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Blood concentrations of prostaglandins, insulin, and glucose in overweight college females

by

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INTRODUCTION

Extensive investigations have been conducted recently on eicosanoids, known more commonly as prostaglandins (PG). Prior to the discovery of two labile eicosanoids, prostacyclin (PGI_2) and thromboxane (TXA_2), most investigators examined PG of the E series, such as PGE_1 and PGE_2. More recently, the physiological effects of PGI_2 and TXA_2 on the vascular endothelium and platelets have been examined in relationship to diabetes and cardiovascular disease (1). The majority of these investigations, however, have been in vitro studies.

Eicosanoids are a family of compounds derived from the 20-carbon polyunsaturated fatty acids (2). They are believed to be synthesized in every tissue. Eicosanoid family members exhibit a wide variety of physiological effects. In many cases, their effects on tissues are antagonistic (3, 4). For example, PGI_2 synthesis is localized mainly in the vascular endothelium. PGI_2 inhibits platelet aggregation (similar to PGE_2) and relaxes the vascular smooth muscle (similar to PGE_2). In contrast, TXA_2 synthesis occurs primarily in the platelets. TXA_2 promotes platelet aggregation (similar to PGE_2) and constricts the smooth muscle. Platelet aggregation augments PGE_2 and PGE_1 syntheses.

Results from several investigations (5-9) imply that a delicate balance between the eicosanoid levels must be maintained in order to prevent the vascular complications associated with diabetes and cardiovascular disease. Current research results suggests that these pathological conditions are accompanied by decreased PGI_2 production and/or increased TXA_2 synthesis (5-8). Whether these changes are a

primary action or a secondary effect of these disease states are unknown. It has been suggested that high circulating glucose concentrations contribute to the eicosanoid imbalances (9). Relationships between eicosanoid production and glucose concentrations in a human population free of microvascular complications, however, have not been examined. In addition, a relationship between excessive PG production and obesity has been suggested based on in vitro studies (10). It is not known, however, whether eicosanoid concentrations are related to body weight status of humans.

Relationships between eicosanoid concentrations in body fluids and various physiological conditions in humans are of recent interest. Except for PGI_2, virtually no information is available on plasma eicosanoid concentrations. Most of the information has been obtained on serum eicosanoid concentrations, mainly TXA_2. The significance of plasma eicosanoid concentrations and their relationships to serum eicosanoid concentrations in humans is unknown.

A standardized procedure for the evaluation of eicosanoids has not been established. Recently, Sullivan and Mathias (11) have suggested that the nutritional state [i.e., collection of samples during a fasting period (fasted) or a postprandial state (fed)] affects eicosanoid concentrations during various dietary treatments in rats. Whether eicosanoid concentrations in fasted blood samples are different from that in fed samples in humans has not been verified.

The main objective of the present study was to examine eicosanoid, insulin, and glucose concentrations in a group of overweight and obese

young women. These females volunteered to participate in a Body Weight Management Program which was introduced into the Iowa State University (ISU) community by the present investigator. This program from September, 1982 to April, 1983 emphasized a nutritionally sensible lifestyle to lose body weight and maintain weight loss. During the program, the participants continued to live in the university residence halls; they ate their meals in their dining halls. The developmental procedures and the evaluation of the Body Weight Management Program are presented in the Appendixes. Financial support for this investigation was provided by the ISU Agriculture and Home Economics Experiment Station, Project No. 2567, entitled Metabolic Characteristics and Body Composition of Women.

LITERATURE REVIEW

Eicosanoid Metabolism

During the last 25 years, no group of compounds has captured as much attention from the scientific community as the eicosanoid family. The 1982 Nobel Prize was awarded to Sune Bergstrom and Bengt Samuelsson from Sweden and John Vane from Great Britain for their contributions to eicosanoid research (12). Since their discovery, these labile members of the arachidonic acid cascade have been studied in relation to a wide variety of clinical situations (3). Most recently, eicosanoids have been linked to the developmental and clinical expressions of atherosclerosis and diabetes mellitus.

Enzymatic oxidation of arachidonic acid leads to the group of labile compounds known collectively as eicosanoids (13). The term eicosanoid applies to the 20-carbon polyunsaturated fatty acid derivatives (Figure 1). This multifunctional family includes prostaglandins (PG) such as PGE_1 and PGE_2, prostacyclin (PGI_2), thromboxane-A2 (TXA_2), and leukotrienes. The term prostaglandin is used traditionally to refer to all eicosanoids but it actually applies only to those compounds with a prostanoic acid skeleton such as PGE_1 and PGE_2 (2). This review focuses on specific eicosanoids formed by the cyclooxygenase pathway: PG, PGI_2, and TXA_2. The products of the lipoxygenase pathway, the leukotrienes, are excluded from this review.

