
17 understand it correctly, let me try to answer it.

18 If you reduce saturated fat, that carries with it
19 a reduction of certain categories of fat, and that also
20 reduces some unsaturated fat that's carried along with that.
21 So that opens the door for a lot of replacement with
22 vegetable oils. So probably you're not going to -- if you
23 get the saturated fat down to the level we want, you're not
24 going to get too much above 30 percent unless, you know, you
25 turn into, you know, somebody from Crete or some place like
26 that. But most of the time the Mediterranean area where
27 that kind of diet is followed, I think it's around 30 to 35
28 percent; isn't that correct?

29 CHAIRMAN GARZA: What about the arguments that are

1 made by some of the individuals in the cancer field, for
2 example, in the American Cancer Institute report that
3 suggested, in fact, 30 percent is too high; that we ought to
4 be aiming at lower fat levels if we really are going to deal
5 with some of the cancer nutrient links? Do you feel that
6 date is just not sufficient to make public policy, that it's
7 wrong? Because that's the only other issue that I can
8 imagine we will be faced with is, well, why are we
9 maintaining 30. I mean, has the database not changed since
10 1995?

11 DR. GRUNDY: Okay, we had a good review of that
12 today, I thought, and, you know, personally I thought that
13 was an adequate review of the diet/cancer fat/cancer link.
14 I agree, as I tried to question the speaker, that, you know,
15 what is -- what are the data that supports these earlier
16 claims and earlier positions, and he wasn't able to
17 articulate those very well, and I think we're still waiting
18 to hear those put forward in a clear-cut manner. I think
19 they basically related to cross-cultural studies in certain
20 populations that have very low fat intake there is a
21 relatively low cancer incidence. But, you know, there are
22 some animal studies that support that too, but I think like
23 you say, we may not be on firm enough ground there to make
24 public health recommendations.

25 Maybe after the women's health trial is over, if
26 it turns out we're soundingly positive, you know, we might
27 have to reconsider that issue.

28 CHAIRMAN GARZA: There was one trial at NCI that
29 was attempting to use low fat on the polyp prevential trial.

1 That's been completed, but I don't know if that's been
2 published.

3 DR. DWYER: It will be published by June, won't
4 it?

5 CHAIRMAN GARZA: Yes. Perhaps we could have them
6 come then at our next meeting if it hasn't been published
7 and review that data. That's the only trial that I'm aware
8 of that was aiming at possibly lower levels of fat.

9 Either anyone on the committee or in the audience
10 who have any other trials that have been completed in the
11 last five years?

12 DR. DWYER: There are two that are in progress.
13 One is the Women's Health Initiative.

14 CHAIRMAN GARZA: Yes, but that's not going to be
15 finished by --

16 DR. DWYER: No, it won't be finished, and there
17 won't be interim results on it.

18 I think Ross Preniss has written a paper that
19 outlines the rationale for that. I know certainly members
20 of the committee disagree with what he said, but basically
21 it's written there.

22 The other trial that's a secondary prevention is
23 that WINN's -- what is it called -- WINN's? That's a cancer
24 adjuvant therapy trial in women who have breast --
25 postmenopausal women who've had breast cancer, so it's a
26 little different than primary prevention.

27 CHAIRMAN GARZA: Yes. This one was also a polyps
28 a secondary prevention of polyps.

29 DR. GRUNDY: I want to make one other comment

1 about that, is I think we have to be careful not to let
2 disease-specific, diseases that are relatively rare in terms
3 of the total population drive a total dietary guideline. I
4 mean, even if a lower fat reduced colon polyps, that
5 wouldn't necessarily mean that we would change our
6 guideline, but it could be noted. I mean, every little
7 possible health problem, even though it's important to the
8 person that got it, might not be enough to justify driving
9 the whole guideline.

10 CHAIRMAN GARZA: Rachel.

11 DR. JOHNSON: I just would like you to address, it
12 can be briefly, but the work, you know, by Dean Ornish that
13 says that seven to 10 percent fat diet actually leads to
14 regression of atherosclerosis and I think he had some more
15 end plinths in terms of MIs and just address that.
16 Certainly that's a very, very low fat diet.

17 DR. GRUNDY: That's a very low saturated fat diet.
18 If you just -- you know, we used to give people in metabolic
19 studies diets that were very high, 40 percent corn oil
20 diets, and their cholesterols fell just as much as what he
21 would obtain from those very low. It's the saturated fat is
22 what would raise the cholesterol unless you believe that
23 there is something magic about just low fat in terms of
24 etherial genesis, which some people have claimed, you know,
25 that's certainly something that's never been proven. So I
26 think it's that they lower cholesterol levels quite
27 effectively.

28 They can get -- they also in those metabolic
29 settings they have patients lose weight in addition to that,

1 and they get levels of LDL deduction similar to what's
2 obtained in clinical trials with drugs. And when you get
3 kind of reductions and you can induce some regression of
4 lesions, there is no doubt about that.

5 But, you know, whether those kind of diets justify
6 making a recommendation that we all eat five percent fat,
7 you know, that's a totally different issue, I think.

8 CHAIRMAN GARZA: Shirika?

9 DR. KUMANYIKA: I wanted to comment on the fish
10 issue just so it doesn't get lost.

11 For say low income populations, urban areas,
12 that's going to mean a fish sandwich, a fried fish sandwich
13 at a -- well, when we make the recommendation, we have to
14 make clear what kind of fish we're talking about because for
15 a lot of people, that's menu choice that would -- they would
16 interpret as something to increase, and so it's not quite a
17 simple as eat more fish. It will be, you know, eat certain
18 types of fish or whatever we would want to say.

19 But I know in scoring diets in studies with
20 African-Americans we have to change the way we score the
21 fats instrument because if people increase their fish
22 consumption, they're supposed to get a better score. But
23 it's always fried.

24 CHAIRMAN GARZA: That's a good point.

25 DR. LICHTENSTEIN: I'd like to say something about
26 the Owen studies, or study with about 35 people in it and no
27 control group, adequately matched control group; that there
28 were really three components to that. One was an extremely
29 low fat diet, and as Scott pointed out, drastically low in

1 saturated fat. There is also an exercise component that was
2 supervised, and there was a stress reduction component of it
3 where they met with a psychologist multiple times per week.

4 What's also interesting with that is they lost
5 about 22 pounds during the first year, which also helped
6 with the dramatic reduction in plasma lipid levels which I'm
7 sure accounted for a lot of the regression in addition to
8 the decrease in saturated fat and cholesterol intake.

9 But interestingly, if you look at the follow-up
10 data over the next five years, they claim good adherence to
11 the diet and weight was flat because I know issues have come
12 up with body weight and total fat intake.

13 So that's the data. It's one single study and a
14 small group of individuals.

15 CHAIRMAN GARZA: I would ask Suzanne for the last
16 comments so we can move on to the alcohol guideline. Then
17 I'm going to be leaving at 2:30 on the Dietary Guideline
18 Advisory Meeting and Suzanne will chair, and we'll work --
19 we'll be in working group sessions after the food safety.

20 so suzanne.

21 DR. MURPHY: Just a quick question that we
22 probably don't want to take a lot of time to discuss right
23 now, but am I understanding that your group is proposing
24 taking total fat out of the guideline wording?

25 DR. GRUNDY: There is two ways to look at fat.
26 One is percentage of fat and one is absolute amount. And we
27 wanted to change the emphasis of total fat to the absolute
28 amount and combine that with an emphasis on the absolute
29 amount of carbohydrate, and put that as the emphasis for

1 people to pay attention to rather than trying to figure out
2 what percentage of fat is in the diet, because if you're
3 eating a low percentage, you're eating a high carbohydrate
4 diet. So if you eat a low fat diet the way it's written
5 now, you'd have to say "eat a low fat, high carbohydrate
6 diet," and that's what we don't want.

