THE USE OF MULTIPLE MEASURES, REPEATED FEEDBACK, GOAL SETTING, SHAPING, AND NUTRITION EDUCATION TO LOWER SERUM CHOLESTEROL LEVELS IN MALES

by

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Abstract

Although the association between elevated serum cholesterol levels and cardiovascular risk has been known for many years, few studies, with freely living individuals have used a full compliment of intervention strategies to attempt to alter practices associated with elevated serum cholesterol. Two studies, (Study 1, n=4; Study 2, n=8) with 12 middle age men (mean age = 47.3 years) and with elevated serum cholesterol ($\bar{x}$= 238.7 mg/dl) are presented that use multiple measures of serum cholesterol (using the Boehringer Mannheim Reflotron and finger stick technique). The main intervention strategies included a combination of procedures using education, frequent serum cholesterol feedback (two to three times per week), and specific dietary feedback (one to two times per week). A less intensive intervention that is similar to recent studies in the literature was also implemented and assessed for half the subjects in Study 2. The results of the two studies indicated that within approximately 14 weeks, the combination of enhanced procedures reduced serum cholesterol by about 14%, or about double that found in prior studies. The less intensive intervention showed reductions of 9.1%. The use of multiple measures also allowed for the study of intraindividual variability. Issues pertaining to maintenance of effect, cost-effectiveness, and generalizability are also discussed.

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INTRODUCTION

The association between coronary heart disease and high levels of serum cholesterol has been evident for a long time. Recent evidence points to serum cholesterol as a major contributing factor to coronary heart disease. (NIH, 1985). The American Heart Association has urged that individuals adopt a diet that reduces total dietary fat intake from the current level of about 40% of total calories to 30% of total calories, that reduces saturated fat intake to less than 10% of total calories, and that reduces daily cholesterol intake to 250 to 300 mg or less.

Literature Review.

While the dietary guidelines developed by the American Heart Association and others seem straightfoward, persuading individuals to engage in the necessary behaviors to achieve the goals of these guidelines is not a simple process. A number of different studies have attempted to lower individual serum cholesterol levels in community, worksite, and hospital settings. They have employed cholesterol screening, nutrition education, information, dietary counseling, and dietary control.

Some of the earliest most basic research in the area of cholesterol was performed on metabolic wards. Keys, Anderson, and Grande (1965) conducted a medical ward study of 22 schizophrenic males. The authors reported that a change from a 250 mg/1000 Cal. to a cholesterol-free diet reduced average serum cholesterol by 24 mg/dl., or roughly a10% reduction. Some of the most impressive reductions to date occured in a study by Connor, Hodges, and Bleiler (1961). They examined the serum cholesterol of six men cycled on controlled high-cholesterol and cholesterol-free diets

that were identical in calories, fat, protein, carbohydrate, minerals, and vitamins. During the first period, a three week cholesterol-free diet, subjects lost a mean of 58 mg of serum cholesterol with a range of 34 to 94 mg losses. Because subject's average cholesterol was 249 mg, this represents a 23% loss.

In general, metabolic ward studies have made it clear that very significant clinical reductions in serum cholesterol are attainable with a dietary intervention. Unfortunately, these kinds of changes have not been replicated outside the controlled setting of a metabolic ward. A number of community and worksite studies have targeted cholesterol and a wide range of high risk behaviors. A representative summary of these studies can be found in Table 1.

INSERT TABLE 1 HERE

Results from worksite and community studies indicate that mean serum cholesterol reductions of 3-12% have been attained. On average, these results have yielded reductions of only 5%, which is roughly equivalent to a 10% reduction in CHD risk.

Why have these studies failed to produce larger and more impressive reductions in serum cholesterol? How can we improve upon the existing body of research?

First, it should be noted that none of these studies have used a full range and/or combination of previously effective intervention techniques to try to lower individual's cholesterol levels (Winett, King & Altman, 1989). These effective techniques for dietary change have included a specific behavioral prescription and protocol, specific goals, and specific feedback (see chapter eight, Winett, King & Altman, 1989). The authors advocate a systematic and step-wise application of specific dietary goals. Individuals

receive specific feedback about their diets, and what what aspects of their diets need to be changed. In addition, salutory dietary behaviors are modelled for individuals. Individuals make successive changes in their diets until they reach desired goals.

Repeated Feedback

Specifically, no study has used repeated feedback as a means of encouraging behavioral dietary change in their participants. Repeated feedback consists of both repeated measures of serum cholesterol and carefully tracking individual's dietary changes. Previous cholesterol studies have typically implemented an intervention and given feedback in the form of a second cholesterol measure some 6 months later. The shortest duration for feedback in these studies was 2 months (Lefebvre et al.,1986). For 2 months, subjects in this study did not know if the dietary changes that they were making were having any effect on their serum cholesterol.

Bandura (1986) suggests the importance of repeated feedback with specific hard but reachable goals as an important behavior change strategy. Positive feedback can motivate individuals to maintain behaviors, whereas negative feedback can work to motivate individuals to discontinue certain behaviors, or to seek alternate behaviors. Feedback is most effective if collected and visually presented on a continuous basis, and if it is placed in the context of appropriate population norms and goals (Elder, 1985). For example, in a weight loss program individuals may graph their own weights; the graph should include their goal or ideal weight based on their height, frame size, and percent overweight.

Further, in an effective feedback approach to behavior change the following elements are important (see Bandura, 1986):

1. The target behaviors that need to be changed are clear (e.g., specific dietary practices).
2. There is a clear direct or indirect indicant of behavior change (e.g., total serum cholesterol).
3. Individuals are aware of the relationship between target behavior's change and the indicant of change (e.g., dietary change and total serum cholesterol change).
4. There are specific hard but reachable goals that relate to target behaviors and indicants of change.
5. An intervention plan is devised so that individuals favorably perceive the relationship between changes in target behaviors, meeting of goals, and changes in indicants of change (i.e., individuals are reinforced for behavior change).
6. Successive approximation and shaping are used (see below) to both increase the probability of reaching specific subgoals (e.g., a 5% reduction in total serum cholesterol) and receiving reinforcement (e.g., positive reinforcement in the form of a lowered cholesterol reading).

However, compared to feedback programs in such areas as obesity and hypertension, there have been barriers to implementing an effective feedback approach in the area of cholesterol. First, there is the necessity for venipuncture by phlebotomists or other trained health professionals to obtain a blood sample. Second, there is the expense of tests that require a medical laboratory and trained personnel. Third, there is often a delay time between when the individual intiates action (goes to get measured) and receives feedback (laboratory results). With the advent of portable light

spectometry machines, such as the Reflotron, that give a measure of total cholesterol in three minutes, these past problems are now largely resolved.

Intraindividual Variability and the Need for Repeated Measures.

A second problem with these past studies is that they failed to employ repeated measures of the dependent variable which could better assess the direct effect that the treatment intervention has on blood cholesterol. Other than the cited metabolic ward studies, it is not clear what is happening to an individual's blood cholesterol during an intervention. Lefebvre, in a personal communication to the author, stated that some individuals may show variations as much as 5-10 points per day in their blood cholesterol.

Other research has shown that intraindividual variability can be much higher. A study by Buzina, Ferber & Keys (1964) reported that intraindividual variability accounted for 77.9% of the total sample variance in the percentage of total calories from fat. That is, dietary and other extraindividual factors apparently accounted for 22.1% of the total sample variance. In addition, this variation can occur within a very short period of time. A study by Hahn et al. (1988) found a variation of 30 points in hourly blood samples taken over a 24 hour period.

Another study by the Hegsted and Nicolosi (1987) showed that there was a mean coefficient of variation of 5% to10%, even in metabolic ward studies where diet was controlled. They went on to conclude that even if there is a mean intraindividual SD of only 5% of the mean value, the reading from a single serum sample may fall within 2 SD above or below the true mean. For example, an individual with a mean serum cholesterol level of 220 mg/dl is likely to fall between a range of 200 mg/dl (no risk) to 240

mg/dl (high risk). The researchers also pointed out that in any large clinical trial that used one or a few blood samples obtained before and after attempts at lowering serum cholesterol levels, that very large numbers of individuals can be expected to improve or deteriorate from chance alone.

Further, large intraindividual variability means that some individuals may be mislabelled in screening studies using a few measurements. Liu et al., (1978) used statistical models to examine intraindividual variability and concluded that studies which examine the relationship between fatty acids and serum cholesterol should employ repeated serum sampling and numerous dietary records. Jacobs et al., (1983) cautioned that drawing conclusions about an individual's coronary risk from a few serum samples can be misleading. As a summary statement, Keys (1988, p.1161) warns that the: "Similar lack of attention to intraindividual variability is frequent in surveys and epidemiological studies and leads to error, controversy, and a waste of time and money."

In addition to natural fluctuations of serum cholesterol levels related to an individual's constitutional or nutritional status, research has shown that plasma lipids can be noticeably influenced by short-term emotional arousal. Dimsdale and Herd (1982) reviewed sixty studies that examined the relationship between emotional arousal and plasma lipid levels. They reported that free fatty acid levels almost uniformly increased in the context of a wide variety of stressful conditions. Most studies also found that cholesterol increased from 8% to 65% above baseline under stressful conditions. Individual subject's responsiveness to stressful events also tended to vary considerably. The authors suggest a minimum of four measures of lipids to form an impression of the patient's baseline condition.

In general, both the intraindividual variability and variability associated with emotional arousal suggest the need for multiple measures of serum cholesterol. This is particularly important at the baseline phase of a study, to draw an accurate estimate of an individual's true risk, and after the intervention phase. A single 6 month, post-intervention measure may grossly overestimate or underestimate the changes of an individual's serum cholesterol level.

It appears important to assess through multiple measurement the actual intraindividual variability in total serum cholesterol shown by freely living individuals. However, from the perspective of risk reduction, it is important to assess (also through multiple measurement) if an intervention can demonstrate a decrease in serum cholesterol that more convincingly can not be attributed to intraindividual variability.

Successive Approximation and Shaping.

A third important shortcoming of many past studies is the abscence of successive approximation and shaping strategies (Kazdin, 1984). One of the basic tenents of behavioral self-management is that habit change can best be shaped in small, workable steps in which initial changes in behavior are more likely to occur and lead to positive reinforcement. This, in turn, can lead to enhancement of an individual's self-efficacy which will increase the probability of a successful next step. Shaping involves the reinforcement of successive approximations of a target behavior until a criterion behavior is attained. For example, "nicotine fading" is a smoking cessation procedure that gradually reduces nicotine dependence. In the area of diet modification, the gradual three-phase approach employed in the Connors' Alternative Diet lends itself to such a process of habit change.

Brownell (1986) suggests that making changes in an individual's dietary behavior can be best achieved by shaping eating patterns gradually towards appropriate goals. He warns that encouraging individuals to make major shifts in the types, and quantities of foods that they eat will be unsuccessful. Brownell also noted that gradual change encourages realistic goal setting in modifying behavior, maximizes opportunities for early success experiences, prevents information overload and confusion, and engenders confidence in the individual for making more difficult changes in the future.

Dietary Modifications.

Fifthly, apparently, only one study, outside of metabolic ward studies, assessed what kinds of dietary changes individuals made as a function of the intervention. Reeves et al., (1983) attempted to do so by measuring subjects reported intake of dietary cholesterol, and found significant reductions in cholesterol over the course of 6 months. It is important to assess the dietary changes in individuals for two reasons. First, if individuals are not successful in the program, this information may suggest a reason for their failure. Second, because of intraindividual variability and variability associated with emotional arousal, changes in serum cholesterol do not uniformly correlate with dietary change. Dietary change must be measured independently from serum cholesterol levels.

Summary.

Briefly, while the history of cholesterol reducing studies has shown some promise, a number of strategies to help individuals lower their levels have not been employed. Short of restricting people to a metabolic ward, we

do not know the best way to help individuals to significantly reduce their serum cholesterol. Further, multiple measurement of cholesterol appears to be necessary.

This paper presents two studies that address these issues. In Study 1, an intervention consisting of the elements of repeated measures, repeated feedback, shaping, dietary feedback, nutrition education and goal setting was tested with four male subjects with elevated serum cholesterol. In Study 2, a similar approach was further tested against a more minimal intervention with eight male subjects (n=4 in each condition) with elevated serum cholesterol. The intervention in Study 2 was different from that in Study 1 in that subjects had more contact with the experimenter, were measured one day more per week, and completed food diaries one day more per week.

It was hypothesized that the combination of these elements of repeated measures, repeated feedback, shaping, dietary feedback, nutrition education and goal setting would yield larger reductions in serum cholesterol than have been demonstrated thus far in the literature. In addition, because of the use of repeated measures of serum cholesterol, it was hypothesized that this approach could better demonstrate the direct effect of a dietary intervention while also assessing patterns of intraindividual variation.

STUDY 1

METHOD

Subjects.

Subjects were males between the ages of 35 and 60, employed at Virginia Polytechnic and State University (V.P.I. & S.U.), and working in Derring Hall. Males in this age range have been shown to be at highest risk for elevated serum cholesterol, and are also, for purposes of comparison, the population at which the majority of research has been directed. Individuals were excluded from this study if:

1) They were taking lipid altering medications.

2) They were taking any other prescribed medications. This criterion is included because of the possibility of an unknown effect of any given medication on cholesterol.

3) They were on any specialized or medically prescribed diet.

4) Their initial serum cholesterol levels were less than 220 mg/dl or greater than 260 mg/dl. This range of serum cholesterol scores places them at moderate to high risk. Individuals who had cholesterol levels greater than 260 mg/dl were disqualified and encouraged to contact a physician.

5) They had a personal history of heart disease, diabetes, or cancer.

Subjects were recruited from Derring Hall by the experimenter. The rationale for recruiting subjects from Derring Hall was that subjects needed to have their cholesterol levels tested twice per week, and subject's proximity to the Reflotron machine, which was located in Derring Hall, would facilitate this procedure.

Initial contact was made by the experimenter who knocked on office doors in Derring Hall. Potential subjects were told that the experimenter was conducting a study about cholesterol and was recruiting individuals for the study. They were told that a free screening of their cholesterol level was being offered the following day, and asked if they would like to be screened. If they agreed to be screened, they were scheduled for a time.

The experimenter knocked on 52 office doors over a period of three days. Of these, he made personal contact in 43 cases; in 4 cases individuals said that they were not interested, and in 5 cases individuals were disqualified because of age or sex criteria. In total, 34 men were screened, and of these 6 had cholesterol levels in the range of 220-260mg/dl. Two men refused to participate due to time constraints, and the remaining 4 agreed to be in the study. The mean age of all subjects was 49.25 years. The mean age of subjects 1 and 2 was 49 years; the mean age of subjects 3 and 4 was 49.5 years. The range of ages for subjects was 45 years to 53 years. Three subjects were professors at V.P.I. & S.U. and one subject was a Computer Systems Engineer. Subjects from Study 1 were recruited at a different time and from a different pool than subjects from Study 2.

Materials.

Total cholesterol was measured by a light spectometry machine called the Reflotron. This machine offers reliable measurement of total plasma cholesterol in a procedure that takes approximately 5 minutes. Research by the manufacturer of the Reflotron, Boehringer-Mannheim, indicates that the Reflotron's accuracy is $\pm$ 5 % and its precision is $\pm$ 2%. Independent studies assessing the reliability of the Reflotron have shown

1% to 4% fluctuations in the measure. (Personal communication from D. Hyman, M.D. to R. Winett, Ph.D., March, 1988).

In order to assure the reliability of the Reflotron, reliability checks with a precalculated check strip were run weekly. In addition, a reliability check with lymphasized blood serum samples supplied by Boehringer-Mannheim were run every six weeks. Both of these tests showed the machine to be running accurately during the course of the study. The machine was also cleaned on a weekly basis during the course of the study, as recommended by the manufacturers.

Educational materials were derived from publications distributed by the American Dietetic Association, the American Heart Association, The National Heart, Lung, and Blood Institute, and Dr. Kenneth Cooper's (1988) book, Controlling Cholesterol (New York, Bantam Books). Specific dietary suggestions for planning meals were derived primarily from Dr. Cooper's book, and to a lesser extent from these other materials. In addition, the experimenter had experience working as a research assistant and running weight loss groups for the Obesity Research Group of the University of Pennsylvania Medical School from March, 1984 to August, 1985.

Design and Procedures.

The purpose of Study 1 was to assess the feasibility of using a repeated blood sampling approach in an intervention, and to see if serum cholesterol reductions larger than those obtained in prior non-metabolic ward studies could be achieved using this approach.

A three-phase intervention was employed in the design for this study. Phase I was the baseline phase, Phase II the intervention phase, and Phase III the maintenance phase. Subjects were assigned to pairs at random, and

pairs received the phases of the intervention in staggered periods. In the first phase of this study, Subjects1 and 2 completed the baseline phase and then received the second phase (the intervention) while Subjects 3 and 4 continued in the baseline phase, thus serving as controls for comparison purposes. In the third phase of this study, Subjects 1 and 2 took part in the maintenance phase while Subjects 3 and 4 began the intervention phase.

Phase I. During the baseline phase subjects were encouraged to continue eating as they normally did, and were measured twice per week. Based on recommendations cited in the study by Keys (1988), and on a telephone conversation that the experimenter had with Dr. David Hyman, it was decided that 6 to 8 measures (3 to 4 weeks) constituted a sufficiently accurate baseline of subject's total serum cholesterol level. Subjects were also required to donate a blood sample for a more complete fasting lipid analysis in the second week of this phase. During this phase, subjects saw each of their cholesterol readings, but no specific comments or advice were given at this time.

Phase II. The intervention phase was multifaceted. It consisted of the following elements:

1) Total cholesterol measures. Subject's total serum cholesterol was measured twice per week on the same days each week. Subject's reading were explained to them in terms of risk category and percentile score based upon their age. In addition to each week seeing the readout of their total cholesterol level, subjects were visually presented with a graph of their cholesterol values from beginning of the study. On average subjects kept

89% (40 of 45) of their scheduled appointments with the experimenter, and ranged between and 85% (38 of45) to 94% (42 of45).

2) Lipid profiles. Fasting lipid profiles obtained in the second week of the study were presented to all subjects. HDL and LDL levels were explained in terms of their modifying effect on CHD risk.

3) Food diaries and feedback. Subjects were required to keep a written diary of all the foods that they consumed on a given day for 3 days per week. Days of the week were randomly assigned. Subjects were educated by the experimenter about how to measure and estimate food quantities. For example, the experimenter brought specific foods (e.g. chicken and broccoli) and showed subjects how many ounces these foods weighed. Subjects were encouraged to weigh foods at home that they were unsure about. In addition, because of the versatility of the Nutritionist 3 software, subjects could and did report their consumed items in a variety of units (ounces, cups, items, tablespoons, grams, etc.). The purpose of food diaries was: 1) to assess the ways in which subjects would need to make qualitative and quantitative dietary changes 2) to chart the progress that subjects in the intervention phase were making at changing their diets 3) to make subjects more conscious of the foods that they eat, and 4) to use dietary records as a source of feedback.

Feedback was offered in two ways. First, the experimenter examined the diaries and praised subjects for food choices that were lower in saturated fat, total fat, and dietary cholesterol. He also suggested alternative foods for foods that were high in saturated fat, total fat, and dietary cholesterol. Second, food diaries for each day were analyzed by the

the experimenter using Nutritionist 3 software. The values that have the most significant effect on blood cholesterol are: 1) dietary cholesterol (mg), 2) saturated fat (g), 3) total fat (g), 4) percent calories from fat and 5) percent calories from carbohydrate. These values were presented and explained to subjects in graphical form once per week in 15 minute sessions.