The typical PG molecule consists of the prostanoid acid skeleton, a five membered ring structure with two side chains (Figure 1). Specific

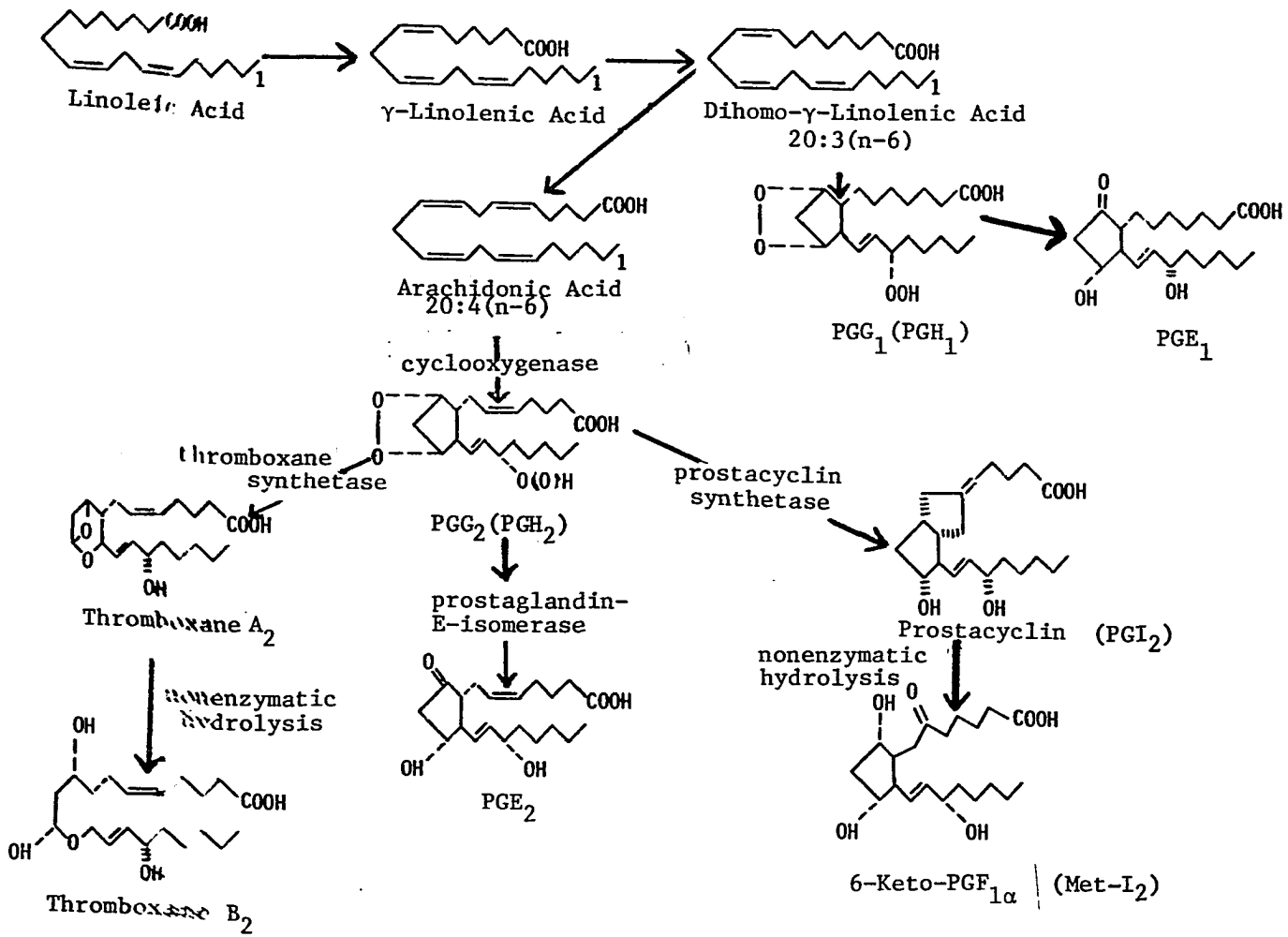


FIGURE 1. Desaturation and elongation of linoleic acid and the subsequent cyclooxygenase pathway

chemical substitutions on the cyclopentane ring are indicated by letter designations such as E for PGE. The number of double bonds on the PG molecule side chains is denoted by subscripts, such as 1 and 2.