7 DR. MURPHY: But the wording in our notebook
8 does -- all the options say nothing about total fat in the
9 guideline itself; is that correct? Alice is nodding.

10 DR. GRUNDY: Well, not --

11 DR. LICHTENSTEIN: According to the way it's
12 written.

13 DR. GRUNDY: It says total, "total fat intake
14 should be discussed in terms of total energy intake,
15 including carbohydrate."

16 DR. MURPHY: But not in the wording of the
17 guideline at all. I think that's something we need to --

18 DR. GRUNDY: Well, let's see here.

19 DR. MURPHY: -- revisit.

20 DR. GRUNDY: What page?

21 DR. MURPHY: It's under "Detailed Outline," the
22 page that has -- and you have A through F?

23 DR. GRUNDY: Um-hmm.

24 DR. MURPHY: And one of the options, it says
25 "Total fat."

26 DR. GRUNDY: Okay, I'm looking here.

27 No, jump over there to the -- on the next page it
28 says, "Choose a diet low enough in fat and carbohydrate
29 calories to achieve appropriate body weight."

1 DR. MURPHY: So that's a new guideline? I just
2 don't -- I'm confused about what's in the guideline and
3 what's in the text.

4 DR. GRUNDY: What's in our -- what we're proposing
5 to be in the new guideline?

6 DR. LICHTENSTEIN: Yeah, the new guideline; not in
7 the guideline, but the absolute guideline itself.

8 DR. STAMPFER: The picky statement that we are
9 going to be making.

10 DR. GRUNDY: Well, I think it would be "Choose a
11 diet low enough in fat..." Isn't that what we want to
12 propose?

13 DR. MURPHY: Well, the proposals right now are
14 "Choose a diet low in saturated fat and carbohydrate.
15 Choose a diet low in saturated fat, or choose a diet low in
16 saturated fat, trans and cholesterol."

17 DR. GRUNDY: No, that's just the option for the
18 saturated fat and cholesterol component of that. That's not
19 for the whole -- that's not for the total thing. That's
20 just for that component.

21 VOICE: Well, what do you propose it to cover?

22 DR. GRUNDY: Well, I don't know. I didn't know we
23 were supposed to propose a cover.

24 DR. MURPHY: No, I'm not implying --

25 DR. GRUNDY: No.

26 DR. MURPHY: I thought you had proposed --

27 DR. GRUNDY: No, we have not.

28 DR. MURPHY: -- it in these A through F.

29 DR. GRUNDY: No, that's not the total. Yeah, I

1 think we're just trying to give some options there for the
2 saturated fat and cholesterol component --

3 CHAIRMAN GARZA: For one of the headings.

4 DR. GRUNDY: Yeah, that component.

5 DR. MURPHY: All right.

6 CHAIRMAN GARZA: Thank you very much --

7 DR. GRUNDY: Sure.

8 CHAIRMAN GARZA: -- Scott, and to the other
9 members of the group. That was very helpful.

10 All right, we'll move on then to the alcohol
11 guideline, Dr. Stampfer.

12 DR. STAMPFER: I would like to start with just a
13 little bit of data, commenting on some of the issues raised
14 by Dr. Gordis and then I'll talk about some of the issues
15 that -- I'm going to use this in a minute.

16 But just, first of all, to comment on a couple of
17 his points. These are data from the Health Professional
18 Follow-up Study.

19 DR. MURPHY: Meir, I can't hear you. I don't know
20 if you're mike is not on or if there is too much
21 distraction.

22 DR. STAMPFER: It's on, isn't it?

23 DR. MURPHY: Thank you.

24 DR. STAMPFER: I'm showing these data to stress
25 two points. First, to underscore that this idea of the sick
26 quitter as an explanation for the lower risk of coronary
27 disease can be pretty much dismissed because you can see
28 those two lines. One is the total cohort, one is with men
29 who have no preexisting important health conditions and they

1 are super-imposable.

2 And the second point I want to raise with this
3 slide is to show the magnitude of the reduction, and it's in
4 the neighborhood of 35 to 40 percent for coronary heart
5 disease, so this does fit in quite nicely with the data that
6 we were presented yesterday regarding the 22 percent
7 reduction in total mortality. It does hang together.

8 His second point was that, well, maybe wine
9 drinkers eat tofu and have a healthy lifestyle and that
10 could be the explanation. This slide shows the
11 characteristics of people in this study, the men, and this
12 is true in women too according to their average alcohol
13 intake, and you can take a look that, for example, if you
14 look across the BMI column, they are all -- mean BMIs are
15 all around 25. They're not especially lean, the drinkers.
16 if you look at smoking, it's well know that people who drink
17 more tend to smoke more. So it's not the case that moderate
18 alcohol consumption is a marker for a healthy lifestyle in
19 this and many, many data sets.

20 In fact, when you adjust for the other risk
21 factors, the protection gets even stronger. And actually,
22 in the -- now, for total mortality, these are data from the
23 Nurses' Study. You can see that CHD mortality is reduced by
24 40 percent among the most moderate drinkers. Total
25 mortality is reduced by about 30 or so percent.

26 But if you -- if you look at women who don't have
27 coronary risk factors, and this is actually a minority of
28 women because risk factors are so prevalent, you don't see
29 much in the way of a reduction in total mortality. So this

1 really supports the biology that it's due -- that the
2 benefit, what there is of it, is due to reduction in
3 coronary disease. And then at the higher levels of
4 drinking, you see an increase in risk as you've explained.

5 DR. DWYER: Meir, is that absolutely alcohol?

6 DR. STAMPFER: This is grams per day of alcohol,
7 right.

8 DR. DWYER: Absolute?

9 DR. STAMPFER: Right.

10 DR. DWYER: So it's one to four grams of
11 absolutely alcohol?

12 DR. STAMPFER: right, so that's about -- so the
13 first category would be up to about half a drink per day.
14 The next category is about a drink per day or a little bit
15 less. Right, this is alcohol, not averaged over the
16 different sources of beverage.

17 Now, this is -- here is another -- this is data
18 from a beer drinking population. Again, you see all cause
19 mortality or CHD mortality. You see that U-shaped curve.
20 it's not just the tofu eating, wine drinkers. This is from
21 Germany. And, again, you see with high levels of
22 consumption, much higher than what we are -- what we have in
23 our guidelines as a limit, there is an increase, but you see
24 a very substantial reduction compared to the non-drinkers
25 either for total or for CHD, either CHD incidence or all-
26 caused mortality. It's big, it's a big effect.

27 This is incidence rate. The vertical access is
28 incidence either of all-caused mortality or CHD incidence,
29 and comparing no alcohol intake to different, different

1 amounts of alcohol. That's from Germany.

2 And this is just to emphasize -- these are the --
3 this is an old slide, but this is the only limited
4 prospective studies of alcohol and coronary disease, 34, and
5 there are actually probably another six or seven since then,
6 and almost all show this, this is remarkably consistent, and
7 with huge body of data. This is looking at SACCO that was
8 mentioned earlier, the moderate alcohol and ischemic stroke,
9 you see the same kind of J-shaped curve with the lowest
10 levels, the lowest incidence at moderate levels of
11 consumption. Then it actually exceeds the level of never
12 drinkers.

13 But pay attention to the axes too. It's a big
14 reduction in the odds ratios going down to say a 40 - 45
15 percent reduction in risk. These are not small.