4) Goal setting. In the same 15 minute session each week, the experimenter set goals with subjects to help them change their dietary habits. Specifically, the experimenter would encourage subjects to make successive qualitative and quantitative changes in their intake of saturated fat, percent calories from fat, total fat, and dietary cholesterol. He would help subjects make successive approximations towards ultimate goals of 20% calories from fat, dietary cholesterol less than 200 mg/day, and saturated fat less than 20 g/day. For example, a steak lover who has consumed 12 ounces of steak, a baked potato, and one stalk of broccoli was encouraged in the future to consume 8 ounces of steak, 2 potatoes, and 2 stalks of broccoli. The experimenter helped subjects plan in choosing foods that are low in cholesterol and saturated fats, and make suggestions about how they can prepare these foods so as to minimize cholesterol intake. Dietary prescriptions were tailored to each subject's food preferences.

5) Education. Subjects were educated about dietary changes by the experimenter and with written materials. The experimenter would point out which foods in their food diaries were high in fat and dietary cholesterol, and would make suggestions about foods that were healthier alternatives. In addition, he would answer any questions that subjects had about

cholesterol and their diet. Written materials consisted of 1) a dietary guide of foods and their cholesterol, fat and saturated fat content 2) a list of two weeks of meals that were low in fat and cholesterol, and 3) a list of a number of recipes for meals that were low in fat and cholesterol.

Phase III. The maintenance phase was the final phase of the study. Subjects were measured once per week, and kept food diaries on two days per week. They continued to receive verbal feedback about their food diaries and their total serum cholesterol values were presented to them graphically.

Dependent Measures.

The major dependent measure was total serum cholesterol which was assessed by the Reflotron. The procedure uses the fingerstick method and takes approximately 4 minutes. Blood samples were taken from fingers on the same hand. Unlike measures of trigylcerides which require a 12 hour fasting blood sample, it has been demonstrated that nonfasting and fasting measurements of total blood cholesterol are equally reliable (U.S. Department of Health and Human Services, 1985).

Measures of dietary cholesterol, total fat, saturated fat, and percent calories from fat were derived from subject's daily food diaries. These measures were obtained using Nutritionist 3 software.

Subjects were measured for a fasting lipid profile on three occasions. The blood sample was obtained by a registered nurse in the Cardiac Rehabilitation Program at V.P.I. & S.U. This sample was analyzed by Roche Laboratories.

All subjects were measured during the second week of Phase I of the study. In addition, Subjects 1 and 2 were measured at the end of Phase II and at the end of Phase III. Subjects 3 and 4 were measured concurrently, which, for them, marked the end of their Phase I and the end of their Phase II. This lipid profile yields measures of total serum cholesterol, HDL, LDL, VLDL, and triglycerides. The measures in the lipid profile are included in this study because they yield ratios of total cholesterol to HDL and LDL. Research has suggested that cardiovascular risk associated with a high total cholesterol level is lessened if an individual has a high HDL count (Cooper, 1988). Cooper (1988) suggests that a total cholesterol/ HDL ratio of less than 4.6/1.0 is important in reducing coronary heart disease.

RESULTS STUDY 1

Figure 1 shows the total serum cholesterol levels for each subject on each measurement day.

INSERT FIGURE 1 HERE

INSERT TABLE 2 HERE

The measures during Phase II are averaged in twenty day segments for purposes of comparison. The mean serum cholesterol is shown at the top of each segment. The mean and standard deviation values for the different phases (I-III) and periods (A-G) within phases are shown in Table 2. The two week period immediately after the beginning of Phase II represents a metabolic "lag" time. This is the average time it takes an individual to reveal changes in his serum cholesterol due to dietary change (Personal communication, Janet Walberg, Ph.D., January, 1989). Note that Period C (Day 98 to Day 118) represented Christmas break, and subjects were

unavailable for measurement at this time. Note also that Subject 3 took an early Christmas vacation and was unavailable for measurement from Days 82 to 108, and was subsequently measured three times in Period C. Phase II for Subjects 3 and 4 was implemented on Day 142 and because of the subsequent lag time, Periods F' and G' are different from F and G.

Intraindividual Variability.

As shown in Figure 1 and Table 2, the intraindividual variability of cholesterol scores for Subjects 1 and 2, as measured by their standard deviation scores, did not appear to increase or decrease during the intervention phase of the study. Both subjects showed standard deviations of ± 9.7 mg/dl during Phase I, and standard deviations of ±11.9 and ±9.4 respectively during the intervention phase. Relative to the other phases, Subject 1 showed his smallest variability in Phase III (±7.9 mg/dl), whereas Subject 2 showed his greatest variability in this phase (±32.0 mg/dl). The variability of Subject 2 will be discussed in more detail later in the paper.

Concurrent measures of serum cholesterol for Subjects 3 and 4 during Days 1-42 revealed variability of ±16.7 mg/dl and ±8.2 mg/dl, respectively. From Days 43-142 Subject 3 continued to show more variability than the other subjects (±20.9 mg/dl). In general, both Subjects 3 and 4 showed greater average variability from Days 43 to 142 (±17.4 mg/dl) than did Subjects 1 and 2 (±10.7 mg/dl). From Days 143-200, Subjects 3 and 4 varied ±17.3 mg/dl and ±8.3 mg/dl respectively. Note also that depending on when Subject 3 was measured during the first three weeks of his baseline phase, his cholesterol level was as high as 246 mg/dl (High risk) or as low as 192 mg/dl (excellent protection).

Note in Study 1 that the comparison between baseline phase and subsequent periods of the study is difficult because the baseline phase was twice as long as any of the periods. In addition, comparison is difficult because there are a different number of measurements taking during these periods. Comparison of macronutrient data between baseline and intervention phases (see Table 3) indicates that during the intervention phase, Subjects 1-4 ate more consistently. Standard deviations were lower during the intervention phase in most comparisons (Subject 1, 30 of 30 comparisons; Subject 2, 25 of 30 comparisons; Subject 3, 29 of 30 comparisons; Subject 4, 26 of 30 comparisons).

Serum Cholesterol Change and Dietary Change.

Days 1-142.

Subject 1 lost an average of 31.2 mg/dl comparing his baseline period to the final period (period D) of Phase II. This is a 12.5% reduction and an estimated 25% reduction in his CHD risk. Subject 1 showed a slight increasing trend in his baseline phase. Thereafter, his serum cholesterol decreased fairly uniformly and reached its lowest levels in the last stage (period D) of Phase II. Subject 2 also showed a significant loss comparing these same periods; this value was 10.5%, or a 21% reduction in CHD risk.

Comparing these periods for the minimum intervention subjects that served as controls indicates that Subject 3 decreased his serum cholesterol level by 9.0%, while Subject 4 increased his level by 6.3%. Hence, Subjects 1 and 2 had a mean serum cholesterol decrease of 11.5%, while Subjects 3 and 4 had a mean decrease of only 1.4%. Subjects 3 and 4 also had lower initial levels of serum cholesterol between Days 1-42 than did Subjects 1 and 2.

During Subject 3 and 4's Phase I (Days 1-142), their mean serum cholesterol levels showed a slight increasing trend until about Day 100. This increasing trend appeared to be stronger for Subject 4. At this point, the serum cholesterol levels of Subjects 3 and 4 began to decrease. The mean change in serum cholesterol from periods C to D ($\bar{x}$ = 35.4 mg/dl for Subject 3 and 18.8 mg/dl for Subject 4) was the largest reduction for both subjects across any two periods.

One hypothesis to explain this drop in serum cholesterol for Subjects 3 and 4 was that their serum cholesterol levels prior to this period were at their highest, and subjects began to make changes on their own because they were concerned by these high values. This hypothesis is not clearly supported by their food intake data, as shown in Figures 4 and 5 and Table 3. Subjects 3 and 4 did not appear to be making uniform changes in their diet in periods C and D.

INSERT FIGURES 2, 3, 4 & 5 HERE

INSERT TABLE 3 HERE

Figures 2, 3, 4, and 5 each show six different macronutrient values derived from subject's self-report food diaries. Mean values are shown for each phase at the top of the graph. The mean and standard deviation values for all macronutrients are shown in Table 3. As shown in Figures 2 and 3 and Table 3, Subjects 1 and 2 showed immediate reductions in their intake of dietary cholesterol, saturated fat, percentage fat, and total fat at the beginning of Phase II, the intervention. The immediate reduction can be seen by comparing the baseline period to the "lag" period (Day 42 to 58). Subjects 1 and 2 decreased their consumption of all these macronutrients. Subjects 3 and 4 showed reductions in some of these macronutrients, but

not all of them. In addition, as can be seen in Figures 4 and 5, their levels of intake of these macronutrients are higher.

Comparing the mean caloric values between Phase I and the lag period of Phase II shows that Subject 1 decreased his intake from 2734 kcal/day to 1702 kcal/day and Subject 2 decreased his intake from 2081 kcal/day to 1695 kcal/day. The same comparison for Subject 4 shows little change (2407 kcal/day to 2217 kcal/day) and a decrease for Subject 3 (2070 kcal/day to 1392 kcal/day).

In addition to the immediate effects of the intervention, the food intake values for Subjects 1 and 2 reflect fairly consistent reductions in dietary cholesterol, saturated fat, percentage fat, and total fat during Phase II (mean values averaged across periods A-D). This is apparent by looking at Figures 2 and 3 across periods A-D. Examining these same periods for Subjects 3 and 4 in Figures 4 and 5 reveals that their intake of dietary cholesterol, saturated fat, percentage fat, and total fat was less consistent, was closer to their baseline values, and was higher than the intake of Subjects 1 and 2.

For Subject 1, his most impressive dietary intake reductions during Phase II were in saturated fat intake (from 22.8 $\pm$ 14.9 g during Phase I to 10.0 $\pm$ 4.9 g during Phase II) and total fat intake (from 100.5 $\pm$ 39.2 g to 51.3 $\pm$ 18.8 g during Phase II). Levels of dietary cholesterol and percentage fat, and total calories were also decreased during Phase II. In addition, mean percentage carbohydrate was increased by 10% from Phase I to Phase II.

As shown in Figure 3 and Table 3, Subject 2 showed his greatest reductions from Phase I to Phase II in dietary cholesterol (from 326.0 $\pm$ 255.6 mg to 121.7 $\pm$ 66.6 mg), saturated fat (from 25.2 $\pm$ 6.6 g to 11.8 $\pm$ 5.2

g), and total fat (from 95.6 ± 21.8 g to 44.6 ± 17.8 g). He also showed reductions in percentage fat and total calories, and a 10% increase in percentage carbohydrate.

Subjects 3 decreased his percentage carbohydrate (-6.4%) and Subject 4 only slightly increased his level (3.6%) across this same period.

Days 143-200.

When the intervention was implemented (Day 143) for the second set of subjects (see Figure 1), Subject 3 made an average reduction in serum cholesterol from Phase I to the last period of Phase II, of 8.9% (17.8% reduction in CHD risk). Subject 4 lost 8.3% (16.6% reduction in CHD risk). Interestingly, Subject 3 showed his greatest reduction in serum cholesterol in the 20 day segment just after the metabolic lag time (Days 158-178). In this period, his serum cholesterol level was at its lowest and was an 18.4% reduction. During this same period, Subject 4's serum cholesterol showed only a 5.8% reduction.

Dietary data from this period as shown in Figures 4 and 5 and Table 3 show that the intervention at Day 143 had an immediate effect at reducing Subject 3 and 4's dietary intake. Specifically, comparing dietary intake for the period prior to the intervention (period D) to the period immediately following the intervention (period E) reveals that Subject 3 reduced his intake of dietary cholesterol (from 133.2 ± 195.7 mg to 78.3 ± 30.6 mg), saturated fat (from 9.1 ± 5.9 g to 8.1 ± 4.5 g) and total fat (from 28.0 ± 20.0 g to 23.0 ± 16.5 g). The reduction was minimal for percentage fat (from 22.1 ± 12.3 % to 20.0 + 9.5%). Subject 4 reduced his dietary cholesterol (from 258.9 ± 19.8 mg to 161.5 ± 64.0 mg), saturated fat (from 25.8 ± 11.4 g to 10.4 ± 4.2 g), and total fat (from 73.9 ± 25.2 g to 55.6 ± 16.7 g). He also showed a reduction in percentage fat (from 37.2 ± 6.3% to 27.3 ± 7.1%).

During this same time period, Subjects 1 and 2 were taking part in a short-term maintenance phase. Comparing their serum cholesterol levels from Period D of Phase II to the last period (Period G) of Phase III reveals that Subject 1 increased his level by 5.5% and Subject 2 increased his level by 8.0%.

Dietary values for these periods of comparison reveal that Subjects 1 and 2 began to make changes in their intake of fats. Specifically, Subject 1 showed an increase in dietary cholesterol (from 127.6 ± 65.3 mg to 193.7 ± 92.8 mg), saturated fat (from 10.0 ± 4.9 g to 16.5 ± 3.1 g) and percentage fat (from 22.0 ± 4.5% to 26.5 ± 3.5%). The dietary pattern during this period for Subject 2 are less consistent. Subject 2 showed decreases in dietary cholesterol (from 121.7 ± 66.6 mg to 62.0 ± 33.3 mg) and saturated fat (from 11.8 ± 5.2 g to 9.9 ± 4.6 g), but increases in percentage fat (from 30.3 ± 5.5 % to 35.0 ± 0.0%) and total fat (from 44.6 ± 17.8 g to 51.1 ± 12.6 g).

Variability and Emotional Arousal.

Related to the issue of intraindivual variability is one of variability brought about by emotional arousal and stress. As shown in Figure1, Subject 2 showed wide variability in his readings from Days 143 to 162. His serum cholesterol level increased to a level of 263 mg/dl and plummeted to 166 mg/dl in a period of one week. Machine error is a very unlikely cause for these readings, because the machine was reading reliably for the other subjects. The subject reported that he had experienced a death in his family, had driven 30 hours in a period of three days to get to the funeral, and had averaged 3.5 hours of sleep per night during this same period. In addition, as shown in Figure 3, the fact that the subject's reported diet prior to this period was unremarkable, makes it unlikely that

these results were due to dietary changes. However, Subject 2's serum cholesterol again peaked to a level of 261 mg/dl on Day 174. The subject reported that he had been experiencing problems at work, and had been having difficulty sleeping at night. He reported that he had not made any significant changes in his diet, and dietary data, as shown in Figure 3, confirm this report. It is unclear why Subject 2 continued to show elevated serum cholesterol levels.

Lipid Analysis.

In addition to multiple measures of serum cholesterol, a more extensive lipid analysis was completed in the baseline phase and at the end of the intervention phase for each subject.

INSERT TABLE 4 HERE

As shown in Table 4, all of the subjects in Study 1 had Total Cholesterol/HDL levels greater than the 4.6/1.0 level recommended by Cooper (1988) prior to the intervention. Subject 2 reduced his Total cholesterol to HDL cholesterol ratio from 5.5 to 4.6, whereas Subject 1 showed little change in this ratio (5.8 to 5.7). It is not clear why Subject 1 did not show a greater change in his ratio. Subjects 3 and 4 were measured at Day 143 to see if they had made any changes in their lipid levels over the course of their Phase I. Results show that both subjects had increased their Total cholesterol/HDL ratio. However, at the end of their Phase II (Day 200), Subjects 3 and 4 showed reductions in their ratios (from 6.3 to 4.3 for Subject 3, and from 5.7 to 4.8 for Subject 4). To summarize, one subject showed little change in his ratio, two dropped their ratio by nearly one point, and one dropped his ratio by two points.

HDL levels for subjects 1 and 2 both decreased at the end of their intervention period. These levels increased and stayed the same for Subjects 3 and 4, respectively. The net change for all four subjects was a 1.2% increase in HDL.

LDL levels decreased for all subjects, and the mean decrease was 12.8%. Trigylceride levels had also decreased for every subject at the end of their intervention. The mean decrease was 45.1%.

Other Dietary Data.

Subjects reduced their caloric intake by a mean of 29% from Phase I to Phase 2 (see Figures 2, 3, 4, and 5). While it was not a goal of this study to make quantitative changes in subject's caloric intake but to qualitatively change their diet, it was, however, realized that such qualitative changes could indirectly result in caloric reduction. Specifically, if subjects are consuming fewer foods that are high in fat and at the same time are replacing them with foods that are low in fat, they will consume fewer total calories.

While one reason that subjects were consuming fewer total calories relates to the reduction in fat from their diet, visual inspection of food diaries revealed that they were also consuming fewer grams of food per day during Phase II. This reduction in quantity of food may have helped subjects reduce their serum cholesterol levels. Specifically, if subjects are eating less total food, they are probably eating less total dietary cholesterol and less total fat. This result also suggests that subjects may have believed it was necessary to reduce their food intake in order to reduce their serum cholesterol level.

STUDY 2.

METHOD STUDY 2

Purpose.

The purpose of Study 2 was two-fold. First, having shown that reductions in serum cholesterol were attainable using the procedures of Study 1, a quasi-replication was attempted. The procedures in Study 2 were similar to Study 1, but with one minor modification. The modification consisted of measuring subject's serum cholesterol three times per week instead of two times per week. This modification was included with the intention of increasing the rate at which subjects lowered their serum cholesterol. It was thought that increasing frequency of contacts and feedback would enhance serum cholesterol reduction. This modified set of procedures was the maximum intervention.

The second objective of Study 2 was to compare two different kinds of interventions. The maximum intervention was compared to a minimum intervention. The purpose of this comparison was to see if subjects who received the same initial baseline period of measurement, the same educational materials and initial dietary counseling (minimum intervention) could reduce their levels of serum cholesterol to an appreciable degree (i.e. show some risk reduction). The minimum intervention was modeled after the interventions used in a number of studies from the literature; these have employed education and dietary counseling without using multiple measures, multiple feedback, or successive approximation towards dietary goals. The comparison of the maximum and minimum intervention relates

to the relative effectiveness of the maximum intervention compared to the more usual interventions and to cost-effectiveness considerations.

Subjects.

Subjects in Study 2 met the same exclusionary criteria as in Study 1. Subjects were recruited from Derring Hall, and the adjacent Pamplin and Robeson Halls by the same procedure as in Study 1. The experimenter knocked on 89 doors over a period of 8 days. Of these, he made personal contact with 66 individuals, and 35 met the criteria and agreed to be screened. Of these, 4 did not keep their appointment to be screened. Of those screened, 21 did not fall into the acceptable range of serum cholesterol; 19 were too low and 2 were too high. Of the remaining 10 subjects, 9 agreed to be in the study. One subject dropped out after week two because he said he no longer had the time to be in the study. The remaining 8 subjects completed the study.

The mean age of all subjects was 46.4 years. The mean age of subjects 1-4 was 42.4 years with a range of 38 to 48.25 years. The mean age of subjects 5-8 was 50.4 years with a range of 35 to 60 years. Five subjects were professors at V.P.I. & S.U. and three were building and maintenance workers. Three professors and one maintenance worker were randomly assigned to the maximum intervention; two professors and two maintenance workers were randomly assigned to the minimum intervention.

Materials.

Total cholesterol was measured in the same way in as in Study 1 with the Reflotron. In addition, educational materials were derived from the same sources.

Design and Procedures.

The design of this study consisted of two phases and two experimental conditions. With the exception of one pair of subjects who were assigned together because they worked together, subjects were randomly assigned to the minimum or maximum intervention experimental condition.

Phase I.

