New scientific evidence showing a beneficial relationship between cholesterol reduction and coronary heart disease (CHD) reduction, which was originally published by the Lipid Research Clinics Coronary Primary Prevention Trial (CPPT), was the focus of a workshop sponsored by the National Heart, Lung and Blood Institute in June 1984 in Bethesda, Maryland.

The NHLBI Director, Dr. Claude Lenfant, opened the sessions and explained that the purposes of the workshop were to review the CPPT results and present additional Trial data; to relate CPPT results to other clinical, epidemiological, and experimental research findings; and to identify issues arising from the Trial and their implications. A final objective was to identify areas for new research.

Coronary Primary Prevention Trial

Background

Dr. Basil Rifkind described the scientific evidence relating to CHD and the research opportunities available in 1969 to 1971 when the CPPT was planned. At that time, numerous epidemiologic, pathologic, and animal experimental studies had already shown that CHD, a major cause of mortality and morbidity in our country, was associated with elevated plasma cholesterol levels and two other major risk factors, cigarette smoking and hypertension. The potential for reducing the incidence of the disease by lowering plasma cholesterol made a clinical test of intervention highly desirable. A test of the hypothesis that lowering the plasma cholesterol level results in a reduction of risk for CHD was proposed. A double-blind trial of diet intervention was not feasible, and therefore a test using a drug to lower cholesterol was undertaken.

Experimental Design

Dr. O. Dale Williams outlined the design of this test. It was a controlled clinical trial with randomized and double-blind treatment assignment to a cholesterol-lowering drug or placebo in healthy men at high risk for CHD because of hypercholesterolemia. The bile acid sequestrant, cholestyramine, was chosen as the study drug because of its demonstrated efficacy and low level of toxicity. Identical diet instructions designed to reduce plasma cholesterol by 3% to 5% were given to the drug-treated group and the placebo group. The stringent design goals included: sample size, participant characteristics, randomization of treatment assignment, treatment efficacy and adherence, and follow-up duration and completeness. The standardization program of the Core Lipid Laboratory serving each clinic was described by Dr. Paul Bachorik. The achieved goals of high accuracy and precision and low inter- and intralaboratory variation for cholesterol and triglyceride assays were outstanding improvements over contemporary clinical laboratory performance and contributed significantly to the efficacy of the Trial. They further demonstrated the standardization program's potential for high quality in other lipid and lipoprotein measurements. Dr. John Wilson summarized the adjudication procedure for primary and secondary endpoints, including the definition of criteria, collection of clinical data, operation of the evaluation teams, and quality control procedures.

Major Findings

Drs. Peter Kwiterovich and Donald Hunninghake reviewed the major findings of the Trial. The drug treatment achieved the goal of lowering total cholesterol levels mainly by lowering low density lipoprotein cholesterol (LDL-C). Comparison of the two randomized treatment groups demonstrated that the drug group's lower levels of total cholesterol and LDL-C were accompanied by a significantly lower combined incidence rate of definite nonfatal myocardial infarction and CHD deaths. Similar trends were seen for other indications of CHD; there was less angina, few-

This report was prepared by the speakers and session chairmen participating in the workshop.

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Lowering Plasma Cholesterol and CHD Risk

The relationship of plasma cholesterol lowering to reduction in CHD risk was considered extensively by Dr. David Gordon. In each treatment group, both baseline and post-treatment levels of total cholesterol and LDL-C were significant and quantitatively similar predictors of subsequent CHD. A strong relationship between the change in total cholesterol and LDL-C and CHD risk was seen in the cholestyramine group: the greater the reduction in cholesterol, the greater the reduction in heart attack rate. In the placebo group, few men achieved substantial cholesterol reduction, and there was no significant relationship between cholesterol change and the combined incidence of definite CHD mortality and nonfatal myocardial infarction (MI). The mean reduction in CHD risk achieved in the drug-treated men was exactly that predicted by their mean LDL-C reduction.

Statistical Methods

Dr. C. Edward Davis reviewed issues of statistical methodology used to analyze Trial data that had been raised by the scientific community. These included the use of the one-sided test of the Trial hypothesis rather than a two-sided test. Explaining the rationale for this choice, Dr. Davis pointed out that a one-sided test was used because the hypothesis of the CPPT was that lowering cholesterol would reduce the incidence of CHD; if the hypothesis had been that lowering cholesterol would change (i.e., increase or decrease) the incidence of CHD, then a two-sided test would have been used. Presenting another point of view, Dr. Richard Kronmal challenged the choice of a one-sided test and cautioned that, in general, the standards of clinical research would be lowered if two-sided tests were not used. Dr. Max Halperin expressed a middle view, pointing out the pros and cons of both sides of the debate, and suggested that in the end, the resolution regarding inference would depend on consensus rather than a statistical test.