16 This is the AC study, an earlier version of the
17 one presented earlier. I'm going to skip through this in
18 the interest of time. There are too many slides here.

19 This is one of many, many experiments showing the
20 effect of alcohol on HDL, percent increase. You can see
21 big, big league increases in HDL. These are not subtle, and
22 HDL, this is one of many, many studies. This is from
23 Framingham looking at the relation between HDL and
24 cardiovascular risk compared to average risk. You can see
25 changes in HDL or differences in the level of HDL are
26 associated with very marked differences in risk. So it's
27 clearly a very important marker. Whether it's causal is
28 controversial. I believe that it is causal.

29 And in terms of mechanism, about 60 percent of the

1 apparent reduction can be explained through changes in HDL
2 and Apo-A1. Insulin sensitive is reduced and hemostasis. I
3 think that's -- oh, just the last slide to again get back to
4 this healthy wine drinker concept. Basically all the
5 studies that looked at moderate consumption, whatever the
6 beverage of moderation is, that's the one that is associated
7 with the most reduction in risk. In fact, in the Health
8 Professional Follow-Up Study, spirits was the best
9 predictor, even better than wine.

10 And this slide just depicts studies that have
11 looked at beer, wine and liquor. it's the number of studies
12 that show this reduction in risk, and basically the studies
13 that looked at different beverages simultaneously pretty
14 much find that alcohol per se is the one that's associated
15 with lower risk.

16 Okay, now, let me turn to the issues that have
17 been raised that we can talk about, and I'll try to be brief
18 on this.

19 So these are some of the issues that have been
20 raised in terms of how we might modify the guidelines if we
21 want to modify them. Perhaps -- and I'll go through each of
22 these, to say a couple of words about each of those topics.

23 The first adverse effect, "Should more stress be
24 placed," and these are all phrased in the form of questions,
25 not recommendations. "Should more stress be placed on the
26 adverse effects of excess intake?" This is something we
27 should consider.

28 And one point that was raised a couple of times is
29 this sentence, which is now the third sentence of the

1 current guidelines, "Alcoholic beverages have been used to
2 enhance enjoyment of meals by many societies throughout
3 human history. Should this be altered?"

4 I think some people have said, well, you can say
5 the same about sugar. You can say the same about salt, and
6 so this should be taken out. The reason that this was put
7 in, I went back to look at the guidelines' report, the
8 reason that this was in originally was to emphasize alcohol
9 as a food in these dietary guidelines rather than as a drug,
10 and I think that was the rationale for it, and we could talk
11 about whether we think that's a reasonable rationale or not.
12 It's certainly not factually incorrect. Nobody could argue
13 that this is false. But whether this has a place in the
14 guidelines, we could discuss.

15 The second point, pregnancy, "Should it be more
16 broadly targeted to women who may become pregnant rather
17 than just pregnant? Should we tighten the language?"

18 Right now it says "Fetal alcohol syndrome has
19 attributed to heavy drinking. Should we consider tightening
20 that to perhaps saying `causes.'"

21 Here is another statement that the question had
22 been raised whether this should remain in the guidelines.
23 "Lack of conclusive evidence that an occasional drink is
24 harmful to the fetus." Dr. Gordis pointed out that
25 accumulating evidence suggests that perhaps a lower and
26 lower threshold is -- would be in order so that perhaps this
27 ignorance is no longer present.

28 Another point that several have raised,
29 individuals, using medications. Right now we say, the

1 guidelines state that if you're using medications, you
2 shouldn't drink. Most older people are using medications of
3 some sort, and is it appropriate that we just on a blanket
4 basis exclude them from any alcohol? I think that's an
5 overstatement and we should consider how to amend that
6 because that's clearly not scientifically defensible.

7 Several people have raised the issue of age
8 targeting for this guideline, and the question is, "Should
9 we emphasize the risk of abuse and lack of benefit for young
10 people?" There is now really good data, I think, showing
11 that the earlier people start drinking regularly, the
12 greater their risk of alcohol abuse later in life. And
13 since the benefit appears to be pretty much for
14 cardiovascular disease, it's not a -- it's a situation where
15 the young really don't benefit and only have a potential for
16 harm in terms of their health risk.

17 Breast cancer, this has been raised a few times.
18 I was actually surprised that Dr. Gordis characterized this
19 as a non-event. I think Tim thought this was a good basis
20 for providing specific guidance to the population, and I
21 think that's something we should talk about.

22 On the benefit side for cardiovascular disease,
23 several people have commented on the phrase "in some
24 individuals." The current guidelines say that "Moderate
25 alcohol consumption is suggested to reduce risk of coronary
26 disease in some individuals," and whether we should define
27 this a little more explicitly because it's kind of left
28 hanging. Again, should we specify that the older
29 individuals more likely to benefit from reduction of CHD,

1 and we could talk about the specifics of the wording.

2 Finally, there are other health benefits of
3 moderate alcohol consumption that we haven't really talked
4 about. Some studies find decreased osteoporosis. This is
5 not completely consistent data. For non-insulin dependent
6 diabetes, actually this is quite a consistent finding,
7 although there are not that many studies, but the ones that
8 have looked do find reduction in non-insulin dependent
9 diabetes, and in short term studies find a better -- better
10 insulin sensitivity with moderate alcohol consumption, and
11 there is a reduction in gallstones with moderate drinking.
12 So I guess the question is should any of those be mentioned
13 or not.

14 Definition of "moderation," some people have
15 claimed that this is hard to understand and should we alter
16 this somehow and I guess we could think about what sorts of
17 recommendations we might make with that.

18 And should we change the summary guideline
19 statement? Actually, there hasn't been much, either from
20 the alcohol -- I wouldn't want to characterize anyone as an
21 alcohol advocate, but for the people who favor the benefits
22 of moderate consumption or the people who worry a lot about
23 the adverse effects, there hasn't -- doesn't seem to be a
24 strong sentiment for changing the actual slogan of the
25 guideline, and we could talk about it, but my sense would be
26 to leave well enough alone.

27 These are some issues that Dr. Sutter raised. How
28 can we estimate the effects of alcohol on motor function?
29 In particular, will moderate alcohol consumption promote the

1 possibility for old people to fall and break their hips and
2 that sort of thing?

3 Actually, I don't know of any data supporting an
4 association of moderate alcohol consumption with fractures
5 in the elderly.

6 Are there effects of alcohol related to the
7 duration of the dietary patterns that include alcohol? And
8 this question also raises the question that had come up
9 earlier about how long do you need to be drinking for a
10 benefit to accrue, and are there any specific potential for
11 abuse among individuals who initiate consumption after the
12 age of 40? And I think it's a very good question, and I
13 don't think there is any data to answer that in any
14 particular group of individuals.

15 So that concludes what I thought seem to be some
16 of the issues that had been raised, but I'm sure there are
17 other. We can talk about it for awhile.

18 DR. MURPHY: Thank you. Questions?

19 Dr. Weinsier.

20 DR. WEINSIER: If I don't drink, but I want to
21 reduce my risk of having a myocardial infarction and death
22 from cardiovascular disease with moderate, reasonable
23 intake, how much time would it take me to recognize that
24 benefit?

25 DR. STAMPFER: It's unknown. There does seem to
26 be a short-term benefit, and several studies have looked at
27 this and found that the alcohol that you did or didn't drink
28 last night may be related to your risk of coronary disease
29 today based on platlett function and thrombolytic function.

1 And epidemiologic studies support that an acute -- an acute
2 beneficial effect, so part of the effect would kick in the
3 next day.