Phase I was the baseline phase of the study. As in Study 1, all subjects were encouraged to continue eating as they normally did. All subjects kept food diares of all foods that they consumed for four days per week during this period. In an effort to increase the frequency of feedback and curtail the length of this study, subjects were measured with the Reflotron three times per week instead of two. These measurements were performed by both the experimenter and a first year graduate student trained to reliably perform the measurements. Seven measures of total cholestrol were obtained to establish a baseline. In addition, a blood sample for a laboratory lipid profile was obtained from these subjects in the middle of this phase.

Phase II.

Minimum Intervention.

In Phase II, those subjects assigned to the minimum intervention received the following within the first three days of this phase:

1) The same education materials described in Phase II of Study 1.

2) The same lipid profile analysis described in Phase II of Study 1.

3) The experimenter had a one hour education session with each of the subjects. The experimenter gave the subject visual feedback in the form of

a graph of their present levels of total serum cholesterol, dietary cholesterol, saturated fat, total fat, percent calories from fat, percent carbohydrate, and total calories. He described goal levels for these values. These values were obtained by using Nutritionist 3 software. In addition, he reviewed the subjects food diaries and gave them specific feedback about the types of food that they ate that were high in cholesterol and fats. He made suggestions about food substitutions that subjects could make. Subjects were encouraged by the experimenter to make the suggested changes in their diet to lower their serum cholesterol. He answered any questions that subjects had about their diet and cholesterol.

During Phase II, minimum intervention subjects ceased to keep dietary records, to be measured for serum cholesterol, or to meet with the experimenter. They were told that they would be contacted in approximately one month (the estimated time for maximum intervention subjects to lower their serum cholesterol levels).

Maximum Intervention.

Subjects in the maximum intervention received the same intervention as described in Study 1. The only difference was that subjects were measured three times per week instead of two times per week.

Dependent Measures.

The same dependent measures employed in Study 1 were also employed in Study 2.

RESULTS STUDY 2

Serum cholesterol change and dietary intervention.

Maximum intervention subjects.

Figure 6 and Table 6 show the total serum cholesterol levels for subjects on the maximum intervention.

INSERT FIGURE 6 HERE

INSERT TABLE 6 HERE

All maximum intervention subjects had lower serum cholesterol levels at the end of the intervention than during baseline. In addition, all subjects had their lowest average levels at the final period of the intervention. Subject 5 decreased 14.1% in serum cholesterol from baseline to the final phase of the intervention. Subject 6 made a reduction of 12.0%; Subject 7 dropped 20.0%, and Subject 8 made a reduction of 13.4% in his serum cholesterol. The mean of these percentage scores is 14.9%. With the exception of Subject 5, subject's serum cholesterol levels began to decrease after the expected lag period of Phase II. Subject 5's level began to drop during his baseline phase.

INSERT FIGURES 7, 8, 9 & 10 HERE

INSERT TABLE 7 HERE

Self-report macronutrient data are shown for Subjects 5-8 in Figures 7, 8, 9, and 10, and in Table 7. As shown in Figure 7, dietary data for Subject 5 does not show that he began to make changes in his diet during Phase I. It is not clear why Subject 5's serum cholesterol level dropped during this period.

In general, dietary data show that the intervention had its strongest and most immediate effect on changing the intake of Subjects 5 and 7

(dietary cholesterol, saturated fat, percentage fat, and total fat). Macronutrient values for Subject 6 are all lower during the Phase II lag than in baseline, however, this decrease is not as pronounced. It becomes more pronounced at the end of the lag period (Days 25-30) see Figure 8. Subject 8 shows reductions in some of these macronutrients during the lag period, however his reduction in these values is more evident during period A.

Comparisons of caloric intake of Subjects 5-8 between baseline and the last period of Phase II showed that Subjects 5, 6, 7 and 8 decreased their caloric intake by 543, 601, 447, and 264 kcal respectively (see also Figures 2, 3, 4 and 5). Reductions in caloric intake may make it difficult for subjects to maintain their cholesterol losses in the long-term because it is likely that subjects will eventually return to their pre-intervention calorie levels (Personal communication from Abby C. King, Ph.D to R. Winett, Ph.D, April, 1989). In this case, subjects may have changed the quantity of food eaten instead of the quality. One strategy that encourages maintenance of dietary changes is compensating for decreased fat intake with increased complex carbohydrate intake (Cooper, 1988). Comparisons of carbohydrate intake for Subjects 5, 6, 7 & 8 during these same periods show increases of 11.2, 1.9, 14.2, and 6.1% respectively. In addition, Subjects 5, 7 and 8 were very near the 60% goal of complex carbohydrates set by the National Cancer Institute and the American Heart Association. However, because they are not eating enough calories maintenance may still be difficult.

INSERT FIGURE 11 HERE

Figure 11 shows the serum cholesterol values for the minimum intervention subjects, Subjects 9-12. The percentage change in mean serum cholesterol levels from the baseline period to the follow-up period indicate that Subject 9 lost 10.9%; Subject 10, 3.7%; Subject 11, 6.5%; and

Subject 12, 15.3%. The mean of these percentages is 9.1%. These results indicate that the minimum intervention was less effective at lowering the serum cholesterol levels than the maximum intervention (14.9 % reduction).

INSERT FIGURE 12, 13, 14 & 15

Figures 12, 13, 14, and 15 show the dietary data for Subjects 9-12. Food diaries for Subjects 9-12 were included so that identical procedures were implemented for minimum and maximum subjects during the baseline period.

Lipid Analysis.

Lipid analyses were measured on Day 6 of the baseline and on Day 78 after the intervention. Results of the lipid analysis for Subjects 5-12 are shown in Table 5. Two of the maximum intervention subjects (Subjects 6 and 8) already had ratios below the 4.6/1.0 level. Their ratios had dropped slightly by the end of the intervention. The other two subjects were not able to decrease their ratios below the recommended 4.6/1.0 level. The net change was a 7.8 % decrease in the ratio, and the mean ratio at baseline was 4.8/1.0.

While HDL levels increased for Subjects 5, 6, and 8, they decreased for Subject 7. The net change was a 5.2% increase in HDL. LDL levels decreased for all subjects in the maximum intervention by a mean of 12.8 %.

Results for subjects in the minimum interventention show that Subject 9 increased his total cholesterol/HDL ratio, while for Subjects 10 and 11 it stayed roughly the same. Only Subject 11 had a level below 4.6/1.0 at baseline. The net change in the ratio was an increase of 5.6%. The mean ratio at baseline was 5.4 /1.0. Subject 12 refused to be measured for the lipid analysis. HDL levels decreased for Subject 9, stayed the same

for Subject 10 and increased slightly for Subject 11. LDL levels stayed the same for Subject 9, decreased for Subject 10, and increased for Subject 11. Hence, there was no consistent pattern of change for these subjects. Overall, there was a net decrease of 3.7% in LDL levels. Minimum intervention subjects all showed increases in triglycerides from baseline, and their net increase was 15.4%.

Intraindividual variability.

Only Subject 5 showed significant variability during his baseline period such that, depending upon when he was measured, he may have been misclassified. Results indicate that all serum cholesterol measures during the baseline period for each maximum intervention subject were higher than in subsequent intervention periods. Is it possible that subjects were eating more consistently and this had some effect on reducing variability of cholesterol scores? A comparison of macronutrient intake between the baseline and the three periods of the intervention phase (see Figures 7-10). shows that in 61 of 72 possible comparisons of macronutrient data, the standard deviations were lower during the intervention phase than during the baseline phase. Note that both the periods of comparison and the number of measures per period are not identical. Minimum intervention subject's serum cholesterol measures were not less variable after the minimum intervention was implemented. Similarly, they had fewer follow-up measures than baseline measures.

OVERALL RESULTS

Comparison of Results from Studies 1 and 2.

Serum cholesterol and variability.

During the baseline periods, subjects in Study 1 had a mean cholesterol level of 232.3 ± 11.1 mg/dl as compared to 241.9 ± 15.2 mg/dl for the subjects in Study 2. Thus cholesterol levels were 9.6 mg/dl higher for subjects in Study 2. Intraindividual variability levels, as measured by the average standard deviation scores for these periods, were also somewhat higher for subjects in Study 2. It should be noted that although there were the same number of measures taken during baseline for the two studies, the baseline period in Study 1 was more than twice as long as that of Study 2.

Comparing the final periods of the intervention reveals that subjects in Study 1 had mean serum cholesterol levels of 211.7 ± 6.1 mg/dl, and Study 2 maximum intervention subjects (Subjects 5, 6, 7, & 8) had levels of 206.9 ± 6.7 mg/dl. Because of the different baseline serum cholesterol levels, this level is an even larger percentage drop for maximum intervention subjects in Study 2. This difference again shows that Study 2 maximum intervention subjects were better able to lower their serum cholesterol levels. These Study 2 subjects also appear to have smaller intraindividual variability for this period than those in Study 1. Again, although the time frames for these two periods differed, the number of measures taken during the course of the intervention phase were roughly the same.

Overall, subjects in Study 1 were able to reduce their mean serum cholesterol at the end of the intervention by 10.1% compared to 14.9% for maximum intervention subjects in Study 2. In addition, every subject in the

maximum intervention was able to lower his cholesterol by at least 12%, whereas only one subject in the Study 1 was able to lower his cholesterol by at least12%. In addition, the maximum intervention subjects in Study 2 were able to achieve this larger reduction in only 47 days. Subjects 1 and 2 lost an average of 11.5% in 84 days, and Subjects 3 and 4 lost 8.6% in 57 days. Subjects in Study 2 were measured three times per week rather than the two times per week for subjects in Study 1. Subjects in the minimum intervention of Study 2 had the smallest losses of serum cholesterol at 9.1%.

Lipdid analyses.

All of the subjects in Study 1 had Total Cholesterol/HDL levels greater than the 4.6/1.0 level recommended by Cooper (1988) prior to the intervention (see Table 4). Two of these four were able to reduce their ratio at or below this 4.6/1.0 level at the end of the intervention. Overall, Study 1 subjects decreased this ratio by 16.7%. Their mean ratio at baseline was 5.8/1.0.

The maximum intervention subjects in Study 2 were different from the subjects in Study 1 in that two of the four subjects (Subjects 6 & 8) already had ratios below the 4.6/1.0 level at baseline (see Table 5). Their ratios dropped slightly at the end of the intervention. The other two subjects were not able to decrease their ratios below the recommended 4.6/1.0 level. The net change was a 7.8 % decrease in the ratio, and the mean ratio at baseline was 4.8/1.0. Hence, these subjects did not decrease the ratio as much as Study 1 subjects, but their average baseline level was 1.2 points less.

Total cholesterol/HDL ratios increased or stayed the same for the three subjects measured in the minimum intervention. Only Subject 11 had a level below 4.6/1.0 at baseline. The net change in the ratio was an increase of 5.6%. The mean ratio at baseline was 5.4/1.0.

LDL levels decreased for every subject in Study 1 at the end of the intervention. Their mean LDL level at baseline was 165.8 mg/dl, and their mean decrease was 12.8%. LDL levels also decreased for every maximum intervention subject in Study 2. Their mean LDL level was 150.0 mg/dl and their mean decrease was 12.2 %. For minimum intervention subjects in Study 2, LDL levels increased for one subject, stayed the same for one, and decreased for another. Their mean LDL level at baseline was 135.6 mg/dl, and their net change was a decrease of only 3.7%.

The changes in HDL levels for Study 1 subjects as a function of the intervention were variable. Two subjects decreased, one stayed the same, and one increased. The net change was a 1.2% increase in HDL. Their mean HDL level at baseline was 42.3 mg/dl. In Study 2, three of four maximum intervention subjects increased their HDL levels. The net change was a 5.2% increase in HDL. Their mean HDL level at baseline was 48.5 mg/dl. For Study 2 minimum intervention subjects, one decreased, one stayed the same, and one increased. The net change was 2.1% decrease in HDL. Their mean HDL level at baseline was 47.0 mg/dl.

Triglyceride measures for subjects in Study 1 reveal that every subject decreased his level at the end of the study. Their mean decrease was 45.1%, and their mean trigylceride level was 183.0 mg/dl at baseline. In Study 2, three of four maximum intervention subjects decreased their triglyceride levels. However, because of Subject 8's increase, the net change was an increase of 0.6%; their mean trigylceride level at baseline

was126.5 mg/dl. Minimum intervention subjects all showed increases in triglycerides from baseline, and their net increase was 15.4%. Their mean triglyceride level at baseline was 157.6 mg/dl.

In summary, Study 1 subjects were better able to reduce their Total cholesterol/HDL ratio than Study 2 maximum intervention subjects. However, their ratios were 1.2 points higher at baseline. In addition, Study 1 subjects better reduced their triglyceride levels than maximum intervention subjects. Subjects in both experimental conditions did about equally well in reducing their LDL levels. However, maximum intervention subjects from Study 2 were better able to increase their HDL levels than Study 1 subjects. Hence, these results are very mixed and show no clear pattern. The clearest result from these data are the poor results of minimum intervention subjects in Study 2. They had increased their Total cholesterol/HDL level, increased their triglycerides, decreased their HDL level, and only slighlty decreased their LDL level at the end of the intervention.

DISCUSSION

Serum Cholesterol Levels and Dietary Intervention.

The results of Study 1 indicate that the use of repeated measures, repeated feedback, shaping, dietary feedback, nutrition education and goal setting reduced the serum cholesterol levels in male subjects, albeit, only consistently for relatively short periods. However, these short-term reductions in serum cholesterol were superior using this intervention than those obtained in non-metabolic ward studies. The average loss in serum

cholesterol for these four subjects at the end of Phase II was 24.0 mg/dl or 10.1%. Because the average loss of non-metabolic studies is approximately 7%, this 10.1% reduction is better than this average.

Evidence for the efficacy of the dietary intervention is shown in two ways in Study 1. First, when the intervention was implemented subjects began to make immediate dietary changes in the form of reducing dietary cholesterol, saturated fat, and total fat. Subjects in the baseline condition did not make these same changes. Second, between 14 and 20 days after dietary changes, subject's serum cholesterol levels began to decrease. The serum cholesterol levels of subjects in the baseline condition remained the same or increased. In Study 2, maximum intervention subjects also began to make immediate dietary changes when the intervention was implemented. With the exception of Subject 5, subject's serum cholesterol levels also began to decrease after the expected lag period of Phase II. In addition, in both studies the reductions in dietary cholesterol, saturated fat, and total fat were sustained throughout the intervention periods and concurrent with serum cholesterol reductions.

It should be noted that the serum cholesterol levels of Subjects 3 and 4 began dropping at the end of their baseline period. The possibility that contact alone with the experimenter or simply being measured brought about these reductions (a placebo effect) is unlikely. Comparisons of dietary macronutrients indicates that Subjects 1 and 2 were reducing their intake of fats, while Subjects 3 and 4 were making no such changes. In addition, comparisons of carbohydrate intake between Subjects 1 and 2 vs. Subjects 3 and 4 shows that the intervention subjects were increasing their carbohydrate intake during the intervention, while the baseline subjects' levels of carbohydrate intake stayed the same. Comparisons of caloric

intake between these two groups of subjects for this period also indicate that the intervention subjects were decreasing their calories while the caloric intake of baseline subjects stayed the same.

At this point the cause of the drop in the serum cholesterol levels of Subjects 3 and 4 remains unclear. One possbility relates to the fact that self-report food diaries were obtained on three days per week and subjects may have been eating differently on the other four days per week. Another possibility is that this drop was caused by some unknown factor.

One of the purposes of Study 2 was to modify the procedures of Study 1 with the hopes of improving the rate and amount of serum cholesterol loss. The mean serum cholesterol loss for subjects in Study 1 was 10.1%, while for maximum intervention subjects in Study 2 the mean was 14.9%. In addition the subjects in Study 2 were able to achieve this larger reduction in the same period of time as Subjects 3 and 4, and in a shorter period of time than Subjects 1 and 2. These results suggest that the maximum intervention in Study 2 was superior to the intervention designed for Study 1. However, the data also suggest that for some individuals, a minimum contact intervention, as employed in Study 2, may be sufficient to lower serum cholesterol levels (e.g. Subject 12).

What is not clear is what specific element or interaction of elements of the maximum intervention, (e.g. increased frequency of contact, increased feedback of serum cholesterol level, subject characteristics) brought about these superior results. However, these results suggest a dose-response relationship. Maximum intervention subjects in Study 2 were exposed to the largest dose of the intervention by getting more feedback and having more contact with the experimenter; they also had the largest serum cholesterol reductions. Study 1 subjects were exposed to a

smaller dose of the intervention and had smaller serum cholesterol reductions. Finally, subjects in the minimum intervention in Study 2 had the smallest dose and their reductions were the smallest of the three groups.

Maintenance.

The success of subjects maintaining their serum cholesterol losses may be dependent on their adopting new patterns of eating that include an increase in complex carbohydrates to compensate for the lost calories from fat in their diets (Personal communication from Abby C. King, Ph.D to R. Winett, Ph.D., April 1989). A diet more suited for maintenance would consist of 60% calories from carbohydrate, and 25% from fat. Results from Study 1 indicate that only Subject 3 approached these goal levels. In addition, the serum cholesterol levels of Subjects 1 and 2 had both increased by 17.3 mg/dl and 12.0 mg/dl by the end of their short-term maintenance phase. Results from Study 2 indicate that Subjects 5, 7 and 8 closely approximated these goal levels, while Subject 6 did not. Future interventions should focus more specifically on increasing carbohydrate intake to goal levels of 60% to compensate for lost calories from fat.

Lipid Analyses and Data.

It appears that using total serum cholesterol alone as a measure of CHD risk may be a misleading, and that additional measures of HDL and LDL are needed. For example, while Subjects 6, 8, and 11 were at risk based on their Reflotron serum cholesterol levels, their Total cholesterol/HDL ratios showed that they were not at risk. Secondly, it appears (see Tables 4 and 5) that a diet alone intervention did not consistently and substantially increase HDL levels. Thus, only Subject 2 showed a large reduction in his

Total cholesterol/HDL ratio. Thirdly, the intervention for the subjects in Study 1 and the maximum intervention in Study 2 did appear to help lower LDL levels. All of these subjects had lower LDL levels after the intervention. In addition, seven of eight of these subjects had lower triglyceride levels after the intervention.

Thus while the intervention had positive effects of lowering total serum cholesterol, LDL, and triglyceride levels, it did not lower HDL levels. Copper (1988) suggests that some low-fat diets may have the unfortunate effect of lowering HDL levels as well as LDL and total cholesterol levels. Combining a dietary and aerobic exercise intervention may beneficially alter the Total cholesterol/HDL ratio (Cooper, 1988). He cites research showing that males who walked briskly five days a week increased their HDL levels 10-15%. Results from Study 1 and Study 2 suggest the need for a more complete intervention that includes an exercise component.

Effective Elements and Cost-effectiveness.

While it is not entirely clear what were the essential elements of the present maximum intervention, frequent contact and feedback distinguished the maximum intervention from other prior interventions (see Table 1). Further, the data from both studies suggest that greater contact and frequency of feedback was associated with greater reductions in serum cholesterol (i.e., maximum intervention Study 2, 14.9%; Study 1 intervention 10.1%, minimum intervention Study 2, 9.1%).

Another issue for future research relates to the cost-effectiveness of the present maximum intervention. While this approach was fairly labor-intensive in terms of the time spent with subjects, there may be ways to make it less so. There is a possibility that weekly dietary feedback could

be automated, and available to subjects at their will. In addition, the procedure for measuring total serum cholesterol with the Reflotron only takes about four minutes, and may be effectively done by a technician.