Additional Data from the Trial

Change in High Density Lipoprotein Cholesterol and Triglyceride and CHD Risk

Dr. John LaRosa discussed the relationship between high density lipoprotein cholesterol (HDL-C) and triglyceride (TG) change and CHD risk reduction. During the course of the Trial, HDL-C and TG levels increased modestly, more so in the cholestyramine than in the placebo group. The baseline HDL-C levels were negatively and significantly related to coronary risk, whereas baseline TG levels were positively but not significantly related to coronary risk. The HDL-C level, averaged over the 2 years preceding CHD events and adjusted for other variables, was significantly lower for CHD cases than for others. The change in HDL-C from baseline was a significant predictor only in the cholestyramine group, implying an independent effect of the drug in raising HDL-C and thereby lowering coronary risk. The small but significant changes in HDL-C in the cholestyramine group were associated with a significant decline in coronary risk, even after controlling for changes in LDL-C and other variables (including HDL-C). The plasma TG level, averaged over the 2 years prior to a CHD event, was significantly higher for CHD cases than the average for others, but this significance disappeared after adjustment for other variables. The change in TG level from baseline was not a significant predictor of CHD risk. It should be noted that only men with plasma TG levels below 300 mg/dl could participate in the Trial, and thus only limited conclusions about TG and coronary risk can be drawn from these results.

Changes in Plasma Total Cholesterol and LDL-C and CHD Risk

The relationship of changes in plasma total cholesterol and LDL-C to CHD risk in the CPPT diet-plus-placebo group was addressed by Dr. Charles Glueck. The data from the placebo and cholestyramine groups were analyzed separately, as if they came from longitudinal observational studies. When only primary CHD events (death from CHD or nonfatal MI) were considered, weak, nonsignificant, positive associations were demonstrated for both total cholesterol and LDL-C in the diet-plus-placebo group. However, the statistical power of this analysis was too small to rule out the possibility that diet had an impact on CHD risk similar to that of the drug.

To increase the statistical power, the combined incidence of definite and suspected CHD death and nonfatal myocardial infarction was considered in relation to cholesterol change in the placebo group. The inclusion of the "suspect" events had little impact on the estimated strength of these associations, but reduced their standard errors sufficiently that the criterion of statistical significance was met. The subsequent inclusion of the Rose Questionnaire angina and newly positive exercise tests as CHD events in
these analyses further narrowed the confidence intervals and increased the statistical significance of the associations of changes in total and LDL-C with CHD risk in men receiving no active drug. The associations between change in LDL-C and incidence of CHD events were consistently weaker in the diet-plus-placebo group than in the diet-plus-cholestyramine group; however, the confidence intervals for the two treatment groups overlapped for each definition of CHD considered, and the differences between treatment groups were not statistically significant. Thus, while it is uncertain whether cholesterol reductions achieved with only dietary instruction (and a placebo) have an impact on CHD incidence equal to that achieved with cholestyramine, and while not all cholesterol reductions in the placebo group can be attributed with certainty to diet, it is clear that non-pharmacologic, as well as pharmacologic, reductions of cholesterol were associated with reduced incidence of CHD in the CPPT.

**Graded Exercise Treadmill Test**

Dr. Cari Rubenstein discussed the predictive values of the graded exercise treadmill test (GXT) for cardiovascular disease in both an earlier LRC cross-sectional population survey and in the CPPT, in which the Bruce Protocol to 90% of maximal effort was used. Data from the population survey, based on 3832 men ages 30 to 90 years without prior myocardial infarction, showed that a positive GXT at the baseline examination was strongly predictive of CHD death. Compared with the negative GXT group, the positive group had an eightfold increase in CHD death and in total cardiovascular disease death, and a fivefold increase in deaths from all causes. Similarly, in the 7- to 10-year follow-up of the 3806 CPPT participants (men ages 35 to 59 years with Type II hyperlipoproteinemia), an asymptomatic, positive GXT at baseline was predictive of CHD death in both treatment groups. During the follow-up, the risk of developing a new, positive GXT was much less in the cholestyramine group (with lower plasma LDL-C levels) than in the placebo group. The development of a positive GXT provided the largest number of cardiac endpoints during the CPPT and was the endpoint that had the most significant difference for the two groups.