4 But in terms of the HDL, if that's truly causal,
5 then my guess is that it would take a couple of years to
6 kick in.

7 I think -- pardon? Oh, you'd see the HDL rise in
8 a few weeks, but the benefit of an elevated HDL on clinical
9 outcomes probably would take, I'm guessing now, a couple
10 years, but I don't think it would take decades. I think
11 even though arteriolosclerosis is a long-term process we
12 know that if you lower cholesterol through drugs, two years
13 after that you start to see clinical benefit. So I think we
14 don't need to start when we're young and build up.

15 DR. WEINSIER: I'm just trying to put it in the
16 context of, you know, the recent data suggesting that, and
17 actually older data suggesting that arteriolosclerosis
18 starting at a very early age, and we're now looking at the
19 beset way to reduce that risk. Do we want to start at
20 younger ages rather than later ages when we're in advanced
21 stages of atherosclerosis. So I'm trying to put it in the
22 context of your suggestion, if I interpreted it correctly,
23 or your questions, whether this should be recommended for
24 the older population rather than the younger population?
25 Are we really at the wrong end of the spectrum, and if so,
26 can you justify it in the younger from the other health
27 standpoint, accident, risk, et cetera, that Dr. Gordis has
28 raised?

29 DR. MURPHY: Dr. Deckelbaum.

1 DR. DECKELBAUM: Just in terms of
2 arteriolosclerosis, you know, beginning at a young age, but
3 there is a number of factors that play into the development
4 of arterioscleroses, you know, through the decades. And
5 then for the final event, we heard some evidence yesterday
6 that they may be, you know, related to the -- the acute
7 event may be related to coagulation factors or sort of
8 ruptured plaques and that kind of thing, so that I think in
9 terms of young people we know that for young people that
10 alcohol intake is a major risk, and I don't think we want to
11 balance, you know, the other approaches that we can have to
12 reduce arteriosclerosis with adding another way just which
13 would affect ADL in this young age group because for that
14 young age group it's a major cause of morbidity and
15 mortality because of accidents and other causes of deaths,
16 so I don't think we really have to concern ourselves that
17 much in the young age group with alcohol intake, except to
18 say that it should be avoided.

19 DR. MURPHY: Dr. Dwyer.

20 DR. DWYER: I want to thank you for a nice
21 presentation, Meir.

22 I didn't hear -- I heard him say "in the
23 evidence," but I didn't hear what the evidence was for Dr.
24 Gordis's statement that an occasional drink causes harm to
25 the fetus. I heard a lot about -- but I didn't hear
26 anything about that, and I think before we take on the one
27 shot a day during pregnancy, we've got to be very sure that
28 the data are there.

29 The second thing is on breast cancer risk I'm

1 still not clear about that from the presentations, and I
2 think we need a little more clarification perhaps on that.

3 And the final thing is I would have -- that we
4 somehow come out very strongly and say that the definition
5 of "moderation" for those who have problems with drinking is
6 zero. You can't sort of fool around with it. You just
7 cannot drink.

8 DR. STAMPFER: Let me just briefly respond to
9 those three.

10 I think for the first one, in terms of the
11 pregnancy, I don't think that there is data for the
12 occasional drink, and I don't think that's what Dr. Gordis
13 was intending, but there are two points of accumulating
14 data.

15 One is that in terms of the threshold for regular
16 consumption there is, I think, more data now -- I can't cite
17 it to you right now, but I can get it to you -- that even
18 low levels of regular consumption are harmful, but we don't
19 have data for, you know, the occasional glass of wine.

20 But the other point of data is an interesting
21 survey that -- in the pack of material that was sent to me
22 where it was a survey of pregnant women asking them about
23 what their understanding was of alcohol during pregnancy,
24 and basically they -- their take on this was that it was
25 actually okay to drink levels. When alcohol was supposed to
26 be limited during pregnancy, the interpretation was limited
27 to, you know, two or three drinks and day and basically not
28 to get drunk very often when you're pregnant. So there is
29 a widespread --

1 DR. DWYER: I need to see that data because I
2 don't remember reading anything at all like that.

3 DR. STAMPFER: Yeah. Well, I'll send you the
4 paper.

5 Basically, there was a striking, striking to me,
6 striking lack of appreciation of the importance of limiting
7 alcohol during pregnancy, and I think if we had a statement
8 that was interpreted as permissive, this could be taken out
9 of context. So that was the -- but I agree with you, I
10 don't know of any data for harm of an occasional glass of
11 Chardoney or even Zinfendel.

12 Let's see, the second was the breast cancer, and I
13 think there is controversy here. My read of the data is
14 basically the same as what Tim presented, which is a real
15 increase in risk of about 10 percent or so with the one
16 drink a day level, and to me, a 10 percent increase in risk
17 of breast cancer is not a non-event; that that's an event.
18 We don't know how to -- we don't know of very many ways to
19 lower breast cancer risk.

20 I think we should consider putting something in
21 the guidelines to inform women, or we should at least
22 discuss it anyway.

23 And the third point about problem drinking, I
24 agree with you entirely. They should be in a category of
25 don't drink at all.

26 DR. MURPHY: Dr. Lichtenstein.

27 DR. LICHTENSTEIN: I would just say as women in
28 child-bearing age it's probably reasonable to err on the
29 side of caution because they tend to have low HDL, or excuse

1 me, low LDL and high HDL levels, so there is probably not
2 much progression of disease in that specific age group.

3 I guess I would take some, or question the
4 assumption that alcohol intake via HDL could have an impact
5 on disease risk in short, relatively short period of time
6 because I don't think it's particular comparable to the data
7 on drug intervention and disease risk where you may see an
8 effect in two years because the effect with drugs are so
9 much more dramatic on LDL than one would expect to see with
10 alcohol in HDL.

11 And lastly, I'm just wondering, is there -- not
12 all HDLs are the same, HDL particles. And I'm just
13 wondering, is there any HDL data, I mean, there is a
14 classification on the basis of density, HDL-2 and 3 and we
15 know that there are differences with respect to reverse
16 cholesterol transport, and then there is another
17 classification depending on whether there is A2 on the
18 particle versus just A1 only on the particle, again,
19 differences with respect to HDL function? Is there anything
20 known about that? Would that at all be helpful with respect
21 to this issue?

22 DR. STAMPFER: Yeah, alcohol raises HDL-2 and HDL-
23 3. It raises HDL-3 proportionately more. The relevance for
24 risk of heart disease is not -- it's controversial. Some
25 studies, including mine, find reduction for both HDL-2 and
26 HDL-3 in relation to risk of heart diseases. But, yeah,
27 both types.

28 In terms of the APOS, I have to go back and check.

29 DR. MURPHY: Dr. Grundy?

1 DR. GRUNDY: I wanted to make a general comment.
2 First, outside the evidence, I think, and the literature
3 suggest that alcohol raises CETP, which is not supposed to
4 be such a protective event, but anyway that's kind of an
5 aside.

6 You know, when Dr. Gordis was here I asked him the
7 question of what the recommendation ought to be, and he said
8 we ought to leave it what it is. Now, I don't know how you
9 feel about that. I'd like to ask you that, whether that's
10 an adequate recommendation that we have now. But in
11 thinking about this and let me just propose this and see if
12 you agree with me or not. It almost seems like we have kind
13 of maxed out in this country on the benefit that can be
14 derived from alcohol, if there is a benefit. About two-
15 thirds of the people drink, what we heard, and maybe one-
16 third don't, and the two-thirds that do, they will have
17 gotten the benefit that there is to get. The one-third that
18 don't at least, you know, a portion of those, that probably
19 half of that, maybe a sixth of people are not going to drink
20 no matter what. They have strong beliefs that they
21 shouldn't and maybe there are alcohol problems in their
22 family, so they're not going to.