The question of cost-effectiveness was examined in a preliminary way. Using costs for each intervention (technician cost at $11.00/hour) it was found that each one percent reduction in Study 2 for the maximum intervention cost $12.11, while for the minimum intervention cost $10.35. Thus, it may be possible to reduce cardiovascular risk in a less costly way by modestly bolstering the minimum intervention while containing its costs. Likewise, and as suggested above, the costs of the maximum intervention may be reduced with, perhaps, its effectiveness maintained.

Food Diaries.

One criticism of this study relates to the use self-report food diaries. This method of obtaining diet information lacks an objective verification of what subjects are eating. It is possible that subjects may have intentionally or unintentionally underestimated the report of fats in their diet. In addition, it is possible that subjects may have eaten less fat on days that required a self-report, but ate more fat on days that did not require such a report. There is not a consistent way, short of confining subjects to a metabolic ward, to assess the accuracy of food diary data. However, it is possible that some food diary entrants may be observable, or other methods, reports or data be used to assess reliability. (Winett, Neal & Williams, 1979).

Intraindividual variability.

Another important finding in these studies relates to the issue of intraindividual variability. The premise that it is important to obtain multiple measures on individual's serum cholesterol was supported by the range of values seen in the baseline phase in two subjects. Without multiple measures, it may be difficult to accurately classify some subjects as either high risk or low risk. For example, depending on when Subject 3 was measured during the first three weeks of his baseline phase, his cholesterol level was as high as 246 mg/dl (High risk) or as low as 192 mg/dl (excellent protection). Subject 5, in Study 2, showed similar variability. Even using more extensive lipid analyses to assess CHD risk may be insufficient by themselves. These tests also have error and are also subject to intraindividual variability. Although the issue of individual variability may be important for some, the baseline data show that only two subjects would have been misdiagnosed. This finding is consistent with Hegsted and Nicolosi's (1987) finding that for a small number of people, the variance may be as much as ten percent.

Another issue relates to the variability of measures as a function of the intervention. In Study 2, the variabilty of all serum cholesterol measures for the maximum intervention subjects are lower in periods after the intervention. This finding must be qualified because these periods of comparison are nearly, but not exactly the same, nor are the number of measures per period identical. However, the hypothesis that subjects were eating more consistently in the intervention phase was also supported.

In Study 1, a similar comparison between baseline phase and subsequent periods of the study does not show this same downward trend in variability of serum cholesterol scores. Note that this comparison is more difficult because the baseline phase was twice as long as any of the

periods; more measures yields smaller variability. The only conclusion from both studies is that subjects seemed to be eating more consistently after the intervention.

Another issue relates to the effects of emotional arousal on serum cholesterol. Based on Subject 3's self-report, his level of emotional arousal had a large effect on elevating his serum cholesterol. Why Subject 3 continued to have elevated serum cholesterol even after these stressful events is not understood. In general, these results suggest that any given serum cholesterol reading may be a complex composite of emotional arousal factors, dietary factors, unknown factors, and measurement error. As such, it is important that multiple measures of serum cholesterol be employed rather than drawing conclusions from any single measure that could be an outlier attributable to emotional arousal.

External Validity.

A final issue relates to the external validity of these results. Eight of twelve subjects were highly educated University professors with a presumably high income. One area for future research is to examine the effectiveness of the present or similar interventions on lower SES populations with less education, and perhaps less motivation to change dietary and other health-related behaviors.

Summary Statement.

The conclusions from Study 1 and Study 2 must be interpreted cautiously because control subjects in one study (Study 1) apparently started dietary changes prior to their Phase II. Maintenance of effect for subjects 1 and 2 was minimal. However, at this point, large short-term

reductions in total serum cholesterol through dietary modifications (that may include reduction of total calories), have been demonstrated. These reductions in serum cholesterol approximate 20 to 30% reductions in CHD. In general, the results of these experiments indicate that an intervention using repeated measures, repeated feedback, shaping, dietary feedback, nutrition education and goal setting shows promise. Future research will need to refine these procedures and extend these findings while considering practical issues of costs, population characteristics, and maintenance of effects as well as further studying basic social, psychological, and biological processes that may influence intraindividual variability of serum choleterol measurement.

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List of Tables

Table 1. Representative Summary of Cholesterol Reduction Studies.

Study	Intervention	Results
National Diet-Heart (1968)	52 week low-cholesterol diet	29.1mg/dl (12% reduction) at 52 weeks.
Stern, Farquar, Maccoby & Russell (1977) - The Stanford Three-Community Study	Nine sessions of behavioral training and two years mass media health education campaign with 77 high-risk subjects.	5% reduction at 12 months.
Foreyt et al., (1979)	Normolipedemic subjects- 3 groups: Group A: diet manual Group B: weekly nutrition lessons for 8 weeks Group C: stimulus control training + dietary self-recording + same nutrition lessons	8.4% reduction for Group C but no maintenance
MRFIT (1981)	Multi-community study of 12,866 high risk male subjects. SI group counseled to reduce their saturated fat intake to less than 8% of total calories and dietary cholesterol to less than 250 mg/day.	15.4 mg/dl (6%) at 12 months; 7.4% at 60 months
Reeves et al. (1983)	Three year community study of normolipedemic subjects. Subjects mailed cookbook of low cholesterol meals, media coverage, and 4 nutrition lessons presented by a dietitian over 12 months.	7.0 mg/dl (3.0%) at 12 months
Bruno et al., (1983)	Worksite study. 8-week group cholesterol reduction programwith food behavior change, nutrition education, physical activity planning, and self-management skills.	17 mg/dl (6.4%) at 3 months which was 1 month post treatment.
Lefebvre et al., (1986)	Community study using volunteer delivery system to 1439 participants. 4 Screening, Counseling,and Referral events (SCOREs). Behavioral counseling and nutrition education + recipe contest + 6 weekly newspaper columns	Of original,1439 subjects, 72% took part in 2 month follow-up. 57% had lowered by an average of 29.1 mg/dl. 41% had increased by an average of 19.6 mg/dl.

Peterson et al., (1986)	Same PHHP study targeting hospital employees. SCOREs, dietary counseling + self-help nutrition kit.	Of original subjects 26% took part in 6 month follow-up. 26 mg/dl (10.9%) reduction.

Table 2. Comparison of Mean Serum Cholesterol Values and Standard Deviations across Phases for Subjects 1-4.

Time Period	Subject 1	Subject 2	Time Period	Subject 3	Subject 4
Phase I (Day 0-42)	248.9 ± 9.7	241.0 ± 9.7	**Day 0-42**	218.3 ± 16.7	221.1 ± 8.2
Phase II (Day 58-142)	227.4 ± 9.4	225.4 ± 11.9	**Day 58-142**	225.6 ± 20.9	246.6 ± 13.8
A	237.3 ± 5.1	230.5 ± 7.7	**A**	241.3 ± 13.1	247.0 ± 12.5
B	231.3 ± 4.4	235.0 ± 6.4	**B**	238.6 ± 5.1	256.4 ± 9.7
C	233.0 ± 0.0	222.0 ± 0.0	**C**	234.0 ± 4.4	254.0 ± 0.0
D	217.7 ± 6.4	215.7 ± 11.3	**D**	198.6 ± 11.4	235.2 ± 12.7
Phase III (Day 143-200)	226.8 ± 7.9	220.0 ± 32.0	**Day 143-200**	193.2 ± 18.8	227.2 ± 10.7
E	226.3 ± 5.5	208.8 ± 45.0			
F	223.0 ± 0.0	223.0 ± 35.9			
G	229.7 ± 5.0	233.0 ± 8.5			
			Phase I (Day 0-142)	222.6 ± 18.8	239.6 ± 16.6
			Phase II (Day 157-200)	191.7 ± 17.3	222.7 ± 8.3
			F'	180.6 ± 14.9	226.2 ± 10.7
			G'	202.8 ± 5.4	219.8 ± 5.1

Table 3. Means and Standard Deviations of Dietary Values across Phases for Subjects 1-4.

Subject 1.

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I Days 0-42	181.0 ± 130.0	22.8 ± 14.9	31.5 ± 6.5	100.5 ± 39.2	44.6 ± 8.5	2734 ± 677
Phase II Days 42-142	127.6 ± 65.3	10.0 ± 4.9	22.0 ± 4.5	51.3 ± 18.8	54.0 ± 6.5	1990 ± 421
lag	121.6 ± 46.7	6.9 ± 1.5	18.8 ± 3.4	36.1 ± 4.8	56.8 ± 8.3	1702 ± 244
A	165.4 ± 27.9	9.5 ± 2.4	23.7 ± 4.3	48.4 ± 7.2	51.7 ± 8.3	1807 ± 259
B	156.3 ± 73.2	12.5 ± 5.8	24.6 ± 4.1	59.4 ± 20.1	52.4 ± 6.6	2114 ± 468
C	112.2 ± 68.1	7.5 ± 4.3	22.1 ± 5.9	56.8 ± 29.9	54.6 ± 7.7	2247 ± 565
D	152.6 ± 78.4	11.4 ± 5.2	22.4 ± 3.4	50.6 ± 13.8	55.4 ± 3.8	1976 ± 311
Phase III Days 142-200	159.2 ± 75.6	11.6 ± 4.7	22.2 ± 5.9	51.4 ± 17.2	54.8 ± 5.5	1941 ± 474
E	171.3 ± 71.3	10.4 ± 4.5	20.9 ± 6.2	45.7 ± 18.8	55.1 ± 5.4	1896 ± 256
F	125.6 ± 76.5	11.5 ± 5.2	25.4 ± 5.0	56.1 ± 13.9	54.8 ± 7.6	1962 ± 250
G	193.7 ± 92.8	16.5 ± 3.1	26.5 ± 3.5	56.5 ± 18.4	53.0 ± 2.8	2163 ± 68

Table 3 cont.

Subject 2.

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I Days 0-42	326.0 ± 255.6	25.2 ± 6.6	41.7 ± 6.9	95.6 ± 21.8	36.2 ± 9.8	2081 ± 474
Phase II Days 42-142	121.7 ± 66.6	11.8 ± 5.2	30.3 ± 5.6	44.6 ± 17.8	46.3 ± 7.0	1302 ± 414
lag	158.0 ± 53.2	15.8 ± 6.8	32.3 ± 5.3	62.7 ± 27.5	44.7 ± 5.6	1695 ± 593
A	171.8 ± 67.6	9.5 ± 1.8	29.4 ± 7.3	45.3 ± 13.3	43.1 ± 3.7	1378 ± 314
B	115.3 ± 60.2	11.8 ± 6.2	29.5 ± 3.5	44.5 ± 17.4	49.0 ± 4.8	1315 ± 428
C	110.5 ± 75.8	11.2 ± 3.9	31.1 ± 5.6	42.3 ± 13.8	41.8 ± 10.3	1205 ± 334
D	85.9 ± 52.5	11.6 ± 4.8	29.5 ± 6.0	37.5 ± 12.9	49.4 ± 6.6	1122 ± 288
Phase III Days 142-200	123.3 ± 76.1	13.9 ± 7.1	32.0 ± 5.7	48.8 ± 18.7	46.5 ± 6.4	1325 ± 379
E	144.4 ± 78.2	14.5 ± 7.2	32.2 ± 5.9	50.3 ± 17.7	46.2 ± 6.9	1376 ± 349
F	88.6 ± 70.5	14.3 ± 9.5	30.3 ± 7.8	46.5 ± 32.0	48.6 ± 7.6	1252 ± 602
G	62.0 ± 33.3	9.9 ± 4.6	35.0 ± 0.0	51.1 ± 12.6	44.5 ± 2.1	1369 ± 403

Table 3 cont.

Subject 3.

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Days 0-42	185.5 ± 230.4	18.6 ± 20.0	26.9 ± 8.0	66.3 ± 52.6	55.6 ± 10.7	2070 ± 1123
Days 42-142	168.2 ± 175.9	16.3 ± 13.1	28.5 ± 9.5	53.9 ± 31.4	52.0 ± 11.7	1632 ± 758
lag	93.9 ± 49.0	12.6 ± 11.0	28.8 ± 9.9	45.3 ± 25.6	52.5 ± 12.8	1392 ± 335
A	171.5 ± 101.0	20.1 ± 12.6	31.0 ± 10.1	65.3 ± 29.2	46.4 ± 10.9	1829 ± 568
B	308.1 ± 284.6	23.7 ± 16.9	31.3 ± 7.3	71.7 ± 35.8	48.5 ± 11.2	2082 ± 1100
C	99.7 ± 64.9	12.5 ± 13.0	27.6 ± 7.1	49.7 ± 29.8	55.3 ± 9.3	1499 ± 625
D	133.2 ± 195.7	9.5 ± 5.9	22.1 ± 12.3	28.0 ± 20.0	56.2 ± 13.1	1149 ± 680
Phase II	85.5 ± 58.4	8.1 ± 4.5	21.2 ± 9.2	28.1 ± 17.8	57.0 ± 10.7	1187 ± 408
E	78.3 ± 30.6	7.2 ± 4.1	20.0 ± 9.5	23.9 ± 16.5	57.8 ± 8.5	1072 ± 381
F'	106.5 ± 88.3	9.9 ± 5.8	24.8 ± 10.0	38.8 ± 19.4	58.7 ± 15.6	1402 ± 379
G'	72.3 ± 22.4	8.0 ± 2.1	19.5 ± 2.1	23.5 ± 9.3	49.0 ± 1.4	1354 ± 548

Table 3 cont.

Subject 4.

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Days 0-42	364.7 ± 175.3	27.4 ± 8.7	31.7 ± 10.2	93.7 ± 28.2	49.9 ± 6.8	2407 ± 436
Days 42-142	296.0 ± 164.0	24.0 ± 11.0	32.2 ± 8.5	87.7 ± 33.2	51.7 ± 8.9	2368 ± 650
lag	235.3 ± 199.0	15.3 ± 7.5	25.3 ± 6.5	65.9 ± 33.1	60.3 ± 9.3	2217 ± 720
A	383.4 ± 194.3	28.1 ± 9.8	35.6 ± 6.7	96.9 ± 29.9	47.4 ± 4.3	2428 ± 463
B	310.3 ± 92.0	27.2 ± 14.5	35.3 ± 5.8	115.9 ± 29.6	49.0 ± 7.8	2906 ± 539
C	278.1 ± 191.5	21.7 ± 7.7	29.8 ± 5.0	90.6 ± 28.3	54.4 ± 9.1	2368 ± 661
D	258.9 ± 119.8	25.8 ± 11.4	37.2 ± 6.3	73.9 ± 25.2	47.8 ± 9.6	1738 ± 320
Phase II	166.0 ± 68.9	12.1 ± 5.4	28.4 ± 6.5	58.0 ± 17.3	53.3 ± 8.6	1864 ± 504
E	161.5 ± 64.0	10.4 ± 4.2	27.3 ± 7.1	55.6 ± 16.7	55.3 ± 10.2	1867 ± 571
F'	128.9 ± 110.9	16.4 ± 7.9	34.0 ± 2.4	73.5 ± 14.8	50.3 ± 5.1	1975 ± 568
G'	203.0 ± 43.2	12.1 ± 3.4	24.7 ± 3.5	45.4 ± 7.8	50.6 ± 5.5	1706 ± 116

Table 4.

Lipid Analysis
Study 1

Measures taken by Roche Laboratories.

Subject	1		2		3			4		
Measurement	Day 14	Day 143	Day 14	Day 143	Day 14	Day 143	Day 200	Day 14	Day 143	Day 200
Total Cholesterol	267	245	230	180	202	233	194	221	249	211
HDL Cholesterol (mg/dl)	46	43	42	39	45	37	45	43	44	44
LDL Cholesterol (mg/dl)	186	177	138	124	141	175	132	151	164	145
Triglycerides (mg/dl) 204 112		177	124	250	81	79	101	85	132	
Total Cholesterol/HDL ratio	5.8	5.7	5.5	4.6	4.4	6.3	4.3	5.2	5.7	4.8
Age	53		44		46			53		

Table 5. Lipid Analysis for Subjects 5-12.

Measures taken by Roche Laboratories.

Maximum Subjects.

Subject	5		6		7		8	
Measurement	Day 6	Day 78	Day 6	Day 78	Day 6	Day 78	Day 6	Day 78
Total Cholesterol	212	207	237	224	244	199	203	205
HDL Cholesterol (mg/dl)	39	44	62	65	39	32	54	63
LDL Cholesterol (mg/dl)	135	130	159	143	171	133	135	121
Triglycerides (mg/dl)	101	190	163	79	78	170	167	67
Total Cholesterol/HDL ratio	5.4	4.7	3.8	3.5	6.3	6.2	3.7	3.3
Age	47		48		36		38	

Minimum Subjects.

Subject	9		10		11		12	
Measurement	Day 6	Day 78	Day 6	Day 78	Day 6	Day 78	Day 6	Day78
Total Cholesterol	198	200	215	214	230	247	refused to be measured	
HDL Cholesterol (mg/dl)	40	35	26	26	75	77		
LDL Cholesterol (mg/dl)	140	140	137	112	130	140		
Triglycerides (mg/dl)		89	123	259	376	125	147	
Total Cholesterol/HDL ratio	4.9	5.7	8.2	8.2	3.1	3.2		
Age	48		61		56		35	

Table 6. Comparison of Mean Serum Cholesterol Values and Standard Deviations across Phases for Subjects 5-12.

Subject	Phase I (Days 1-17)	lag (Days 18-32)	Phase II (Days 33-51)	(Days 52-67)
5	225.7 ± 14.5	212.0 ± 8.0	196.4 ± 3.9	193.8 ± 4.9
6	241.9 ± 20.6	232.0 ± 10.1	217.3 ± 14.6	212.8 ± 5.3
7	264.7 ± 18.7	246.8 ± 16.3	234.0 ± 11.3	211.7 ± 5.1
8	237.3 ± 19.5	238.0 ± 0.0	242.5 ± 10.5	209.3 ± 11.4

	(Days 1-17)	(Days 56-64)
9	239.7 ± 16.4	213.5 ± 2.6
10	240.8 ± 5.6	232.0 ± 4.3
11	238.4 ± 18.1	222.8 ± 20.4
12	246.8 ± 7.9	209.0 ± 17.6

Table 7. Means and Standard Deviations of Dietary Values across Phases for Subjects 5-12.

Subject 5

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	178.3 ± 90.2	14.9 ± 8.3	28.5 ± 11.9	60.8 ± 29.4	47.8 ± 16.5	1902 ± 517
Phase II						
Lag (Days 18-31)	115.8 ± 51.3	7.8 ± 7.6	19.2 ± 7.1	34.4 ± 20.4	53.2 ± 2.5	1527 ± 526
A (Days 32-51)	109.1 ± 45.5	13.2 ± 6.5	26.2 ± 4.9	54.1 ± 30.0	51.0 ± 14.4	2010 ± 419
B (Days 52-End)	83.1 ± 32.4	11.2 ± 5.1	22.3 ± 5.3	32.1 ± 16.8	59.0 ± 6.7	1359 ± 237

Subject 6

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	221.0 ± 155.8	38.6 ± 11.3	32.4 ± 6.1	99.3 ± 22.0	50.9 ± 6.5	2782 ± 591
Phase II						
Lag (Days 18-31)	199.6 ± 154.9	35.4 ± 15.9	30.8 ± 7.0	83.8 ± 35.9	54.3 ± 9.2	2103 ± 597
A (Days 32-51)	165.3 ± 76.2	32.4 ± 10.3	27.7 ± 4.8	71.9 ± 19.1	57.4 ± 4.3	2172 ± 448
B (Days 52-End)	108.3 ± 33.0	30.0 ± 9.4	28.2 ± 5.8	65.1 ± 19.8	52.8 ± 10.7	2181 ± 569

Table 7 cont.