**Relation to Other Clinical Trials, Epidemiologic Findings, and Experimental Research**

**Framingham Study**

Dr. William Castelli summarized the observational epidemiologic cast from the Framingham Study, which established serum total cholesterol, LDL-C, and HDL-C levels as coronary risk factors. He stressed the usefulness of the total cholesterol/HDL-C ratio as a predictor of coronary events in all age groups. This is in contrast to total cholesterol alone, which loses much of its predictive ability beyond the age of 50 years.

**Oslo Study**

Dr. Ingvar Hjermann contrasted the results of the Oslo diet-smoking intervention study. Approximately 1200 men with top quartile cholesterol levels were divided into an experimental group and a control group. Men in the experimental group were instructed in a diet high in polyunsaturated fats and complex carbohydrate but low in total fat and cholesterol. Serum cholesterol levels dropped by 10% and coronary events were significantly less in the experimental group. Smoking also was reduced in the experimental group but coronary risk reduction was attributed primarily to cholesterol lowering.

**Oslo Study and Belgium Study**

Dr. Salim Yusuf presented data from the combined results of several primary and secondary lipid-lowering trials. He calculated the odds-ratios for major CHD events between treated and control groups, as well as the 95% confidence limits of 17 trials individually and for the several trials combined. In general, a lowering of total cholesterol and LDL-C produced risk ratios favoring the treated groups. These ratios attained “significant” p values in some, but not all, studies; however, the combined risk ratio attained a significant p value. The data gave additional evidence to support the lipid hypothesis by showing a remarkable consistency over a variety of agents and various degrees of lipid lowering. Dr. Yusuf’s talk, subtitled “There Are Two Sides to the Coin,” noted that when all-cause mortality, rather than coronary risk, was used as an endpoint, no effect from treatment was demonstrated in any clinical trial or in the combination of trials. This was because the reduction in cardiac deaths was apparently countered by an increase of similar magnitude in noncardiac deaths. Dr. Yusuf stressed that it was unclear if the increase in noncardiac deaths was causally related to cholesterol lowering.

**Coronary Drug Project**

Dr. Paul Canner reported on the postclinical trial mortality experience of the Coronary Drug Project.
(CDP) participants. The previously reported adverse effects of estrogen and thyroid were presented along with new findings resulting from a mortality follow-up study of CDP participants. These showed that nicotinic acid significantly reduced all-cause mortality, a finding that augmented the original results, which showed that nicotinic acid reduced the incidence of recurrent nonfatal myocardial infarction.

**Relationship to Other Studies**

Dr. Herman A. Tyroler summarized the relationship of the CPPT results to those of other studies, stressing in particular the internal consistency of the CPPT data and the compatibility of its outcome with prospective epidemiologic observational studies and with other clinical trials. He noted that equally important was the fact that the CPPT avoided the biases that had been problems in other studies and that could have adversely affected the data interpretation. These biases included the healthy participant effect, the adherence-placebo effect, and the prolonged time-to-efficacy effect.

**Studies on Nonhuman Primates**

Turning to evidence from experiments using nonhuman primates, Dr. Thomas Clarkson discussed his study of induction and regression of atherosclerosis by dietary manipulations. Rhesus monkeys were fed an atherogenic diet for 38 months to induce lesions. At that point, their diets were adjusted to maintain cholesterol levels of 300 mg/dl or 200 mg/dl for an additional 2 to 4 years. At those times, 16 segments of the coronary arteries were examined by quantitative morphometry methods. The lower cholesterol levels were associated with significantly fewer and less severe lesions, confirming the "cholesterol hypothesis" in nonhuman primates.

**Studies on Humans: The NHLBI Type II Intervention Trial**

The comparable effects of cholesterol lowering on coronary atherosclerotic lesion development and regression in humans were discussed by Dr. Katherine Detre, who presented the results of the NHLBI intervention trial in Type II patients. Approximately 140 patients with primary hypercholesterolemia and coronary heart disease were randomized into placebo and cholestyramine treatment groups; all followed a cholesterol-reducing diet. Coronary angiography was performed at the beginning of study and again after 5 years. The cholestyramine group experienced significantly less lesion progression than the placebo group.