23 So you're left -- if you're going to be positive
24 about it, a very small portion of the population would
25 derive some benefit from a positive statement. But this
26 moderation statement almost seems like it covers both sides
27 adequately. I just wanted to throw that out and see if you
28 agree with that as a general comment.

29 DR. STAMPFER: Yes, I do. I think there are -- I

1 would not propose to change this slogan of the guideline,
2 but I think there are a few places, just minor tinkering
3 where we could just clarify the issues a bit. But since you
4 asked my opinion about it, I think it's pretty close to
5 where I'd like to see it.

6 DR. MURPHY: Okay, Dr. Deckelbaum.

7 DR. DECKELBAUM: Just two points relating to women
8 actually. You brought up an interesting point and I don't
9 know if there is any data on it, is that, you know, benefits
10 of folic acid and other micro nutrients probably come before
11 conception. And you brought up the point whether alcohol
12 risk. Is there any data about alcohol, you know, prior to
13 conception or post-conception in terms of the risk?

14 And the other factor related to women, I think you
15 may have addressed it in the September meeting, but I don't
16 really, is it's a very striking cutoff between men and women
17 in terms of one drink versus two, two drinks is moderate for
18 men and one for women. So do body fat and weight
19 differences account for women being discriminated against
20 here with one versus two?

21 DR. GRUNDY: I'm afraid we're --

22 DR. DECKELBAUM: You can see which side I'm on.

23 DR. KUMANYIKA: I'm under the impression that both
24 the dilution's base and the first pass metabolism are
25 working against women there, so there may be enough factors
26 for that to be a real, a real difference by gender.

27 But I had another comment unless somebody else
28 wants to contribute to the one versus two.

29 DR. MURPHY: Go ahead.

1 DR. DECKELBAUM: Is there a science base for that?

2 DR. STAMPFER: No, it's --

3 DR. DECKELBAUM: It's 100 percent --

4 DR. STAMPFER: Yes, it's -- it's both body size.
5 cholesterol Shirika was saying, there is evidence for that.
6 It's not just pulled out of thin air.

7 DR. DECKELBAUM: I know, but when you look at
8 your --

9 DR. STAMPFER: And in fact, when we look at our
10 data on the protective side, you also see difference in that
11 the reduction of risk of coronary disease, as Johanna was
12 pointing out on that side, you could see the one to five
13 grams a day. That's half a drink per day, you've already
14 saw pretty substantial reduction of risk of coronary disease
15 in women. But in men, it's shifted over. So for both
16 benefit and risk, inebriation and so on, women are more
17 sensitive.

18 DR. LICHTENSTEIN: Can I comment on that specific
19 one?

20 DR. MURPHY: Sure.

21 DR. LICHTENSTEIN: Aren't also the enzymes that
22 are on metabolized alcohol, they are inducible, so there are
23 a lot of other factors besides just first pass and just body
24 water space, because if you're a habitual drinker, you're
25 going to end up clearing it a lot faster than if you're a
26 binge drinker.

27 DR. DECKELBAUM: But the other point in terms of
28 conception and alcohol.

29 DR. STAMPFER: Well, with folate, it's a time

1 of -- specific time that the fetus is developing at the
2 very, very early stage. I don't know of any data. Does
3 anybody know of data that's sort of specific for the very
4 early --

5 DR. LICHTENSTEIN: Yes, Richard, the 1990
6 committee set the two and one for the first time, and they
7 based it on -- I don't remember whose work it was, but it wa
8 reported in The Diet and Health Study, but I believe it was
9 only one study at that time, and it was confirmed during
10 their deliberations by some work by Charles Lieber, and we
11 could get you that paper. There may have been work since
12 then, but that was the basis on which they did the initial
13 two and one, I believe.

14 DR. MURPHY: Okay, Dr. Kumanyika.

15 DR. KUMANYIKA: The other point that I had has to
16 do with the way this guideline is framed, not the statement
17 but the text. It discusses alcohol consumption as if the
18 main reason for deciding whether to drink or how much has to
19 do with the fact that it's a food or a beverage. And the
20 other information about whether one would decide to drink or
21 not is a -- you know, after you've thought about all the
22 health benefits or risk, then there area certain people who
23 shouldn't partake of this because of, you know, children,
24 adolescents and so forth. And it seemed to me after
25 listening to Dr. Gordis that it would be okay to talk about
26 alcohol consumption as a more general factor, and then say
27 that for those who consume alcohol, there may be these
28 health issues, because right now it just seems -- it seems
29 backwards in terms of the social consequences of alcohol

1 consumption, the discussion is basically dietary, even
2 though it's a dietary guideline, but there could still be an
3 opening paragraph or something to talk about the decision to
4 consume alcohol and what that might do to you, and then talk
5 about specific health issues.

6 Do you -- you don't know what I mean?

7 DR. STAMPFER: I'm not sure what you're
8 suggesting.

9 DR. KUMANYIKA: Well, who should not drink, people
10 who plan to drive and so forth? The emphasis seems wrong --

11 DR. JOHNSON: I get it.

12 DR. KUMANYIKA: Do you know what I mean?

13 DR. MURPHY: I think what she is saying that you
14 should make the decision first if you're going to drink or
15 not drink. And if you choose to drink, there are these
16 added health benefits, but don't choose to drink for the
17 health benefits if there is other issues like you're
18 pregnant or you're planning to have a child or you have
19 alcoholism in your family, I mean that should come first.

20 DR. STAMPFER: So you think who should not drink
21 should come first; is that what you're saying?

22 DR. JOHNSON: Yeah.

23 DR. STAMPFER: Okay.

24 DR. KUMANYIKA: Or the decision to drink or
25 something about what we know about drinking, and that's
26 exactly what I meant; not to decide on the basis of your
27 heart disease risk.

28 DR. GRUNDY: What you just said worries me; that
29 if there should be any implication here that you should

1 drink for health reasons, that bothers me. If you do drink,
2 you know, maybe you get some benefit probably, but not to
3 make a chose. I think Roland brought that up too. Anything
4 in here that would encourage people to drink for health
5 reasons bothers me. And I think it like Dr. Gordis. I
6 don't know how you feel about that, but to start drinking
7 for that purpose.

8 DR. STAMPFER: Well, I think it's too big of a
9 risk. I think the science alone -- if I could be sure if I
10 recommended a 60-year-old who didn't drink or a nation of
11 people that old, if I could be completely sure that they
12 would adhere to the guidelines and drink moderately, then
13 I'd say the science support it. But we know that that's not
14 true and we can't make up these guidelines in isolation from
15 the real world, and therefore I agree with you. I don't
16 think we should say in the slogan or the text to recommend
17 drinking. I agree with you even though I think
18 scientifically it could be justified.

19 DR. MURPHY: Okay. Who was first? Dr. Johnson.
20 I don't know.

21 DR. JOHNSON: I just wondered if it makes sense in
22 terms of the alcohol and breast cancer thing to somehow
23 suggest that women need to assess their risk fort he
24 different diseases. I mean, that's a lot to get into but
25 there are certain known risk for breast cancer, there is
26 known risk for heart disease. And as a woman, I suppose you
27 have to balance those in making your decision.

28 Does that make sense?

29 DR. STAMPFER: Yeah, it makes sense. I don't know

1 how to put it in the text, but I welcome your suggestion.