Many excellent reviews on eicosanoid metabolism have been published during the last few years (1, 2, 14). A review of eicosanoid metabolism is essential before integrating their functions into physiological situations. In contrast to the conflicting reports of the physiological effects of the eicosanoid family members, the metabolism is quite well-defined.

Eicosanoids are extremely potent, ubiquitous compounds synthesized from 20-carbon polyunsaturated fatty acids (2). The essential fatty acid, linoleic acid, is the physiological precursor of the "1" and "2" series of eicosanoids. The pathway by which linoleic acid is converted to arachidonic acid (eicosatetraenoic acid) is illustrated in Figure 1. Linoleic acid is desaturated and elongated to form dihomo-gamma-linolenic acid. The latter fatty acid is desaturated to form arachidonic acid. In general, arachidonic acid yields compounds with subscript "2" as in PGE_2. Prostaglandins of the "1" series as in PGE_1 are synthesized from dihomo-gamma-linolenic acid.

Once absorbed, arachidonic acid is transported in the blood in several ways: bound to albumin, chylomicrons, and lipoproteins. Arachidonic acid is incorporated into the subcellular structures of tissues. In general, membrane phospholipids are the richest source of arachidonic acid. Arachidonic acid is enzymatically released by phospholipase A activity from the beta-position of the phospholipids in

the membrane upon mechanical, hormonal, and other stimulation. About 20% of this arachidonic acid is available for eicosanoid metabolism via two pathways with the remainder being reacylated to phospholipids (3, 5).

One pathway of eicosanoid biosynthesis through lipoxygenase activity results in the formation of leukotrienes. These compounds are believed to have immunologic properties but the full importance of these substances is still not known (1, 15). A second pathway by cyclooxygenase activity leads to the formation of PG, PGI_2, and TXA_2 as illustrated in Figure 1. These labile compounds are involved in the functions of the cardiovascular system (1) and platelets (2) and of lipid metabolism (14).

Two steps are required for the formation of PG, PGI_2, and TXA_2 following the release of arachidonic acid from tissue stores (Figure 1). The first step results in the formation of the cyclic endoperoxides, PGG and PGH. These cyclic endoperoxides are rapidly formed from arachidonic acid by cyclooxygenase in the presence of molecular oxygen. In the second step, these unstable and biologically active endoperoxides are converted to one or more compounds of the eicosanoid family by tissue-specific synthetases or isomerases.

Tissue-specific synthetases are involved in the formation of TXA_2 and PGI_2 (Figure 1). Thromboxane synthetase, localized mainly in the platelets, catalyses the conversion of the endoperoxide to TXA_2. This extremely labile eicosanoid has a half-life of about 30 seconds and is converted nonenzymatically to an inactive metabolite, thromboxane B2

(TXB_2). Localized in the vascular endothelium, prostacyclin synthetase activity results in the formation of PGI_2 which has a biological half-life of about 2 minutes. The most important physiological metabolite of PGI_2 is the biologically inactive 6-keto-$PGF_{1\alpha}$ (Met-I_2) (3).

Tissue-specific isomerases are responsible for the formation of PG (Figure 1). Endoperoxide-E-isomerase activity on the arachidonic acid-derived endoperoxides results in the formation of PGE_2. PGE_2 is efficiently inactivated (90% in one pass) in the lungs. In contrast to PGE_2, PGE_1 is synthesized from dihomo-gamma-linolenic acid. It requires also the cyclooxygenase and isomerase activities. Similar to PGE_2, circulating concentrations of PGE_1 are low due to efficient inactivated by in the lung (3).

In virtually every mammalian tissue, arachidonic acid is metabolized as illustrated in Figure 1. As implied earlier, one particular eicosanoid is made in greatest abundance by a specific cell type. For example, endothelial cells can produce PGE_2 and PGI_2 but PGI_2 production accounts for at least 90% of the total production. On the other hand, TXA_2 is the most potent and quantitatively important metabolite formed in platelets (2).

Although the eicosanoid family members are derived from the same precursor, they exhibit diverse and in many cases, opposing physiological effects (Table 1) (1, 3, 4). For example, circulating endogenous TXA_2 constricts arteries and promotes platelet aggregation. In contrast, PGI_2 relaxes vascular smooth muscle and inhibits platelet aggregation. PGE_2 relaxes vascular smooth muscle (similar to PGI_2) and

promotes platelet aggregation (analogous to TXA_2). PGE_1 is similar to PGI_2 in its anti-aggregatory nature.