**Current Prevention Trials**

Dr. David Blankenhorn outlined about 20 coronary prevention trials currently in progress which are using diets, medication, and intestinal bypass surgery in various combinations to lower plasma cholesterol levels and are using coronary angiography to quantify lesion progression or regression. It is anticipated that over the next few years much new information will become available on the efficacy of lipid-lowering in affecting the development of coronary atherosclerotic lesions in man.

**Issues and Implications**

This session explored a broad array of screening and treatment implications arising from the CPPT.

**Identifying Hypercholesterolemia**

The feasibility of identifying hypercholesterolemia was addressed by Drs. Michael Oliver and Stephen Hulley. Dr. Oliver was of the opinion that there was insufficient internal and external consistency in the CPPT results to provide a basis for extrapolating the findings to a much broader population than that represented by the Trial participants. He proposed that only high-risk individuals be targeted for cholesterol-lowering intervention, and was opposed to advising entire populations to reduce their saturated fat consumption and to mass screening of all adult men. He favored this targeting alternative on the basis that the evidence of benefit for this group is consistent, and thus the motivation of doctors and of the targeted group would be high. In focusing on this high-risk group, he suggested the following strategies: identification of familial hypercholesterolemia in families with premature CHD, measurement of plasma cholesterol in all patients with CHD who are younger than 50 years old, and establishing liaison with dermatologists and ophthalmologists who are likely to be consulted by patients for some characteristic manifestations of hypercholesterolemia, e.g., xanthoma, xanthelasma, and corneal arcus. He suggested an alternative definition of a risk factor as the degree of deviation from normal where it is certain that both detection and treatment will do more good than harm.

**Screening for Cholesterol**

Dr. Stephen Hulley asserted that population-wide screening for cholesterol has the potential to be just as feasible as the now commonplace measurement of blood pressure. The technical requirements associated with the need for repeated measurements, the education of physicians and the public, and the development of standardized laboratory methods can be overcome, as they have been with blood pressure measurement techniques. He pointed out that serum cholesterol level now meets the two criteria that determine the need for risk factor screening: evidence that intervention will reduce the incidence in some persons, and evidence that the level of the risk factor influences the intervention decision.
Treating Hypercholesterolemia

Diet

Dr. Fred Mattson opened the discussion regarding the feasibility of treating hypercholesterolemia by diet and/or drugs. He asked to whom should dietary advice be given: the patient, the wife of a male patient, or the family? He emphasized that this advice should normally be given by a dietitian rather than by the physician, since physicians lack time and specialized training in this area. Follow-up nutritional counseling should be provided at least annually after the prescribed diet has been attained.

Accordingly, some provision is needed through third-party payers to underwrite the costs of professional services by dietitians. Professional organizations, the media, public service organizations, and the food industry have a role in nutritional education for the general public. Several questions were identified: Does the cholesterol lowering in response to diet of those below the 95th percentile for total cholesterol differ from those above? At what age should dietary modification be introduced? Can responders and nonresponders to cholesterol lowering by diet be identified? Will long-term use of the American Heart Association Phase III diet produce any nutritional deficiencies?

Drugs

Dr. Virgil Brown proposed that two of the major issues to be resolved are: 1) a consensus definition of those individuals who will require drug treatment for adequate cholesterol control, and 2) agreement concerning a treatment goal. He suggested that a goal for LDL-C of 150 mg/dl might be appropriate.

In considering individual drugs, he noted that the bile acid sequestrants are attractive because their mode of action is through enhancement of physiologic pathways for cholesterol removal; however, their general acceptability is a problem because patients find them burdensome to take and their cost is too high for the average person. Nicotinic acid is a promising drug currently arousing interest, but caution must be exercised in using it with some individuals, e.g., those with cardiac arrhythmias. The fibric acid derivatives can be effective in Type III and Type III hyperlipoproteinemia, but are often used inappropriately without clear documentation of a reduction in serum lipids. Newer drugs such as mevinolin and compactin have been greeted enthusiastically even though mevinolin is only now entering the second phase of the evaluation for FDA approval. Dr. Brown concluded that the public is waiting for a clear public health policy statement to fill the current void.