2 DR. MURPHY: Dr. Weinsier.

3 DR. WEINSIER: Yes, real quick. Rachel keeps
4 reminding us, and I think it's important, that with the
5 limited calorie intake for the average U.S. population every
6 choice has an impact and probably a critical impact. We're
7 talking about here for women, maybe a five percent; you
8 know, swing for men, maybe, you know, five, six or seven
9 percent swing in calorie intake if you choose to drink
10 moderately versus if not.

11 And I'm trying to think through what I would have
12 to give up for the sake of taking this additional alcohol.
13 In the studies that show a benefit -- in the studies, Meir,
14 that show the benefit of short-term intake, what calories
15 were substituted? How was the HDL increase demonstrated?
16 Was this addition over and above the basic diet and you're
17 adding more calories?

18 DR. STAMPFER: You mean in the controlled studies?

19 DR. WEINSIER: Yeah.

20 DR. STAMPFER: I don't recall. I think it was --
21 in the control studies, I'm not sure. I think it was
22 carbohydrate. But in terms of what happens in populations
23 there are data to -- there are data available. Basically,
24 it's interesting. In women it's pretty much substituted for
25 sugar, calorie for calorie on the average.

26 DR. WEINSIER: So when women choose to drink
27 alcohol they are usually substituted for --

28 DR. STAMPFER: Their sugar goes --

29 DR. WEINSIER: Oh, for sugar?

1 DR. STAMPFER: Their sugar goes down. Sugar
2 specifically, yeah. But for men, there is no specific
3 change in the diet composition that goes along with
4 drinking, so it seems to just substitute for just lower
5 calories across the board.

6 DR. MURPHY: Okay, Dr. Grundy and then Dr. Dwyer.

7 DR. GRUNDY: You didn't know that alcohol doesn't,
8 you don't have to substitute for either one. It doesn't add
9 to weight gain. It's burned up independently of caloric
10 intake.

11 DR. WEINSIER: If I recall those date, we've seen
12 both sides. Doesn't it depend on the level intake? It's a
13 Luzin study. That was eight years ago, give or take about a
14 year. I thought that it did substitute it. The levels they
15 were using which I think were in the --

16 DR. GRUNDY: Could substitute but, you know, we've
17 done metabolic studies where we substitute 20 percent of
18 calories and it has absolutely no effect on weight. It's
19 burned up independently of the other calories. I mean, we
20 probably don't need to get into that.

21 DR. STAMPFER: Yes, it's probably a separate
22 issue. I was thinking about the nutrient content of the
23 other foods, not so much the calories, but that's an
24 important point too.

25 DR. MURPHY: Dr. Dwyer.

26 DR. DWYER: Just the same point, and maybe we
27 could address it later, but it's the whole issue of appetite
28 hunger and what these alcohol calories do. I believe it's
29 Dr. Hall said it at Davis who suggested that there is a

1 bypassing of satiety mechanisms, and so the question you
2 asked about the substitution, maybe you've answered it
3 metabolically, Scott, but it struck me that there is a
4 literature on that; that it bypasses. So it sort of doesn't
5 get counted by the mechanisms regulating food intake. And
6 if that is in fact true, then there might be some problems
7 in terms of weight.

8 DR. MURPHY: All right. We need to move on. Are
9 there any final comments on the alcohol discussion?

10 Yes, Dr. Lichenstein.

11 DR. LICHTENSTEIN: I think perhaps maybe there
12 should be some advice also for individuals if they are
13 uncertain to consult with their physician, because I'm
14 thinking of individuals that are hypertensive. There is a
15 relationship between alcohol consumption, blood pressure and
16 individual that are hyperglycemic, that alcohol can
17 exacerbate that situation. So perhaps just some cautionary
18 note to consult the physician.

19 DR. MURPHY: That makes sense to me.

20 Okay, very good. Let's move on then, and is Dr.
21 Dwyer ready to talk about food safety perhaps?

22 DR. DWYER: I would like to thank Etta Saltos who
23 up until today has been collecting references, and I believe
24 she is going to pass out three that are somewhat relevant to
25 some of the custodians that came up yesterday about the
26 amount of -- the amount of foodborne disease, bacterial
27 disease that you could contribute -- attribute to home-
28 prepared, things going on in the home vis-a-vis the food
29 system.

1 I'd also like to thank Joan Lyon and Shanthy, Dr.
2 Johnson, whose gone through many drafts with me, Dr. Tinker
3 who is in comunicado, but I'm sure has been reading all of
4 these drafts, and Dr. McMurry for collecting many things.
5 In addition to that, someone who is not here right now, but
6 who's been very helpful, has been -- has been the people in
7 various other parts of USDA who deal with this on an
8 everyday basis, such as Sandy Fansinoli, and the people at
9 the National Agricultural Library.

10 The task force report is in your booklet here, and
11 what I'd like to do is to go through very quickly the
12 possible guideline or slogan, "Handle food safety from
13 market to table." The list of the consultants is given in
14 your books and it's about 20 people because this isn't
15 something that's certainly my area.

16 Is there a clicker up here or do I click? That's
17 fine. Thank you.

18 So I'll try to not duplicate what Dr. Woteki said
19 yesterday -- thank you very much -- and just go quickly
20 through this because I know you're all tired.

21 The first things that we thought needed to be
22 considered in such a guideline should it be deemed
23 appropriate would be to emphasize this whole notion of
24 keeping food safe by handling them safely from market to
25 table, and you'll notice that the various things that are
26 suggested there -- clean, separate, cook, chill, follow the
27 labels safely, and if you doubt, throw it out -- are pretty
28 much in line with the Fight Back Campaign that Dr. Woteki
29 talked about yesterday.

1 The second thing is to identify and define what
2 foodborne illness is, and basically, as you know, there are
3 many different kinds of foodborne illness, but this would
4 focus primarily on bacterial, and I'll try to get around to
5 why that would be in a few moments.

6 But basically, bacteria, bacterial foodborne
7 disease is really the most common and it's probably the one,
8 at least from existing data, not from maybe there is some
9 data we haven't been able to find. It seems as though our
10 experts felt that that was the easiest form of illness for
11 consumers to do something about if you really believe this
12 is something where consumers should have -- be able to do
13 something rather than the various food safety branches of
14 state, local and federal governments.

15 So that's basically it. The other kinds of things
16 that would be covered in the page or two would be the
17 rational -- the first essential for healthy eating be that
18 food must be safe when they're produced but also they need
19 to be handled after the time they are purchased, whether
20 it's at a market or a store and eaten.

21 And, again, to emphasize continuity, it isn't that
22 producers, distributors and preparers of food outside the
23 home aren't important -- they are very important -- but
24 consumers are a critical link in that chain as well.

25 The other thing is this enormous and probably
26 growing recognition that foodborne illness is really quite a
27 prevalent public health problem. The estimates are given in
28 the text from the latest data we could find, and some more
29 data is being passed out now.

1 The other thing we thought might be useful would
2 be some kind of a little guide like this. It's in text in
3 your books but here it is in graphics, just summarizing
4 thoroughly cooking of foods.

5 People who need to be especially careful,
6 certainly these groups -- pregnant women, very young
7 children, older adults not only because of possible
8 immunosuppression, but when you get above 85 where large
9 numbers of people have problems with memory, if they are
10 still living independently at home there are questions about
11 how long things stay in the refrigerator, and then there is
12 a large group of immunosuppressed people, both, I guess, the
13 people who everyone thinks of right away is patients with
14 HIV, but there are a large number of people in chemo therapy
15 for various disorders, be it cerotic arthritis down to other
16 things, organ transplants, renal disease, so forth, all
17 sorts of weaken immune systems.