Subject 7

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	338.9 ± 279.3	19.9 ± 6.1	34.8 ± 7.9	74.5 ± 22.2	47.8 ± 10.1	1909 ± 679
Phase II						
Lag (Days 18-31)	137.9 ± 118.1	7.7 ± 3.0	20.5 ± 6.5	28.3 ± 9.0	63.2 ± 4.4	1219 ± 148
A (Days 32-51)	99.0 ± 76.1	9.0 ± 4.0	16.8 ± 4.0	21.0 ± 6.4	66.5 ± 3.8	1318 ± 220
B (Days 52-End)	81.5 ± 19.8	10.6 ± 1.7	22.8 ± 3.3	23.7 ± 6.5	62.0 ± 5.0	1462 ± 197

Subject 8

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	159.3 ± 181.0	16.8 ± 27.1	29.0 ± 15.9	64.7 ± 66.0	43.5 ± 16.4	1644 ± 902
Phase II						
Lag (Days 18-31)	149.1 ± 29.0	17.2 ± 4.3	23.3 ± 21.0	43.6 ± 3.7	57.0 ± 11.3	1221 ± 332
A (Days 32-51)	113.2 ± 82.5	9.9 ± 7.8	19.9 ± 9.2	28.8 ± 19.2	63.1 ± 10.3	1153 ± 487
B (Days 52-End)	124.4 ± 72.1	9.1 ± 2.8	26.6 ± 4.9	26.7 ± 4.9	60.3 ± 8.9	1380 ± 272

Table 7 cont.

Subject 9

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	183.5 ± 96.7	19.6 ± 11.4	32.2 ± 7.8	65.0 ± 19.2	41.6 ± 7.1	1720 ± 339

Subject 10

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	237.5 ± 170.0	25.6 ± 7.2	29.7 ± 6.4	64.1 ± 21.1	53.2 ± 4.4	1783 ± 297

Subject 11

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	134.7 ± 107.3	11.8 ± 3.7	24.5 ± 4.7	45.0 ± 13.2	57.0 ± 5.3	1849 ± 203

Subject 12

Phase	Dietary Cholesterol	Saturated Fat	Percentage Fat	Total Fat	Percentage Carbohydrate	Total Calories
Phase I (Days 0-17)	325.2 ± 184.8	15.8 ± 7.2	42.5 ± 10.5	58.6 ± 17.9	33.6 ± 5.2	1660 ± 274

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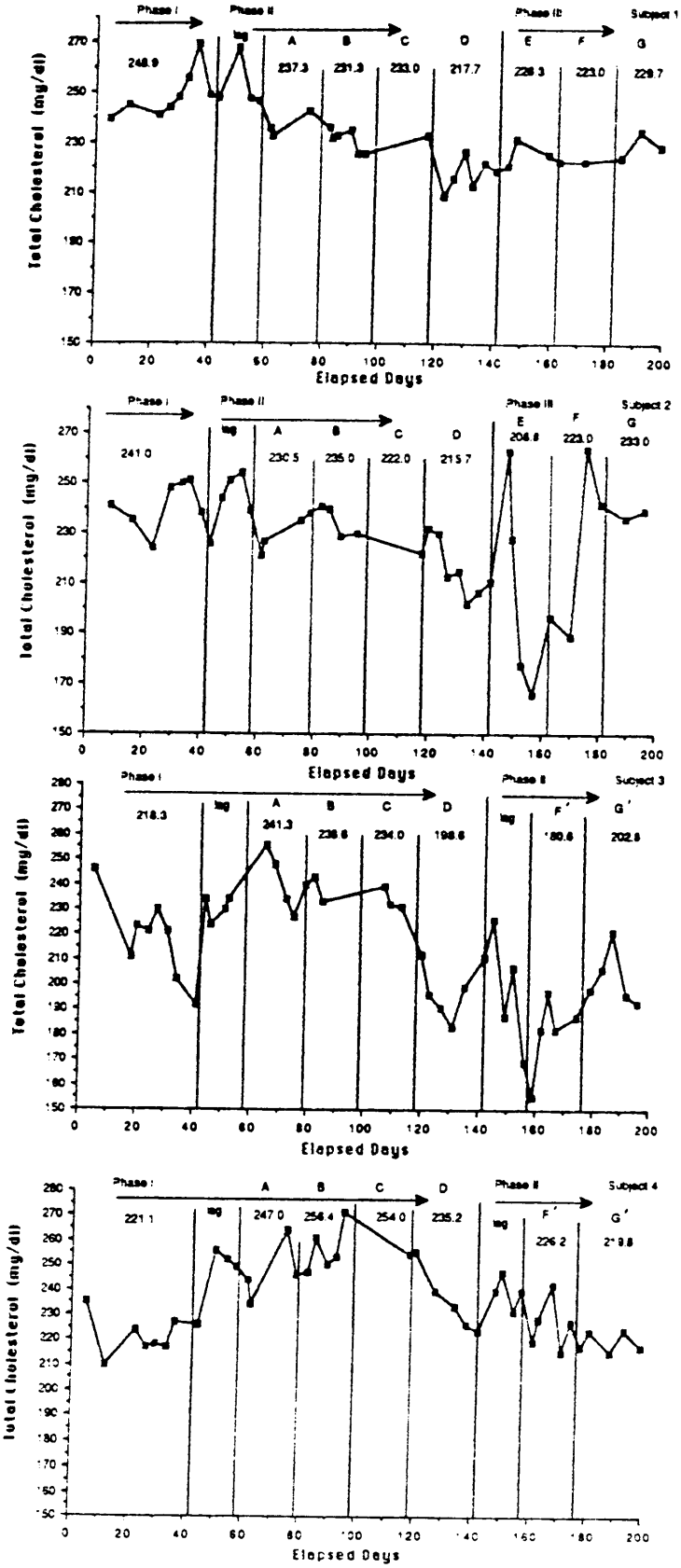
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Phase II
Phase III
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Subject 2
Subject 3
Subject 4
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B
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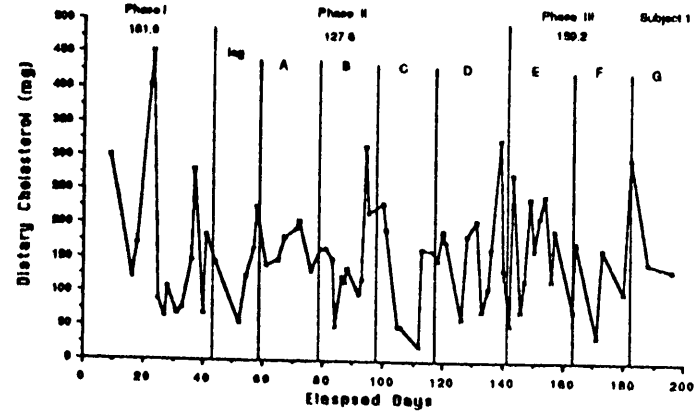
Total Cholesterol (mg/dl)
Elapsed Days

Figure 1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F
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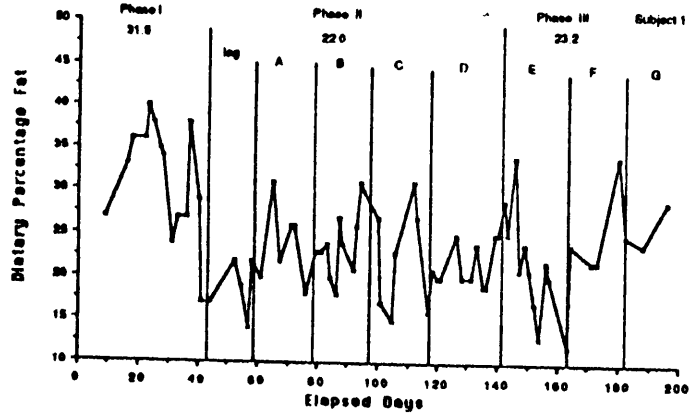
Dietary Cholesterol (mg)
Elapsed Days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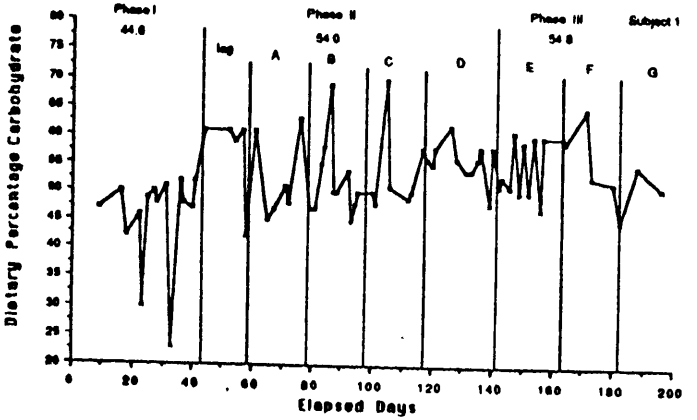
Dietary Percentage Fat
Elapsed Days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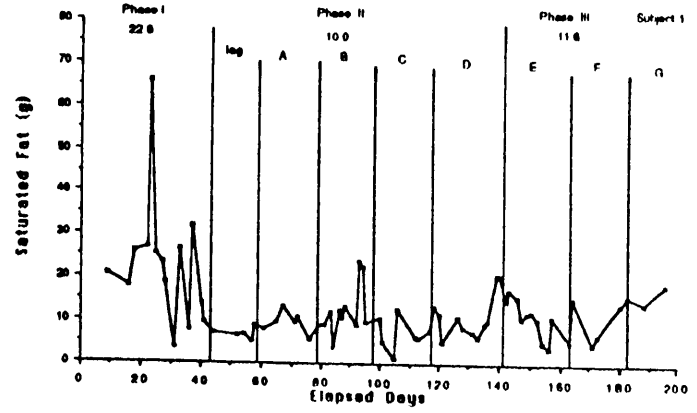
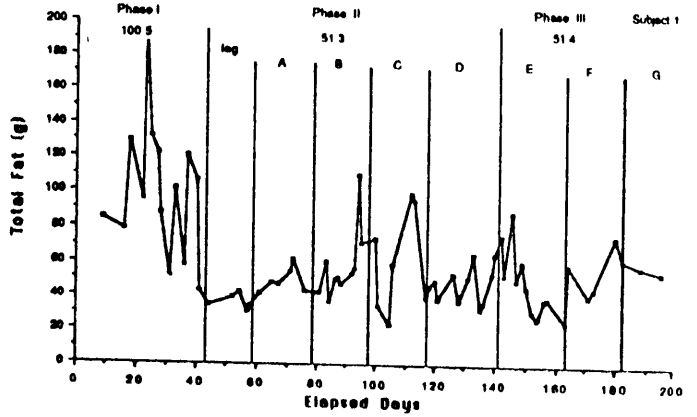
Dietary Percentage Carbohydrate
Elapsed Days


Phase I
22.8
Phase II
10.0
Phase III
11.6
Subject 1
lag
A
B
C
D
E
F
G
Saturated Fat (g)
Elapsed Days


Phase I
100.5
Phase II
51.3
Phase III
51.4
Subject 1
lag
A
B
C
D
E
F
G
Total Fat (g)
Elapsed Days

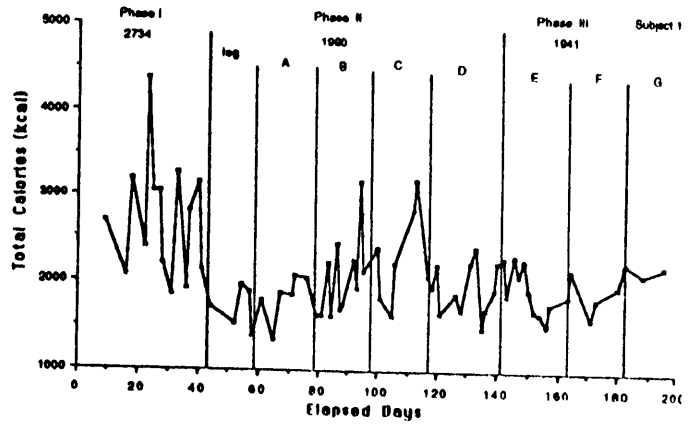
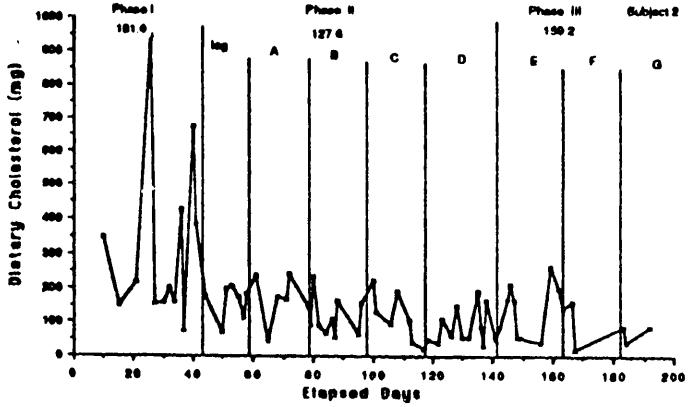
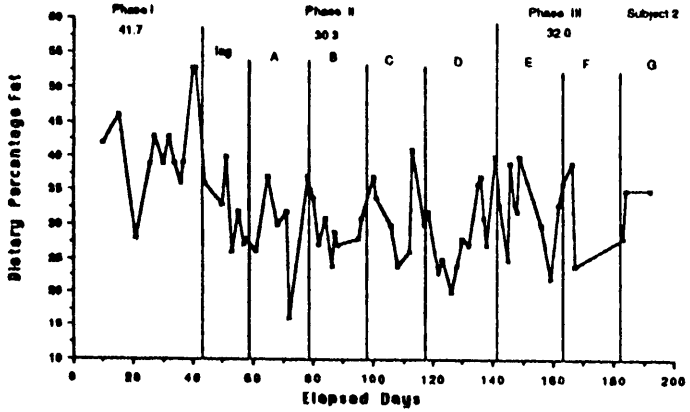
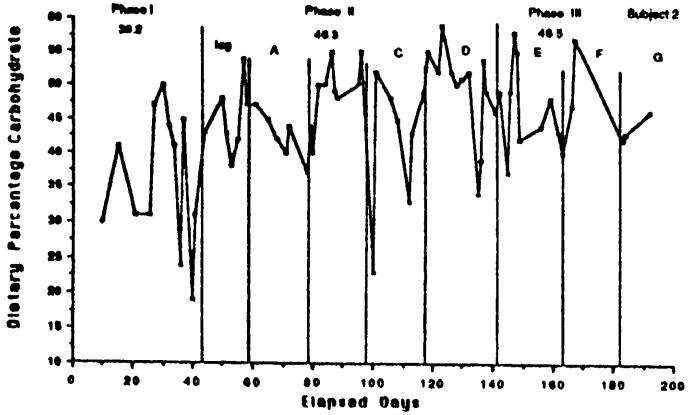
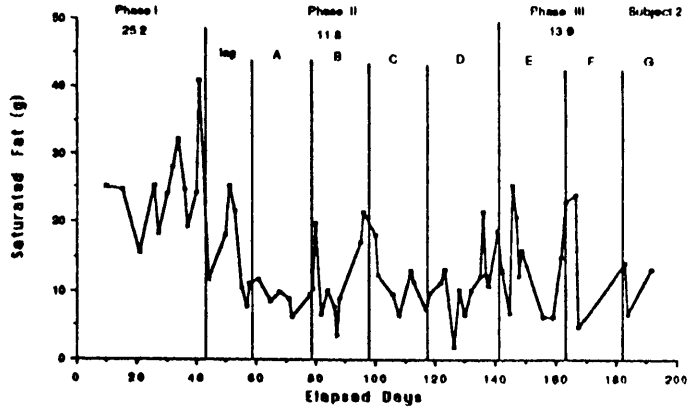

Phase I
2734
Phase II
1980
Phase III
1941
Subject 1
lag
A
B
C
D
E
F
G
Total Calories (kcal)
Elapsed Days

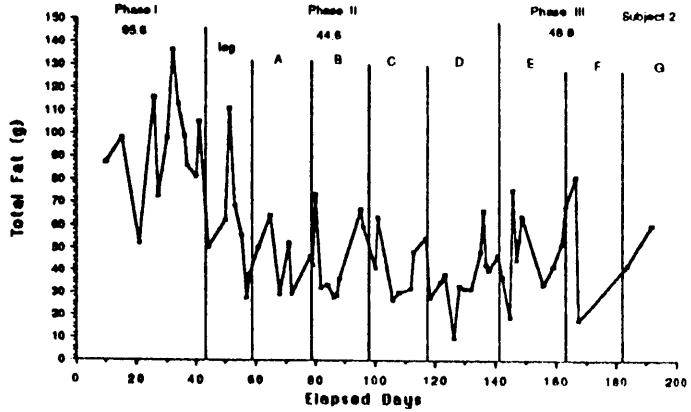
Figure 2


Phase I
181.6
Phase II
127.6
Phase III
150.2
Subject 2
lag
A
B
C
D
E
F
G
Dietary Cholesterol (mg)
Elapsed Days


Phase I
41.7
Phase II
30.3
Phase III
32.0
Subject 2
lag
A
B
C
D
E
F
G
Dietary Percentage Fat
Elapsed Days


Phase I
38.2
Phase II
46.3
Phase III
46.5
Subject 2
lag
A
C
D
E
F
G
Dietary Percentage Carbohydrate
Elapsed Days


Phase I
25.2
Phase II
11.8
Phase III
13.9
Subject 2
lag
A
B
C
D
E
F
G
Saturated Fat (g)
Elapsed Days


Phase I
85.6
Phase II
44.6
Phase III
46.8
Subject 2
lag
A
B
C
D
E
F
G
Total Fat (g)
Elapsed Days