Food Industry Perspectives

Mr. Walter Meyer observed that the public has recently adopted changes in the diet that would be expected to lower plasma cholesterol. For example, in the 1950s the P/S ratio of the principal visible fat was no more than 0.4 and that of the overall diet was 0.1 to 0.2; now the P/S ratio of visible fat is about 1.0 and the overall diet has a P/S ratio of about 0.5. The food industry will respond with further changes as they are influenced by regulatory policies on labeling the contents of food and even more by medical advisory statements.

Generalizing CPPT Results

The issue of generalizability of the CPPT results to lower blood cholesterol and risk levels, to women, to different age groups, and to the general population, was introduced by Dr. Henry Blackburn. He outlined the congruence of CPPT results with other trial data and observational studies, and the way the results tested the cholesterol-lowering hypothesis and supported the extrapolation of CPPT results. He outlined the logic system and criteria for extrapolation and proposed that widespread cultural factors and their associated elevated blood cholesterol levels were major determinants of the population risk of atherosclerosis and CHD. Thus, a strategy focused only on the highest risk adults would fail to address the public health issues of prevention.

Lipoprotein Structure and Analysis

Dr. Ronald Krauss discussed the generalizability of the CPPT results from the viewpoint of lipoprotein structure and metabolism. He noted that cholestyramine basically augments the physiologic mechanism for LDL and intermediate density lipoprotein clearance. A major unresolved issue is the question of whether reducing CHD risk depends on the specific effects of cholestyramine on lipoprotein structure and metabolism. What are the implications for CHD risk reduction from the metabolic effects and mechanisms of lowering cholesterol by diet vs drug interventions? The question has been raised as to whether the hepatic receptor response to cholestyramine is less in older people, since in animal models the number of receptors declined with age. Will any long-term toxicity of cholestyramine present concerns of particular significance to younger people? In women, will sex hormones affect the response to cholestyramine and its effect on CHD?

Disseminating Information

Mr. Michael White enumerated the mechanisms used by the NHLBI for disseminating information in the National High Blood Pressure Education Program, and suggested that a similar model could be adopted for disseminating CPPT results to professionals and the public.

Physician Attitudes

Dr. Ronald Goor presented the results of a recent NHLBI survey to ascertain physician attitudes, knowledge, and practice regarding CHD prevention. The first of a two-part survey was conducted before...
the release of the CPPT results and provided insights regarding physicians’ attitudes about cholesterol lowering. (The post-trial survey will be conducted late in 1985.) The number of physicians who believed that lowering blood cholesterol has a large effect on CHD risk was substantially lower than the number who believed that reducing blood pressure or stopping smoking would have a large effect. Diet changes were prescribed only for patients with blood cholesterol levels in the top 5%; drug therapy was limited to the top 1%. The cholesterol-lowering therapies that the physicians cited, and particularly the drugs being prescribed, were less than optimal. Based on the initial survey results, Dr. Goor recommended that professional education activities should include increasing physician awareness of the substantial benefits of lowering plasma cholesterol as well as the desirability of intervening at lower cholesterol levels and prescribing effective diet and drug therapies.

Public Awareness

Ms. Beth Schucker summarized the results from the NHLBI companion survey to assess public attitudes, knowledge, and behavior concerning blood cholesterol and heart disease. This survey, also conducted before the announcement of the CPPT study results, showed that the number of people who were informed about the relationship of blood cholesterol and heart disease was less than those who were informed about smoking and high blood pressure. A substantial proportion did not understand how blood cholesterol can be controlled. The majority of people were not concerned about their cholesterol level and very few people had made dietary changes to lower cholesterol. This survey confirmed the need for a public education program and will serve as a baseline for developing these programs and tracking changes.

Summary

In summarizing the issues and implications, Dr. Robert Levy stated that the question has now been changed from whether to treat hypercholesterolemia to how to treat hypercholesterolemia, at what level, by whom, and in whom. He asserted that it was unlikely that a study similar to the CPPT would be forthcoming in the near future, at least not until newer noninvasive diagnostic technology becomes available. Consequently, it is imperative that the implications of the CPPT findings be fully explored in the light of other available information. He urged that the NHLBI, American Heart Association, and others mount a public education program concerning blood cholesterol and heart disease similar to the hypertension education program that has been conducted so successfully during the past several years; he also noted that this cooperative venture was based on consensus building and was relatively inexpensive. Dr. Levy concluded that recent advances permitted the cardiovascular community to offer patients more than warnings about heart disease; now we can also tell them how they should be treated.