18 The section would conclude with a little bit about
19 a few useful resources: web pages, local and state
20 resources and the USDA hot line.

21 Now, that basically is -- that is pretty much what
22 we've suggested. There are a couple of other -- in the
23 course of interviewing about 100 people in all, a lot of
24 people had different ideas about what a good slogan would
25 be, and the slogan "Handle food safety from market to table"
26 was one that I think that you and I felt probably was the
27 best of all of them, but that doesn't mean that one of the
28 others might not be more appropriate. Some of them are
29 listed here. Some people thought "The fight back" was best,

1 some "Eating a variety of foods," some -- this is the
2 Supreme Court one that has everything in it. These are
3 written by lawyers, these two. You can see that there is
4 excess of verbiage. But they are all good. The question is
5 whether they are actionable and whether they are memorable
6 because they are so long.

7 So basically that's the kind of thing that we
8 think might be useful in a separate guideline.

9 Now, what are some of the -- oh, I've got a few
10 other suggestions. There are some others. They are all
11 listed in your book with the name of the groups or group
12 that recommended them.

13 Let me just spend five more minutes talking about
14 a couple of issues that came up yesterday. The first is
15 this issue about reporting, and in reading this literature
16 it becomes apparent right away that reports of foodborne
17 illness are really quite dramatically under-counted. And I
18 think that this sort of bull's-eye diagram illustrates what
19 we found.

20 There is a small group of cases, the center of the
21 bull's eye, if you will, where the food, the agent and the
22 causes are all known, and there are some cases where this
23 all fits together.

24 There is a much larger group of cases where there
25 are reports, but fecal samples haven't been taken, or there
26 is no sample of the food available so you really can't tell
27 what's going on. And then there is a very large group of
28 cases where the level of suspicion is high but it's not
29 clear.

1 Now, that center of the bull's eye includes a lot
2 of cases in institutions, for example, in hospitals, getting
3 to Dr. Grundy's very good question yesterday. How do you
4 really know the percent of cases that are due to things
5 people do after they buy the food or purchase the good in a
6 store. You can answer that to some degree in a hospital or
7 in another institution where the food is all produced andy
8 you could take samples of the food and culture them
9 theoretically, and then because the people are in an
10 institution, if they get sick you can also easily get
11 biological specimens and get some idea of what made them
12 sick.

13 But aside from those sorts of situations it's very
14 difficult for me to think of how to -- how to find out with
15 a great deal of precision exactly how many cases of disease
16 are caused by things where the food left the market pristine
17 pure, and then it was contaminated later. So it's an issue
18 with the techniques we've got right now that remains, I
19 guess, with the Scottish verdict, unproven but highly
20 suspicious.

21 The handouts that Dr. Saltos has been passing out,
22 I guess, summarize the views of the IFTX expert panel of
23 food safety and nutrition in 1995, and some other work by
24 Allen Levy about what their view -- these people are experts
25 and I certainly am not -- about this issue.

26 So we're pretty sure that foodborne illness, this
27 bacterial foodborne illness particularly, is under-reported.
28 We know that CDC investigations compared to estimates, such
29 as the ones we heard yesterday, are quite dramatically

1 different. Whether it's illness or death, this is illness,
2 the CDC investigated over on the left estimates, the low
3 estimate and then the high estimate, an enormous
4 variability, but clear really very serious under-reporting
5 Public Citizen claimed in a recent manifesto that
6 about one to five percent of foodborne illness was actually
7 reported. That was their view.

8 Now, why is it that this under-reporting occurs?
9 Some of the issues, I think, we've already talked about, but
10 the CDC definition calls for a couple of cases of similar
11 illness for ingestion of food. So if you're an old lady and
12 you forgot that it's now March 8th or 9th and the turkey has
13 been in the refrigerator since November 25th, believe me, I
14 worked on a hot line in Massachusetts for about six years,
15 and that is not a hypothetical question. We constantly got
16 calls in March about what do you think about the
17 Thanksgiving turkey, and that's what led to the "if in
18 doubt, throw it out."

19 You have to have two or more cases for CDC to call
20 that an event. The small outbreaks tend to be invisible.
21 You tend to confuse them with the GI flu. We lack methods
22 at present, and hopefully this won't be forever, to rapidly
23 detect pathogens in food and in blood and in stools, so we
24 don't have the easy techniques we do for some other things
25 right now, and we have a voluntary reporting system where
26 every state is not equally determined to find these
27 problems. We have some states that are doing an admiral
28 job, but it's a real problem.

29 The other problem is what happens as a result of

1 this, how many of us are out of work because of events that
2 take place. Again, our uncertainty because of the reasons
3 I've already suggested is considerable, but the estimates
4 are really quite dramatic in terms of lost productivity a
5 year. This is about -- ranging from about 10 to 40 billion
6 dollar a year, so we're not talking about small potatoes
7 here -- wherever they go, whatever food group they go in.

8 The other thing, Richard Lavens at Harvard and
9 others have talked about the constantly emerging, emerging
10 changes in infectious disease, and one example perhaps of
11 this is foodborne illness. The first reason, we know that
12 foodborne illness is going to rise in the next 10 years, and
13 I believe Dr. Woteki said this in her swearing in, is that
14 we know that our statistics are getting better. So
15 regardless of what -- even if we -- even if things are going
16 along the same, as reporting gets better, the perception of
17 foodborne illness rising will clearly be there.

18 The second reason why foodborne illness probably
19 may be on an upswing is demographics. We have an aging
20 population compared to 20 years ago, there are more people
21 who are immunosuppressed today both because of HIV and
22 transplants and chemo therapy than probably there ever were
23 before.

24 And, finally, foodborne illness is small but I
25 think preventable proportion, involves lack of consumer
26 awareness and education.

27 There are other reasons too at different levels
28 farther back in the food chain, but those three certainly
29 may be important.

1 The other big reason why foodborne illness is
2 probably on the increase is because of environmental
3 exposures that are increased, and I've tried to outline them
4 in the draft rationale we put together. Clearly, there are
5 ne strains of foodborne bacterial, and Cathy talked about
6 some of them yesterday. We have sufe and other minimally
7 processed foods in supermarkets. These are very high profit
8 margin items, the suvid where you draw a vacuum and the food
9 stays sanitary or safe for quite awhile. Then there are
10 also things like partially cooked foods which are sold for
11 take-out. All of those things increase environmental
12 exposures. And then the issue of a global food economy,
13 think Dr. Woteki talked about that. There isn't good
14 evidence that foreign foods are less hygienic than ours,
15 nevertheless it's a global economy now.

16 What's argued in the text is that interventions
17 can prevent and lapses can cause foodborne illness, and
18 these interventions include things that are under our own
19 control as consumers, as well as things that must be
20 controlled by our elected represented, and appointed
21 representatives in government at other points in the food
22 chain.

23 But there are four essentials that also can occur
24 at one. One are bacterial cells as spores; second, the food
25 vehicle; third, conditions allowing bacteria to survive and
26 thrive; and, fourth, a vulnerable food consumer.

27 So basically, our lifestyles as well as our food-
28 use patterns at home and in our daily lives can pose
29 needless and preventable foodborne illness hazards, and it

1 seems to me that we need to do something about it. We can
2 avoid some of these problems with appropriate handling, both
3 the food prepared outside the home and then food that's
4 bought at the store where the handling depends on what we do
5 to it.