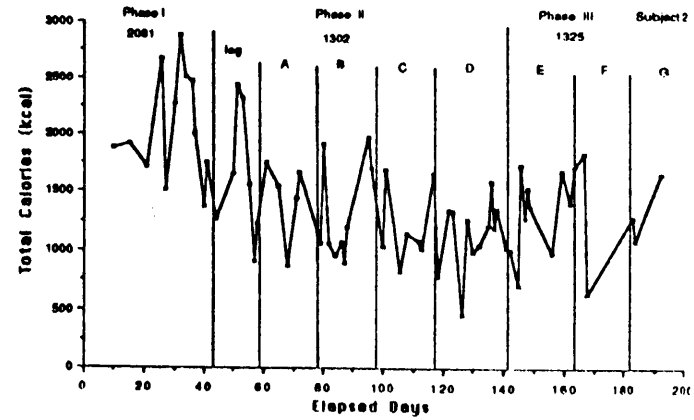
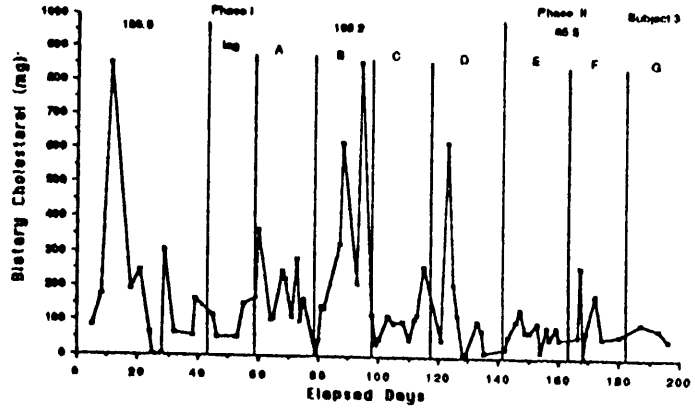
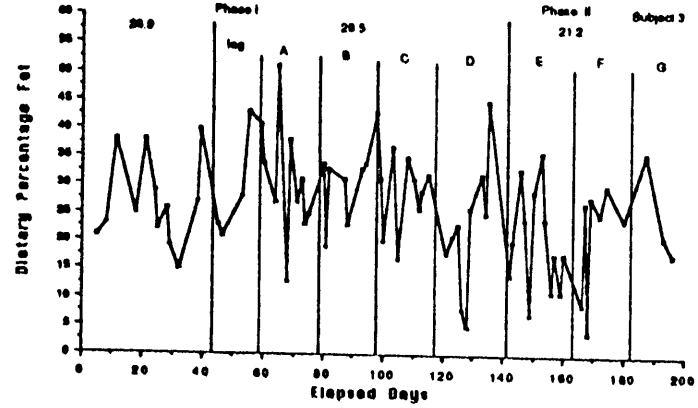
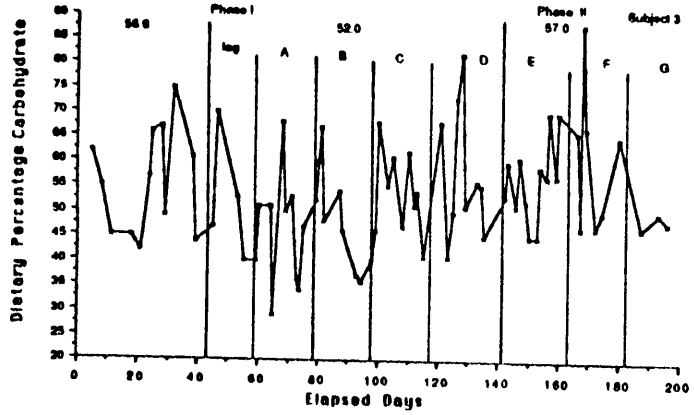
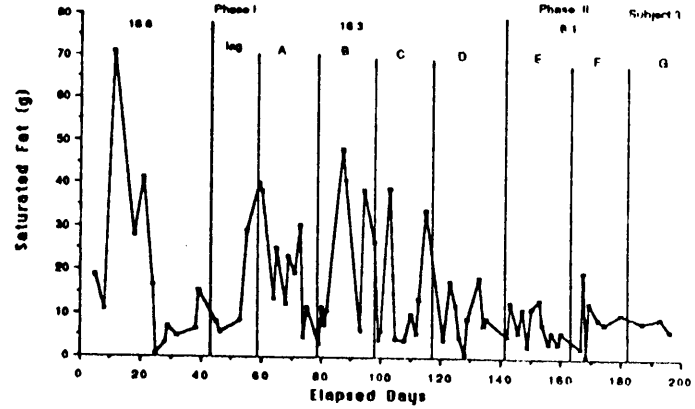

Phase I
2081
Phase II
1302
Phase III
1325
Subject 2
lag
A
B
C
D
E
F
G
Total Calories (kcal)
Elapsed Days

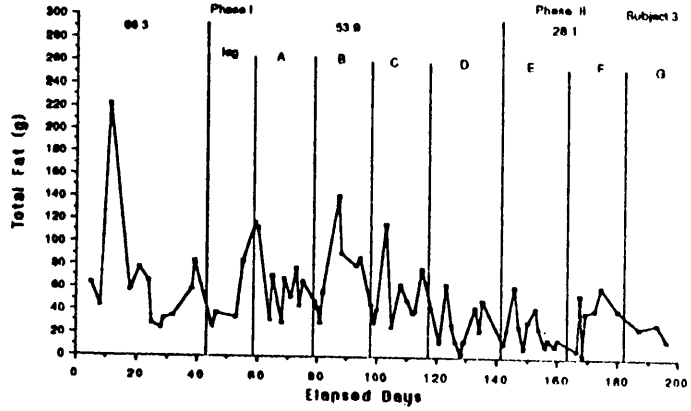
Figure 3


Dietary Cholesterol (mg)
Phase I
Phase II
Subject 3
lag
A
B
C
D
E
F
G
Elapsed Days


Dietary Percentage Fat
Phase I
Phase II
Subject 3
lag
A
B
C
D
E
F
G
Elapsed Days


Dietary Percentage Carbohydrate
Phase I
Phase II
Subject 3
lag
A
B
C
D
E
F
G
Elapsed Days


Saturated Fat (g)
Phase I
Phase II
Subject 3
lag
A
B
C
D
E
F
G
Elapsed Days


Total Fat (g)
Phase I
Phase II
Subject 3
lag
A
B
C
D
E
F
G
Elapsed Days

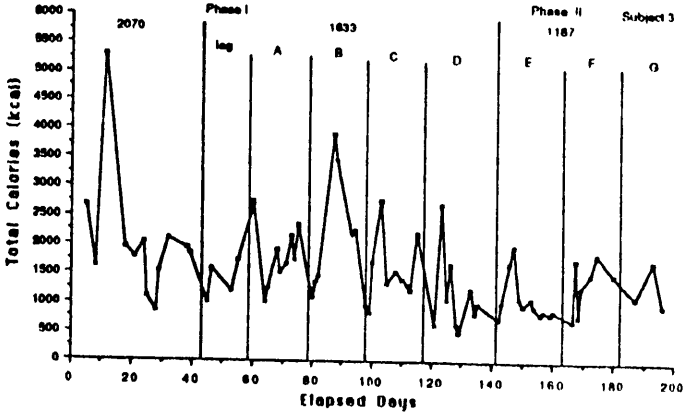
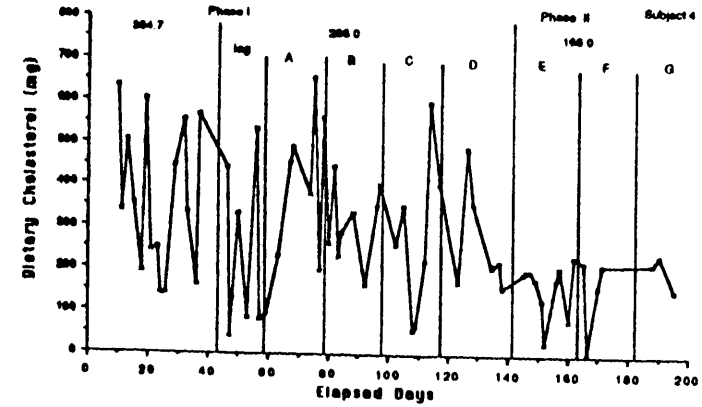
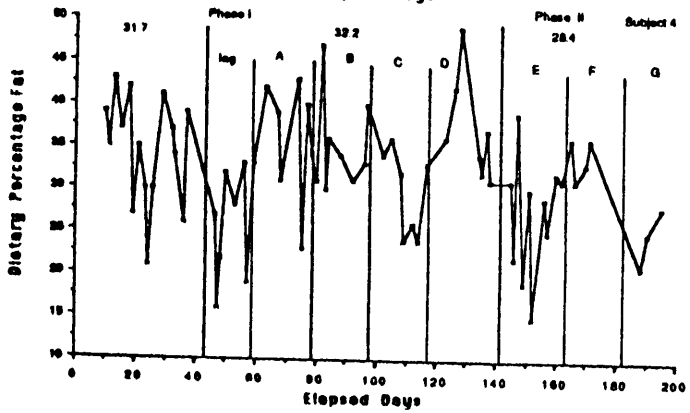
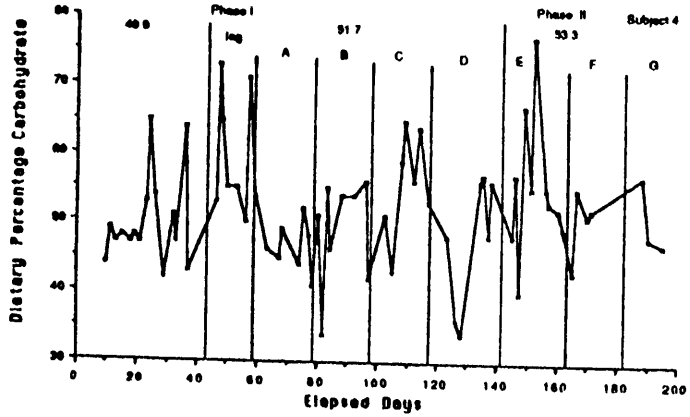
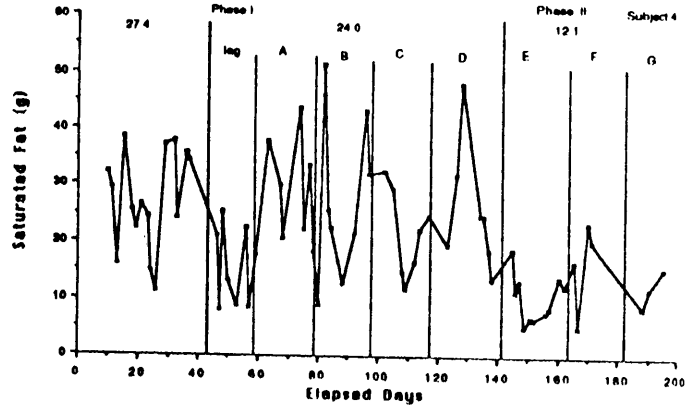

Total Calories (kcal)
Phase I
Phase II
Subject 3
lag
A
B
C
D
E
F
G
Elapsed Days

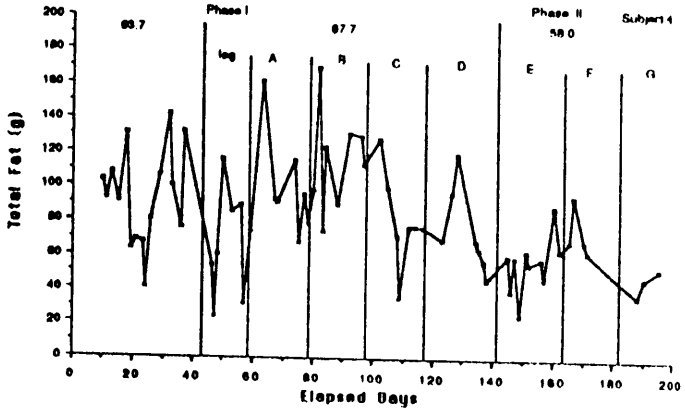
Figure 4


Dietary Cholesterol (mg)
Phase I
Phase II
Subject 4
Elapsed Days


Dietary Percentage Fat
Phase I
Phase II
Subject 4
Elapsed Days


Dietary Percentage Carbohydrate
Phase I
Phase II
Subject 4
Elapsed Days


Saturated Fat (g)
Phase I
Phase II
Subject 4
Elapsed Days


Total Fat (g)
Phase I
Phase II
Subject 4
Elapsed Days

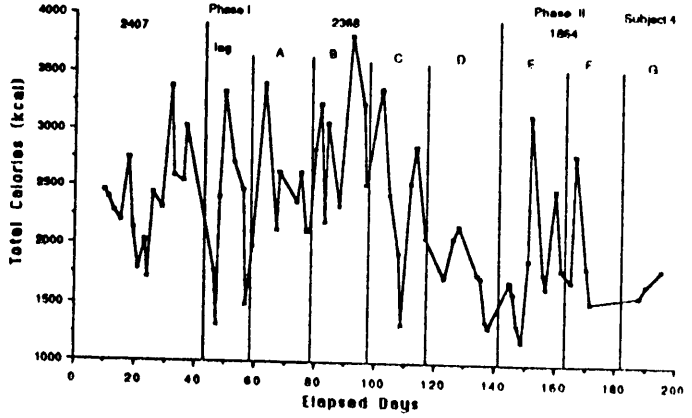

Total Calories (kcal)
Phase I
Phase II
Subject 4
Elapsed Days

Figure 5

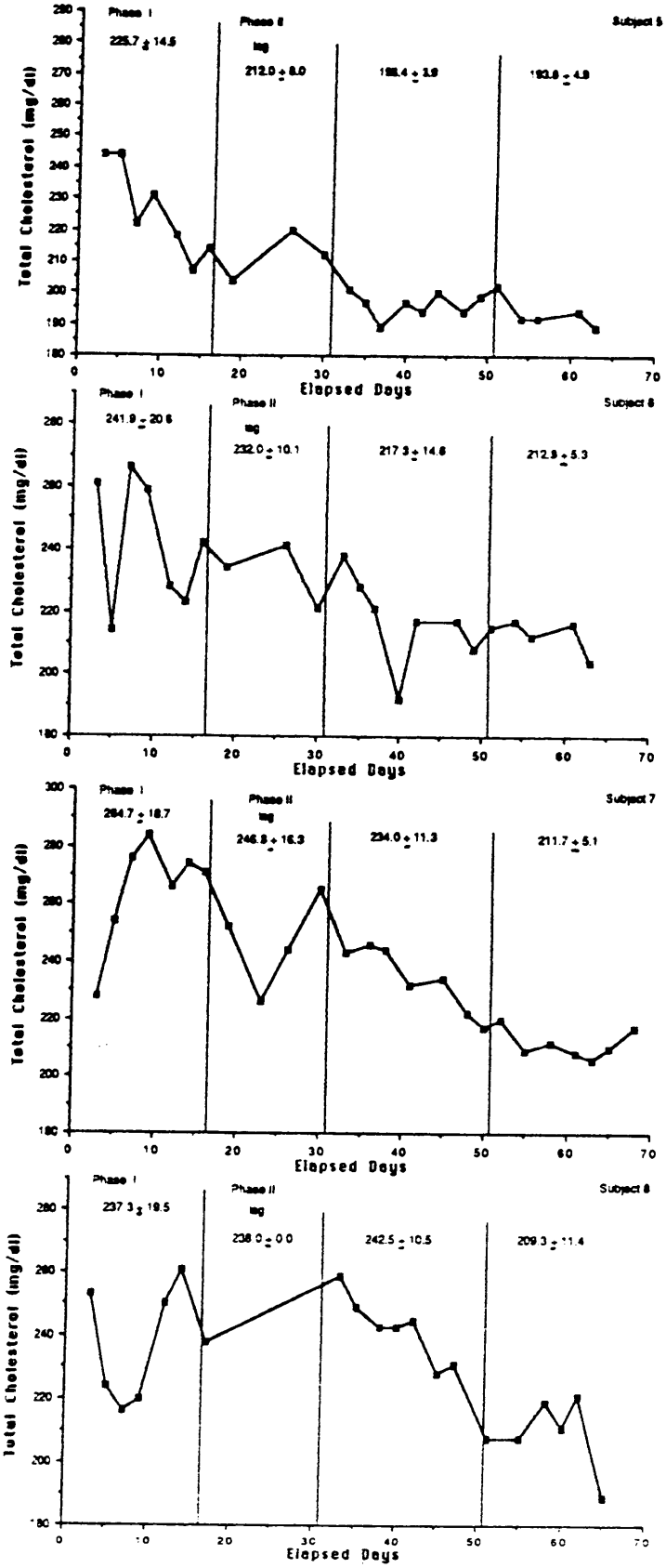
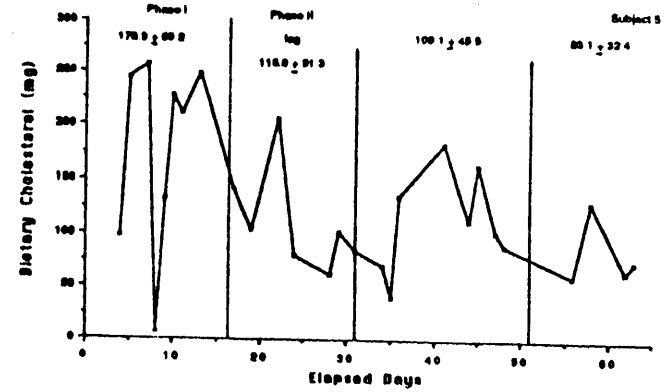
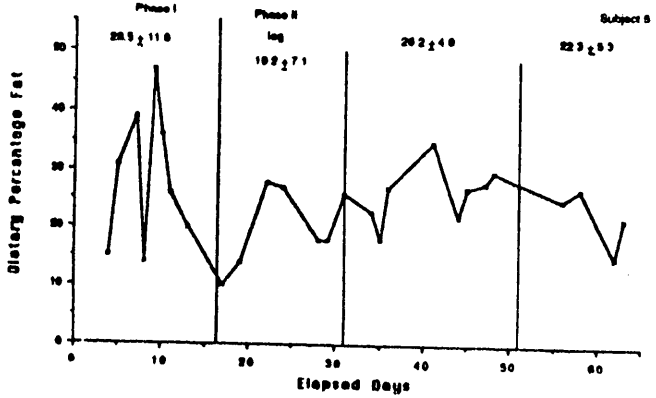
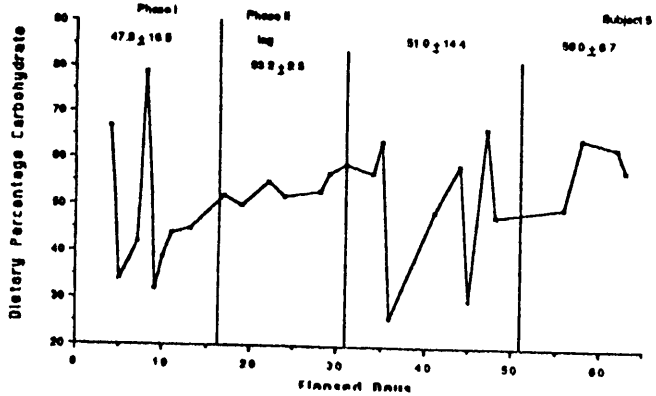
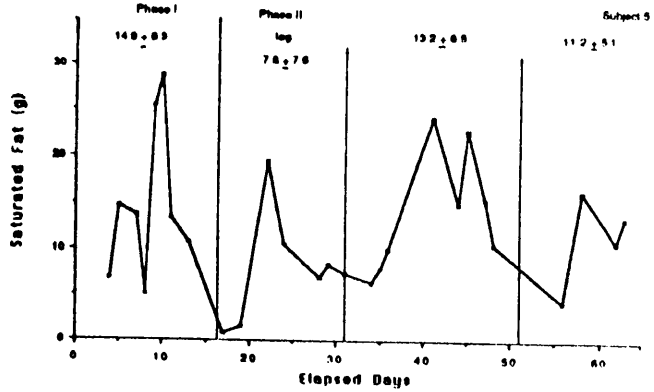

Phase I
225.7 ± 14.5
Phase II
lag
212.0 ± 8.0
Subject 5
Total Cholesterol (mg/dl)
Elapsed Days
Phase I
241.9 ± 20.6
Phase II
lag
232.0 ± 10.1
217.3 ± 14.6
212.8 ± 5.3
Subject 6
Total Cholesterol (mg/dl)
Elapsed Days
Phase I
284.7 ± 18.7
Phase II
lag
246.8 ± 16.3
234.0 ± 11.3
211.7 ± 5.1
Subject 7
Total Cholesterol (mg/dl)
Elapsed Days
Phase I
237.3 ± 19.5
Phase II
lag
238.0 ± 0.0
242.5 ± 10.5
Subject 8
Total Cholesterol (mg/dl)
Elapsed Days