Research Recommendations

CPPT Data Analyses and Follow-Up

Dr. Lewis Kuller focused on analyses that need to be done in the CPPT data set and on priorities for a long-term follow-up. With respect to the CPPT results, he noted that the predictive power of the GXT was impressive, whereas the association between adherence to the drug regimen and cholesterol change was disappointing and not as good as expected. Both issues need more investigation. He felt that determinants of total cholesterol, LDL-C, and HDL-C need more investigation in terms of biological, individual, and clinic variables. If these could be defined, then the practicing physician might be better able to anticipate a response given a known set of circumstances. An individualized analysis should extend to the role of other cardiovascular disease (CVD) risk factors and anti-risk factors, including family history and weight loss as predictors of cardiovascular disease endpoints in the CPPT cohort. Any analysis of CVD endpoints should begin with a separate analysis of each endpoint category. Regarding follow-up of the cohort, Dr. Kuller emphasized the importance of ascertaining gastrointestinal cancer.

Role of Cholestyramine

Dr. Robert Mahley pointed out that understanding of lipoprotein physiology has progressed greatly since the beginning of the CPPT in 1971, and that new questions can now be posed about the role of the bile acid binding resins, such as cholestyramine, in this process. The liver is the key organ controlling lipoprotein metabolism through the E and B/E receptors. In animal models, the B/E receptor is increased and the catabolism of B/E-containing lipoproteins is enhanced when bile acid binding resin is given. What is the effect of cholestyramine on this process in humans, specifically as related to the E and B/E receptors? Likewise, reverse cholesterol transport from cells has the liver as its terminus and involves HDL, LCAT, and cholesterol transfer protein. What is the effect of cholestyramine on this system? We already know that HDL-C increases modestly with this treatment.

Even less is known about the effect of cholestyramine on LDL heterogeneity, the conversion of VLDL to LDL, the “hepatic shunt” that diverts a fraction of IDL from conversion to LDL, the metabolism of post-prandial lipoproteins, and the distinction between the effects on lipoproteins containing apo-B-48 and those containing apo B-100.

Advantage should be taken of experiments of nature with respect to abnormal apoproteins as seen in
apo E, which can be analyzed using recombinant DNA technology. Animal models are also useful in compressing pathophysiological events in time. Finally, more work on the molecular genetics of site-specific atherosclerotic lesions is needed, along with improved methods of assessment in vivo.

Dietary Studies and Other Treatments

Dr. Virgil Brown discussed clinical studies needed to answer the question of where LDL comes from. Mathematical modelling can help distinguish subtle biochemical mechanisms in genetic diseases. Dietary investigations also are needed to answer the following questions: are omega-3 fatty acids beneficial, how do polyunsaturates work, are monounsaturates beneficial, and what are the effects of proteins and amino acids, complex carbohydrates and fatty acids? The AHA Phase II and III diets also need additional study. Dr. Brown indicated that better treatments for hypercholesterolemia are needed, since the currently available bile acid sequestrants are too expensive and other resins, as well as the promising HMG-CoA reductase inhibitors mevinolin or compactin, still need evaluation.

Toxicity

Dr. Henry Blackburn agreed that follow-up of the CPPT cohort for toxicity is needed, and he cited other scientific questions related to the causal and prevention role of lipoprotein fractions and subfractions. More studies of interventions in the midrange cholesterol levels are needed, but such studies will have to await more sensitive quantitative continuous measures of outcome. He also noted that risk characteristics in children, youth, and young adults, as well as the correlations between youth and adult values in individuals and populations, need to be defined.

Public Policy Decisions

Many workshop participants shared the view expressed by Dr. Richard Carleton that enough is now known to make public policy decisions even though some issues involving the generalizability of the CPPT results remain unresolved. He noted that by relying on a cholesterol level of 275 mg/dl as the standard treatment for hypercholesterolemia, still need evaluation.

Dr. Carleton proposed a randomized evaluation of diet in the CPPT placebo follow-up cohort as an important step to answer prospectively the question of whether diet achieves the same benefit as drug therapy.

Discussion of the implications of the CPPT and related research findings continued at the Consensus Development Conference, "Lowering Blood Cholesterol to Prevent Heart Disease," sponsored by the National Institutes of Health in December 1984.

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Workshop Speakers and Session Chairmen

NHLBI workshop on the Lipid Research Clinics Coronary Primary Prevention Trial.

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