6 So why bother to do all of this? First of all,
7 it's an actionable measure that we can take to eat in a
8 health way that really does make a difference in terms of
9 morbidity as well as mortality. It's something where
10 consumers are concerned, and where they are worried, and
11 where they do need help. We need to bother about it because
12 the dietary guidelines really weren't conceived at the very
13 beginnings as solely for chronic degenerative disease. They
14 are about health. They are not about a specific turf of the
15 medical area.

16 And if we look at Healthy People 210, I think
17 everybody got a copy of it, it's the great, big, fat, yellow
18 book. If you look at Healthy People 210, it fits very well
19 in with some of the things that experts in our various
20 cabinet-level departments as well as thousands of experts in
21 public health from all around the country are thinking of.

22 Now, another question that came up last time and
23 that we've tried to answer in the 15 or so pages of text is
24 what really works. Are there any examples of things that
25 involve information to consumers that really work?

26 Well, the first thing that we talked about
27 yesterday was the Fight Back Campaign, and that's only been
28 up and running now for about a year, and so it's a little
29 early to look at hard end points. They've got some

1 interesting focus group data. They have some other data
2 that's referenced in your text. But there is not too much
3 yet on that campaign.

4 The other campaign that we saw fairly good data
5 on, talking to Dr. Levy, I believe it was something out of
6 the Department of Health and Human Services a fair number of
7 years ago where there were demonstrable effects from a raw
8 shell fish campaign, and those of you who are in FDA may be
9 able to talk more about it, but it's basically the problem
10 hepatitis associated with people eating raw shell fish. And
11 if any of you come to Boston, you'll go to the Union Oyster
12 House and see people eating raw shell fish there.

13 It turns out that people who do that on a chronic
14 basis have very, very high risks of hepatitis, so the
15 targeted group was those people, and they were able to show
16 significant decreases in that particular behavior.

17 So does this kind of -- this kind of informational
18 campaign work? I guess we'd have to say probably yes; more
19 evidence probably is also going to be helpful.

20 In terms of some other questions that were asked
21 by Dr. Sutor, some of those things were mistakes in the text
22 and I think they are changed in the version of the text that
23 you have. Thank you.

24 DR. MURPHY: Thank you. Anybody have questions?
25 Everybody is numb.

26 Dr. Stampfer.

27 DR. STAMPFER: Johanna, this thing called Factoid
28 Watch came around.

29 DR. DWYER: Yes, I'm sorry. I just got that too,

1 Meir.

2 DR. STAMPFER: I'll be curious to get our reaction
3 when you've had a chance to read it.

4 DR. SALTOS: We just got this from FSIS and they
5 did give us some better references, journal articles that
6 are more vigorous but we couldn't get copies for everybody,
7 but we can get those.

8 DR. STAMPFER: It can wait until tomorrow then.
9 You don't have to read it right now, Johanna.

10 DR. DWYER: Okay.

11 DR. MURPHY: All right, Dr. Grundy.

12 DR. DWYER: Very good. It says that food
13 poisoning is a phony figure from the Columbia Journalism
14 Review. Well, I don't know. We found all of the data we
15 could, and I think that food poisoning is a real problem. I
16 don't know what the exact numbers are, and I think, as I've
17 indicated on the slides I showed you, that the confidence
18 estimates are rather broad, but they are all far above
19 levels of zero.

20 DR. GRUNDY: You know, I've noticed that most of
21 the time when I get sick from eating food, it's from eating
22 out and not from eating at home, and I know most of the
23 emphasis here was on eating at home.

24 Is there any -- have you been thinking about
25 having any comments about trying to avoid when you eat out?

26 DR. DWYER: Well, Scott, the way we tried to go
27 about it, and Dr. Johnson may want to comment too, was to
28 pick the things where people really all agreed. It turns
29 out, it's the same as heart disease. Until you get into it,

1 you never realize how many arguments there are about it.
2 And so to make a statement like "Don't be an idiot, don't
3 eat sushi unless you know where its source is from," or
4 "Don't eat shell fish unless you're sure" or something like
5 that seemed to be less fraught with consensus, if you will,
6 than what's in the draft that's presented there.

7 We tried to rely on our information experts from
8 the CDC, the FDS, the FSIS, and universities around the
9 country to try to get what it is that they all felt was the
10 nub of agreement. There certainly are additional things,
11 additional caveats that could be added, but I think what's
12 here is sort of the kernel, if you will, of what everybody
13 agrees on. They really -- there doesn't seem to be much
14 dispute about that.

15 You will notice that I don't think we included any
16 kinds of estimates of millions of people who were dying or
17 dead as a result of this because, again, these are very
18 difficult issues to resolve, so we stuck to what we know the
19 best. That's the reason also for sticking with bacterial
20 foodborne illness rather than including -- trying to get
21 into things about pesticides and so forth. Again, it's just
22 that the experts in this field, of which I am not one, seem
23 to feel that those are the messages that are unlikely to be
24 reversed in four or five years or 10 years.

25 DR. JOHNSON: I just wanted to add that I think
26 this is a case for us to really think about the broad policy
27 implications of the dietary guidelines. In the conference
28 calls that Johanna brought together, and she brought some
29 really excellent people, and we kept hearing the message

1 over and over again from people in local health departments
2 who are clearly doing food safety programming, from people
3 in the extension system that are doing food safety
4 programming and the fact that they use the dietary
5 guidelines as a teaching tool, that they felt that
6 incorporating food safety in with the dietary guidelines
7 really help, would help them in terms of strengthening some
8 of their educational programs that they provide to the
9 public.

10 And also thinking about the school nutrition
11 programs as well, certainly food safety is an absolute
12 critical component of any massive feeding program, of which
13 nutrition is one, so I think they also felt that
14 strengthening that aspect in the guidelines would help them
15 in terms of overall policy.

16 DR. DWYER: One final thing I didn't get a chance
17 to do slides of because I didn't get it until yesterday, but
18 you might enjoy a copy of it. Etta, I don't know if we can
19 get copies --

20 DR. SALTOS: We can get copies.

21 DR. DWYER: Can we get copies?

22 What this does, it's called "The Food Safety
23 Educator." It just came out, I guess. What it does is
24 focuses on the interesting consumer research that's been
25 done now on food safety education, and it goes into rather
26 exhaustive detail about some of the questions that were also
27 raised yesterday about the kind of food safety risks in
28 homes, and it goes through a series of various consumer
29 perceptions, and some of the things that people are not

1 seemingly getting, if you will, with respect to this issue,
2 people just don't seem to know, for example, that some
3 people are at higher risk of foodborne illness than others.
4 They don't seem to really understanding what to do about
5 things like cross-contamination and so forth. So there is
6 some pretty good consumer data now suggesting that this
7 isn't just sort of nonsense.

8 DR. MURPHY: Okay, any other comments?

9 All right, we're a tired group, I think.

10 Nonetheless, we all have another large assignment. If you
11 look at your agenda, we're next supposed to talk about a
12 review of the third day of DGA. I think we recommend we
13 skip that and we keep the third day just what it is, and
14 move on to the working groups that we're going to have, at
15 least the members of the committee are going to have for the
16 rest of the afternoon.

17 So, in effect, we are adjourning the public
18 meeting for now but please, members, don't go away. We need
19 to talk about the logistics as I look at Linda and hope that
20 she has information on the logistics.

21 (Whereupon, at 3:28 p.m., the meeting was
22 recessed, to resumed at 9:00 a.m., on Wednesday, March 10,
23 1999.)

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In Re: Dietary Guidelines Advisory Committee
 Name of Hearing or Event

N/A
 Docket No.

Washington, DC
 Place of Hearing

March 9, 1999
 Date of Hearing

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