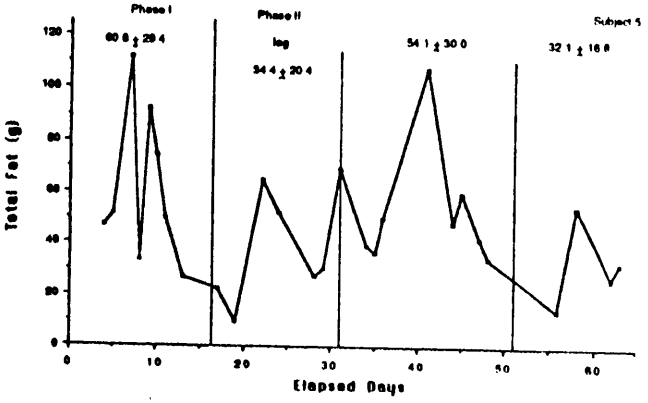
Figure 6


Phase I
Phase II
log
Subject 5
Dietary Cholesterol (mg)
Elapsed Days


Phase I
Phase II
log
Subject 5
Dietary Percentage Fat
Elapsed Days


Phase I
Phase II
log
Subject 5
Dietary Percentage Carbohydrate
Elapsed Days


Phase I
Phase II
log
Subject 5
Saturated Fat (g)
Elapsed Days


Phase I
Phase II
log
Subject 5
Total Fat (g)
Elapsed Days

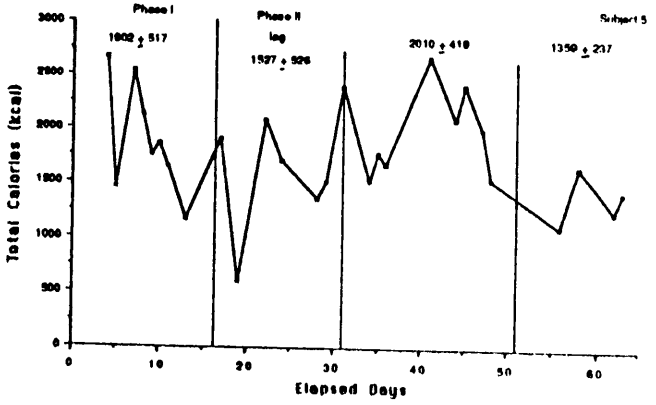

Phase I
Phase II
log
Subject 5
Total Calories (kcal)
Elapsed Days

Figure 7

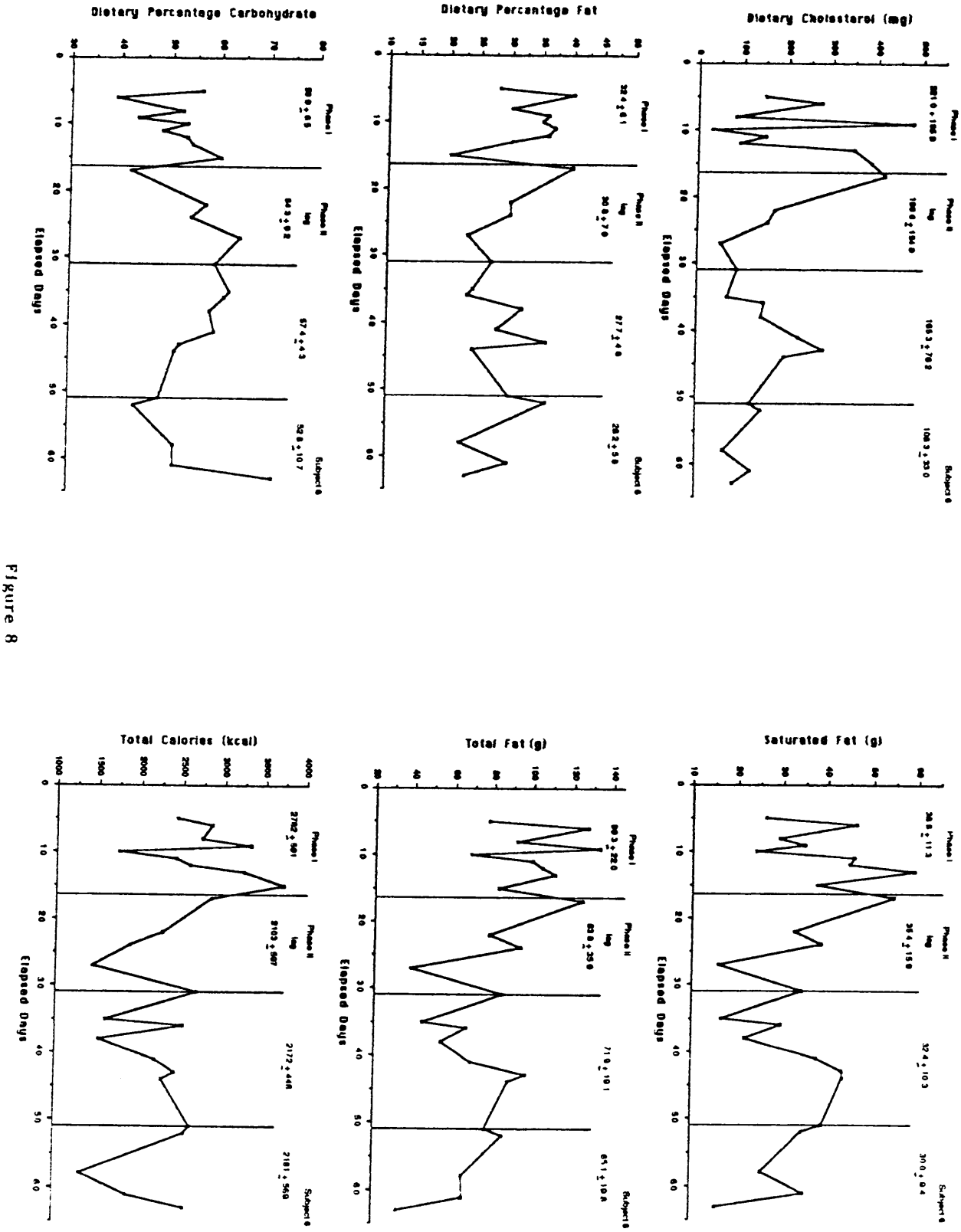
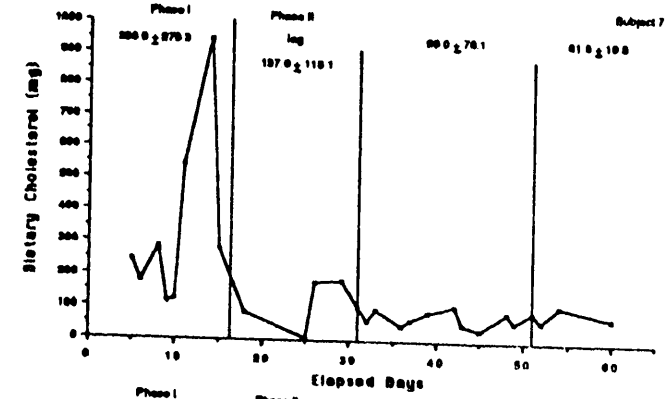
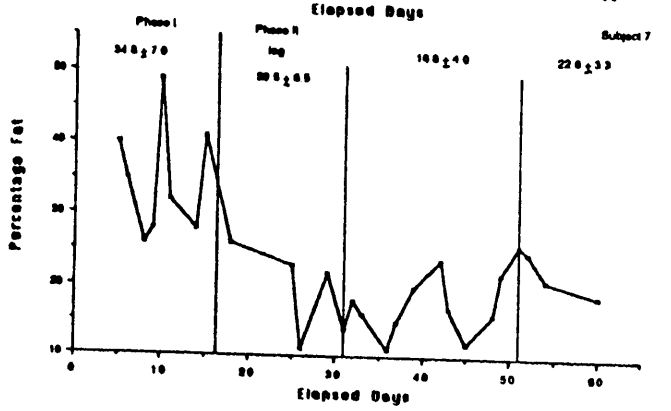
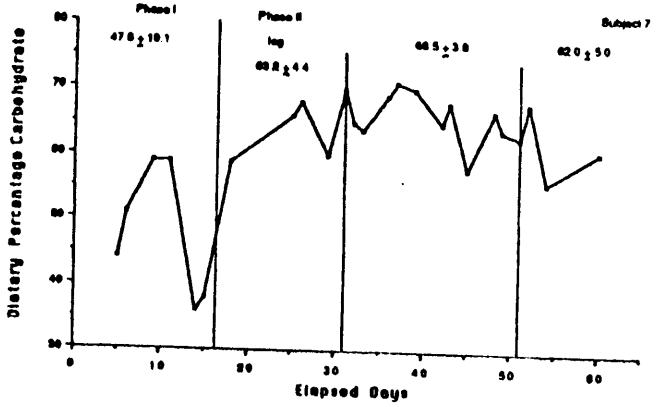
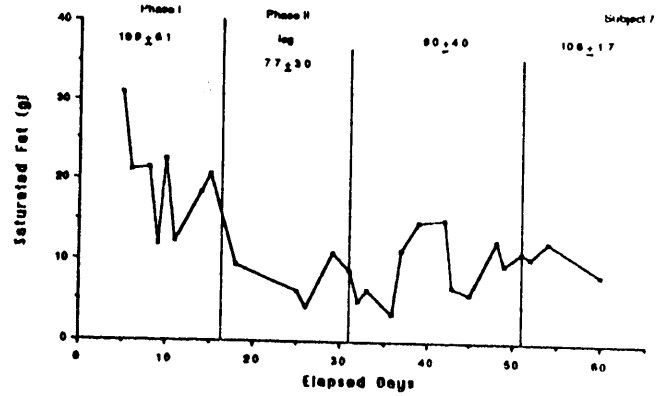

Dietary Cholesterol (mg)
Dietary Percentage Fat
Dietary Percentage Carbohydrate
Saturated Fat (g)
Total Fat (g)
Total Calories (kcal)
Elapsed Days

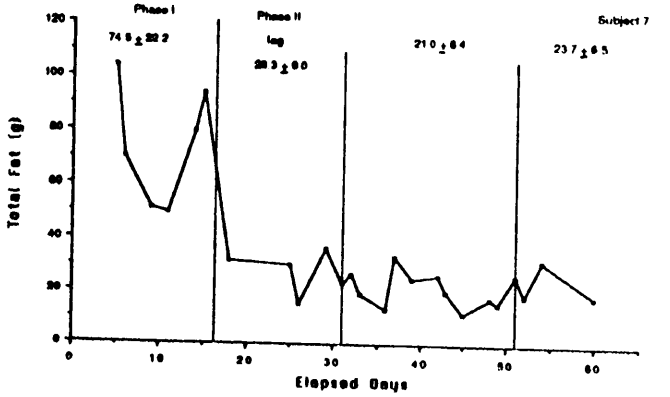
Figure 8


Phase I
Phase II
Subject 7
Dietary Cholesterol (mg)
Elapsed Days


Phase I
Phase II
Subject 7
Percentage Fat
Elapsed Days


Phase I
Phase II
Subject 7
Dietary Percentage Carbohydrate
Elapsed Days


Phase I
Phase II
Subject 7
Saturated Fat (g)
Elapsed Days


Phase I
Phase II
Subject 7
Total Fat (g)
Elapsed Days

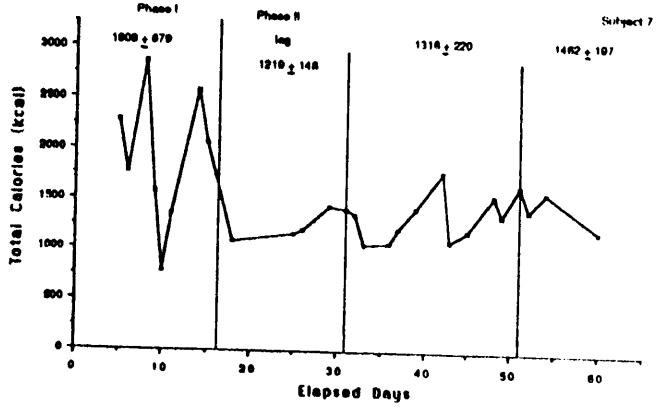

Phase I
Phase II
Subject 7
Total Calories (kcal)
Elapsed Days

Figure 9

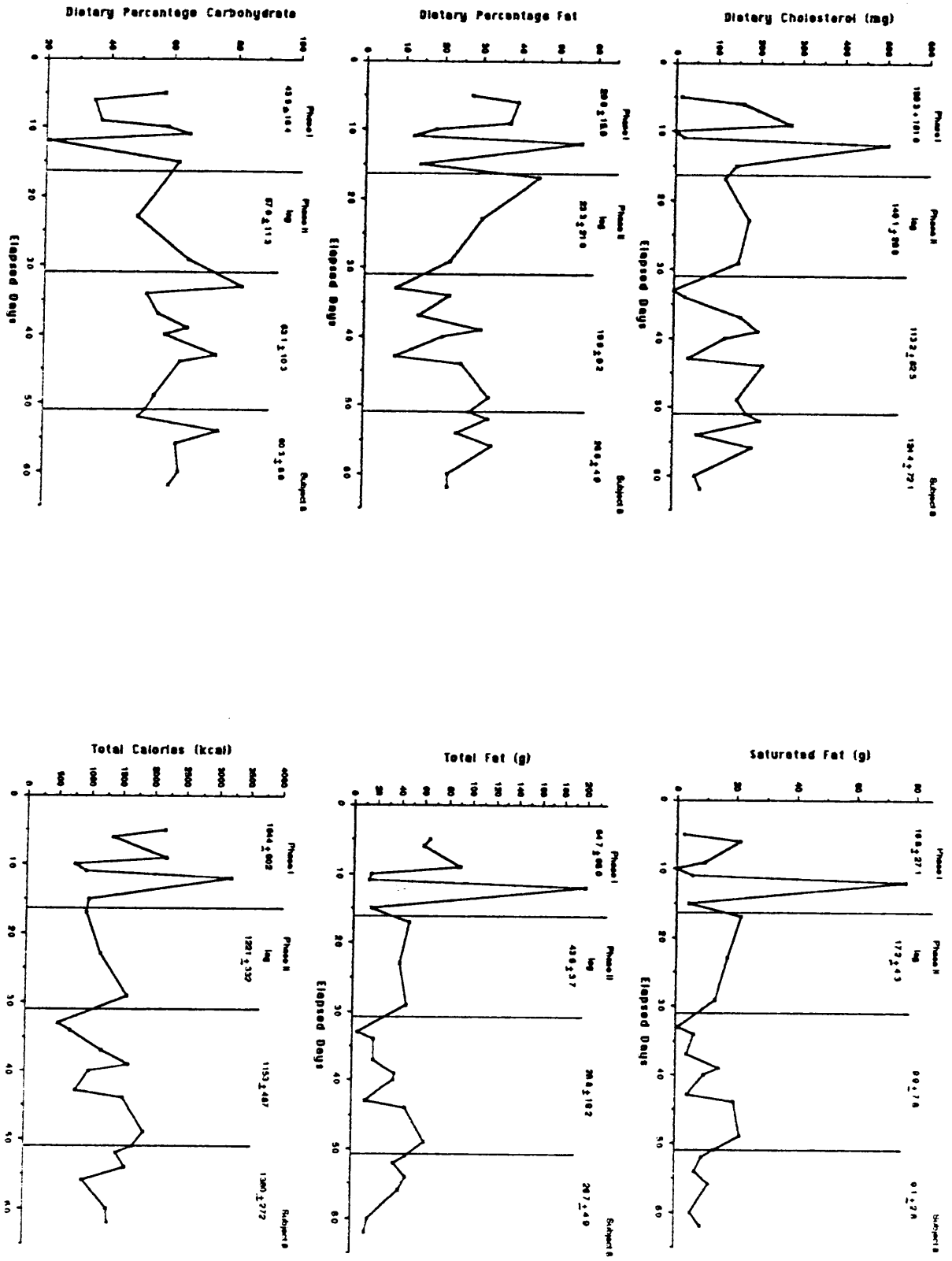

Dietary Cholesterol (mg)
Dietary Percentage Fat
Dietary Percentage Carbohydrate
Saturated Fat (g)
Total Fat (g)
Total Calories (kcal)
Elapsed Days

Figure 10

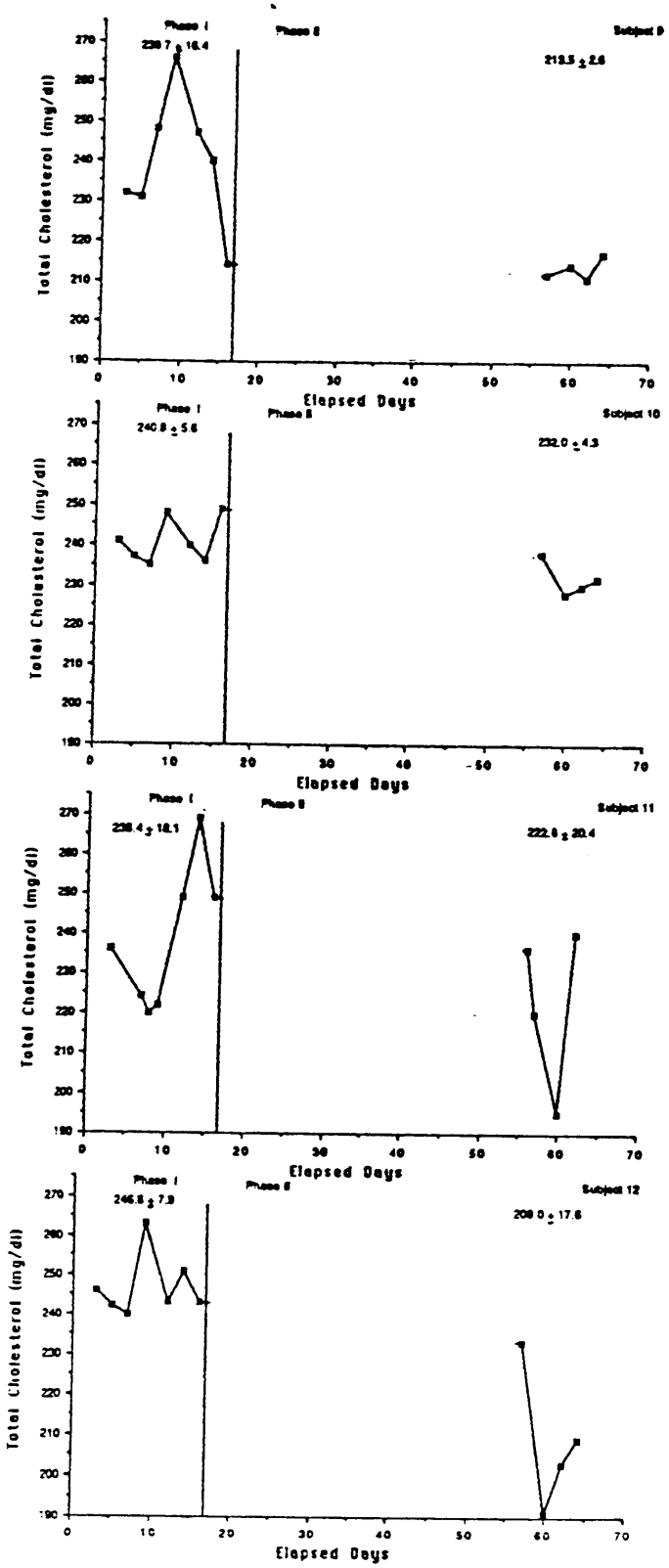

Phase I
Phase II
Subject 9
239.7 ± 16.4
213.5 ± 2.6
Total Cholesterol (mg/dl)
Elapsed Days
Phase I
Phase II
Subject 10
240.8 ± 5.8
232.0 ± 4.3
Total Cholesterol (mg/dl)
Elapsed Days
Phase I
Phase II
Subject 11
238.4 ± 18.1
222.6 ± 20.4
Total Cholesterol (mg/dl)
Elapsed Days
Phase I
Phase II
Subject 12
246.8 ± 7.9
208.0 ± 17.6
Total Cholesterol (mg/dl)
Elapsed Days

Figure 11

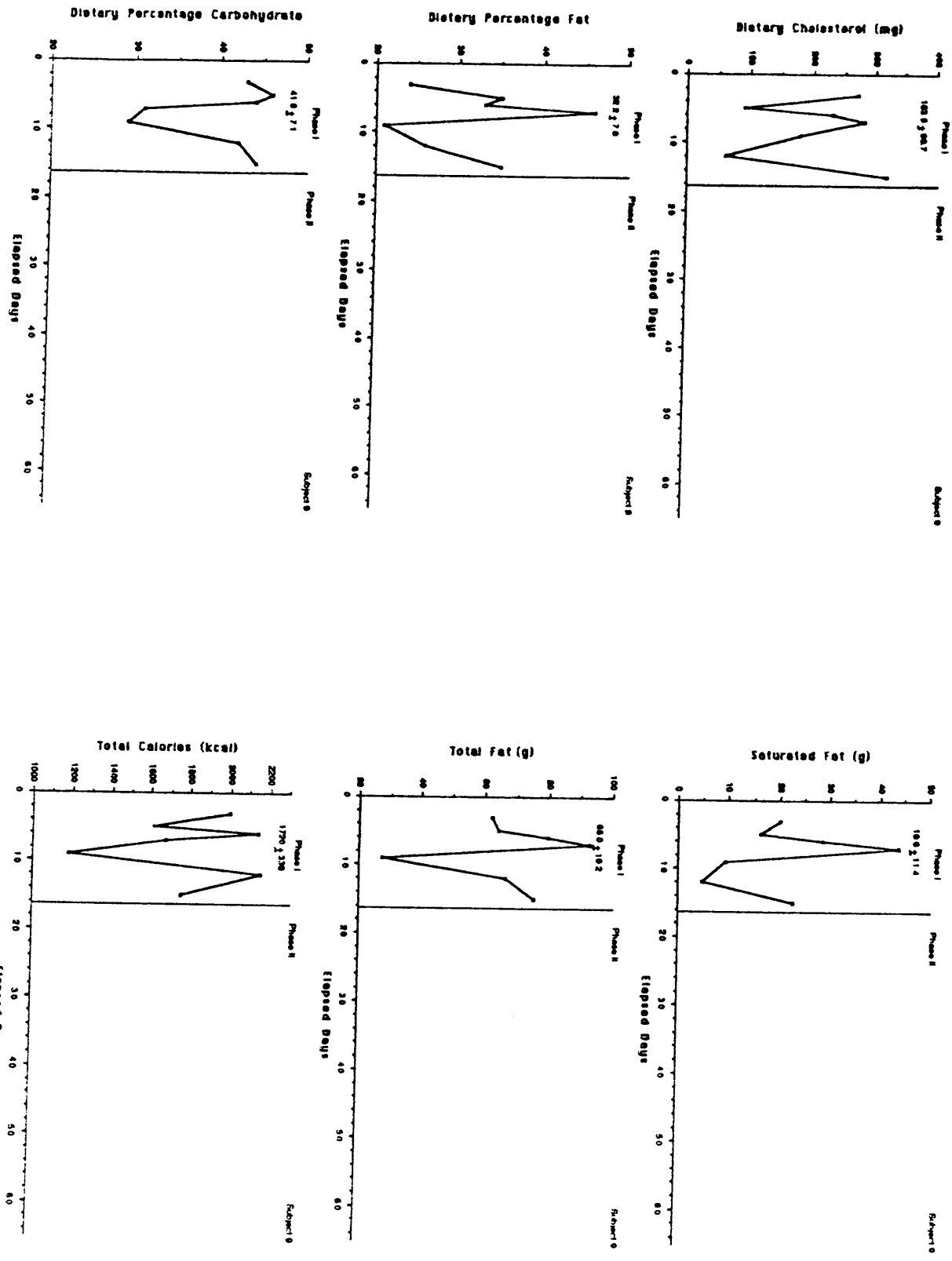

Dietary Percentage Carbohydrate
Dietary Percentage Fat
Dietary Cholesterol (mg)
Total Calories (kcal)
Total Fat (g)
Saturated Fat (g)
Elapsed Days
Phase I
Phase II
Subject 9

Figure 12

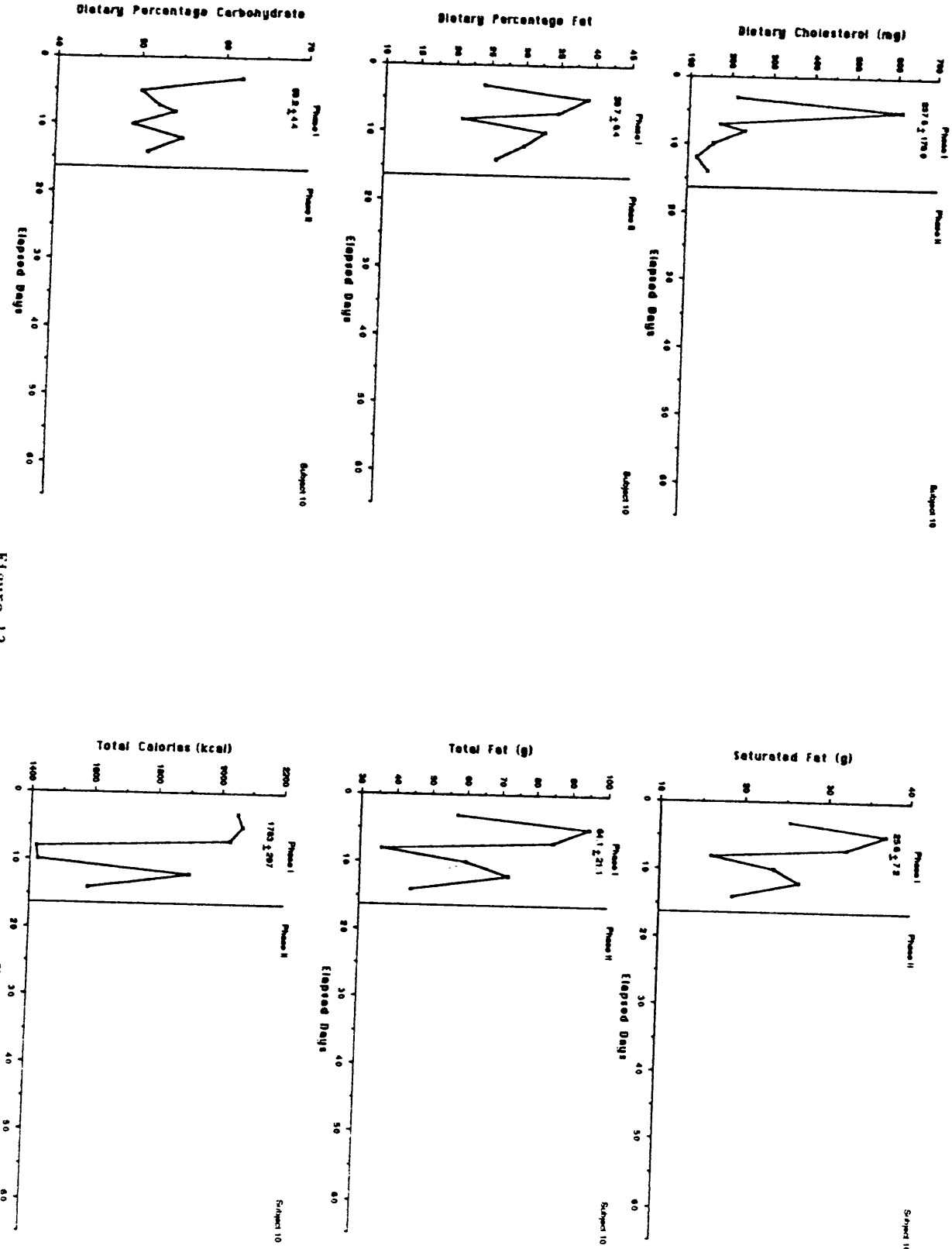
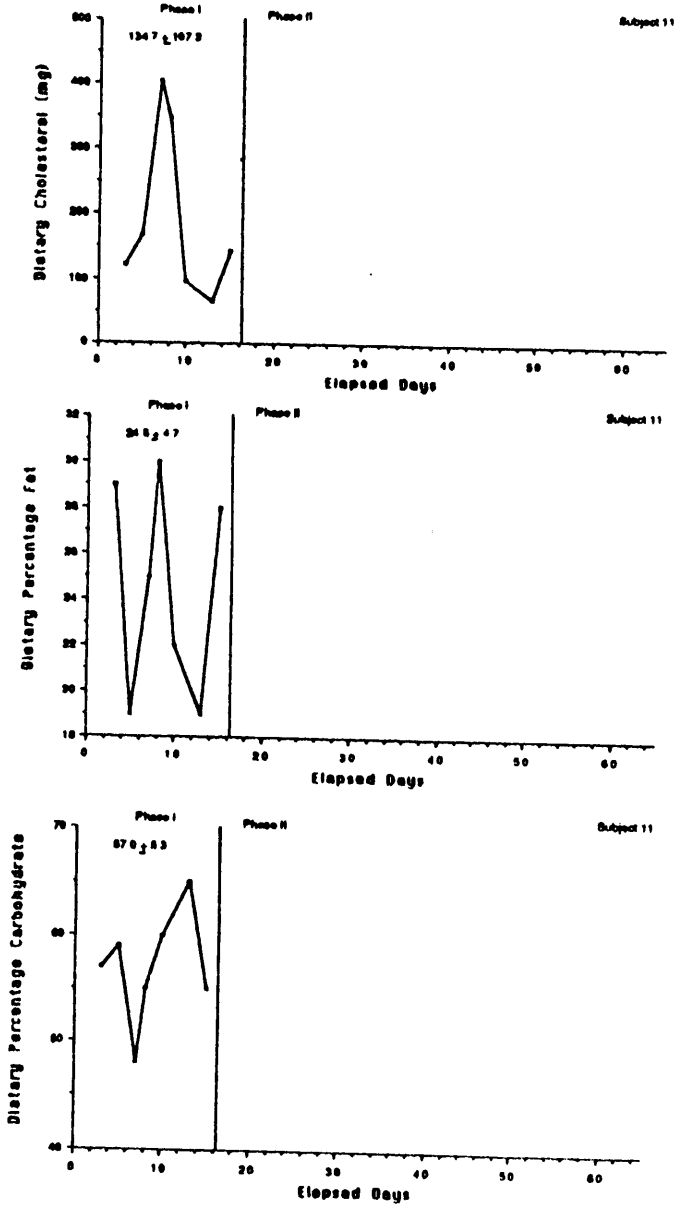

Dietary Cholesterol (mg)
Dietary Percentage Fat
Dietary Percentage Carbohydrate
Saturated Fat (g)
Total Fat (g)
Total Calories (kcal)
Elapsed Days
Phase I
Phase II
Subject 10

Figure 13


Phase I
Phase II
Subject 11
Dietary Cholesterol (mg)
Dietary Percentage Fat
Dietary Percentage Carbohydrate
Elapsed Days

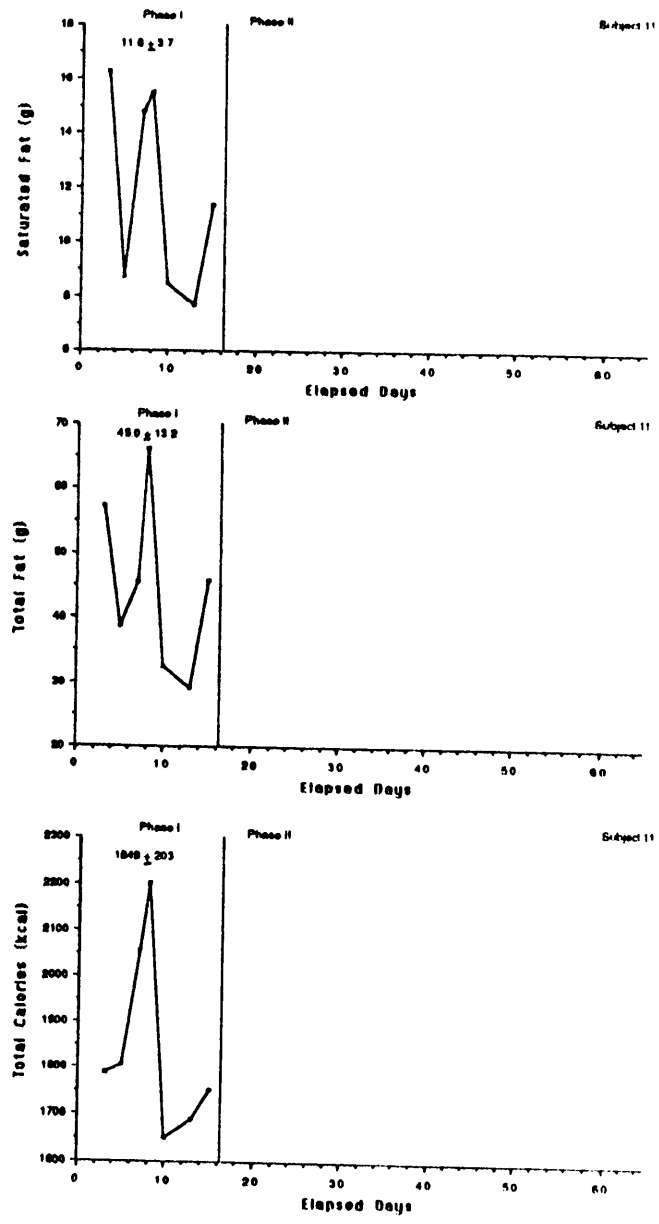
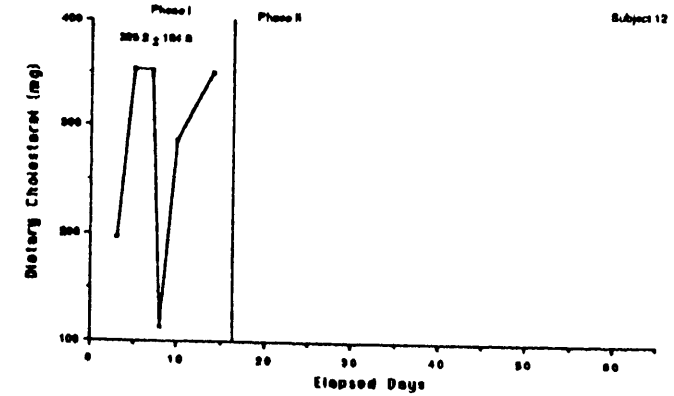
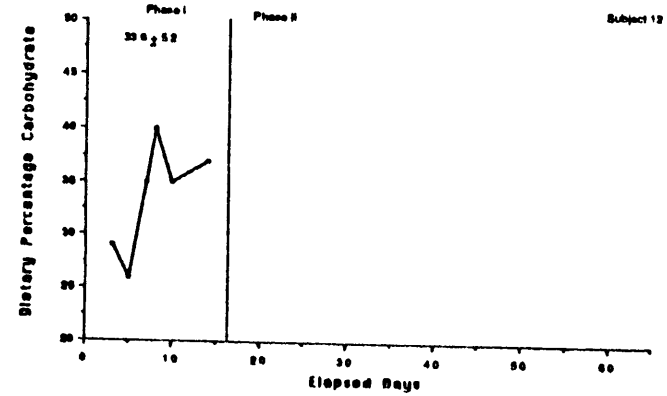
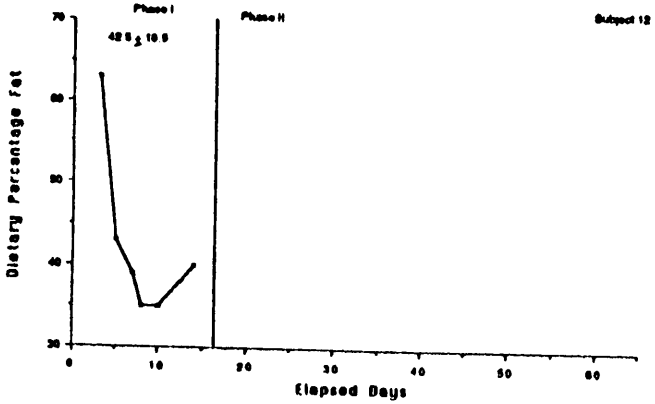
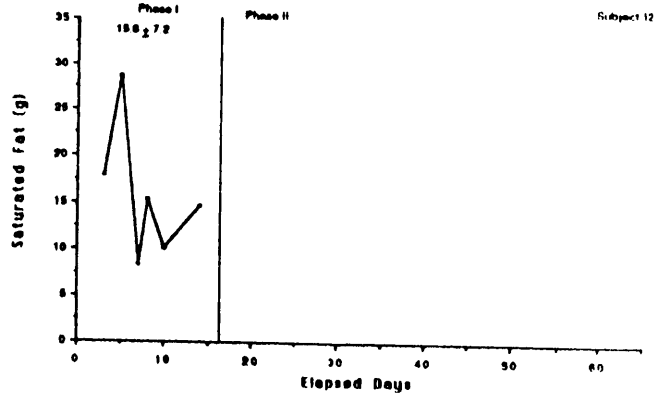

Phase I
Phase II
Subject 11
Saturated Fat (g)
Total Fat (g)
Total Calories (kcal)
Elapsed Days

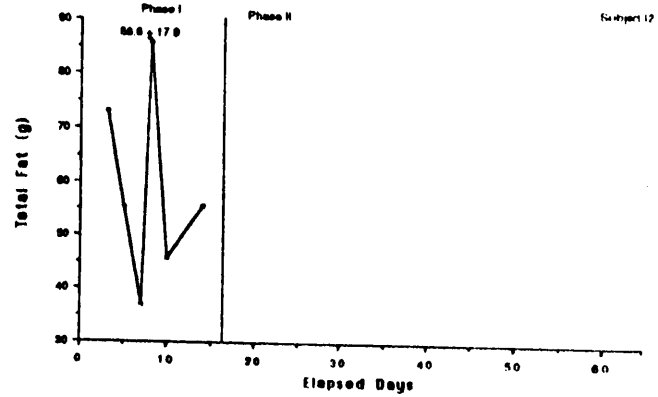
Figure 14


Phase I
Phase II
Subject 12
Dietary Cholesterol (mg)
Elapsed Days


Phase I
Phase II
Subject 12
Dietary Percentage Carbohydrate
Elapsed Days


Phase I
Phase II
Subject 12
Dietary Percentage Fat
Elapsed Days


Phase I
Phase II
Subject 12
Saturated Fat (g)
Elapsed Days


Phase I
Phase II
Subject 12
Total Fat (g)
Elapsed Days

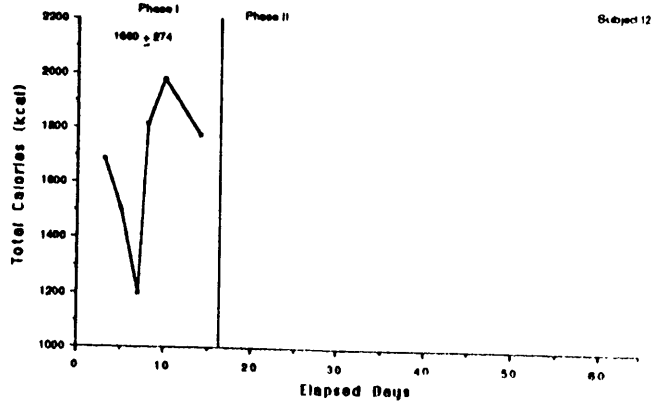

Phase I
Phase II
Subject 12
Total Calories (kcal)
Elapsed Days

Figure 15

The vita has been removed from the scanned document