It is generally considered that eicosanoids should be classified as intracellular messengers rather than as hormones (1, 3, 15). If a hormone is defined as a substance that is produced and stored in a gland for subsequent release into the circulation to act upon a distant target site, eicosanoids certainly do not qualify. There is no overwhelming evidence to indicate that they are stored as such, or that they respond to a physiological signal to act at a distant target site. They appear to behave analogously to cyclic adenosine monophosphate (cAMP). In this respect, they are synthesized rapidly within a given tissue and serve to modulate physiological processes locally.

Many compounds can interfere with eicosanoid synthesis (2, 13). The effect of aspirin (acetylsalicylic acid) is probably the most widely known. Aspirin irreversibly and chemically inactivates cyclooxygenase. Biosynthetic activity is restored only when new enzyme protein is synthesized by the tissue. Hence, a dose of aspirin would have a longer inhibitory effect on platelet synthesis of escosanoids than on the endothelial production. Platelets do not possess the ability to synthesize protein, and eicosanoid activity returns only after new platelets have been formed.

Plasma and Serum Eicosanoid Levels

Conflicting results have been obtained in all facets of eicosanoid research. A major factor promoting this confusion stems in part from

TABLE 1. Effect of eicosanoids on vascular smooth muscle and platelet aggregation

Eicosanoid	Vascular Smooth Muscle	Platelet Aggregation
Prostaglandin E_1 (PGE_1)	unknown	inhibits
Prostacyclin (PGI_2)	dilates	inhibits
Prostaglandin E_2 (PGE_2)	dilates	promotes
Thromboxane A_2 (TXA_2)	constricts	promotes

the investigative models. Eicosanoid research has been conducted on a full gamut of research models ranging from cell biology to human investigations usually in conjunction with a disease state. In vitro studies have used different experimental approaches during the investigations. For example, PGI_2 synthesis is evaluated by using aortic strips or rings in various media. In vivo studies have been conducted by observing a physiological response during the infusion of PG or during the inhibition of eicosanoids by aspirin. The diversity in experimental approaches used makes it difficult to extrapolate the results from these investigations and apply them to a normal population group.

There is growing interest in the significance of eicosanoid concentrations in body fluids during various clinical conditions in humans. This interest has been generated by the recognition that endogenous eicosanoids modulate the functional activities in many tissues. Most frequently, eicosanoid concentrations in serum, and to a lesser extent in plasma are being determined. But reports of eicosanoid values from blood vary widely (16), and comparison among the various studies becomes impossible. Thus, normal ranges of eicosanoid concentrations in serum and plasma are ill-defined.

Many factors affect eicosanoid concentrations in blood and contribute to the disparity in reported values. Methodology is critical in eicosanoid research. The collection and processing of samples for eicosanoid analysis are not standardized. Variables such as temperature and duration of incubation, and centrifugation temperature influence

eicosanoid concentrations (16). In addition, the use of a cuff during the blood collection period may elevate eicosanoid concentrations (11). Second, samples must be processed immediately after blood is drawn (16). Samples from clinical studies may not be processed immediately due to inadequate facilities and insufficient personnel. Third, eicosanoid concentrations have been analyzed by gas chromatography, mass spectrometry, or radioimmunoassay (16, 17). The results from these techniques may not correlate well in samples from the same subject at a given time. Finally, the time blood is drawn in relation to the last meal affects eicosanoid concentrations in blood (11). Other factors such as exercise level (18), dietary intake (1, 5), and disease (1, 5) influence blood eicosanoid concentrations.

Plasma eicosanoid concentrations theoretically reflect eicosanoid metabolism in the blood vessels. McGiff (3) indicates that plasma Met-I_2 concentrations reflect PGI_2 synthesis by the vascular endothelium. Since PGE_1 and PGE_2 are efficiently inactivated by pulmonary circulation, concentrations of these eicosanoids may represent regional vascular synthesis in the area of the blood withdrawal. Storms (19) found significant correlations (r=0.6) among plasma PGE_1, plasma PGE_2, and PGI_2 in four insulin-dependent diabetics. These observations may suggest a similar origin, the vascular endothelium, for these eicosanoids. Presumably, plasma TXB_2 concentrations indicate the amount of platelets synthesis during aggregation (4).

Serum eicosanoid measurements are analogous to performing an aggregating platelet incubation (4). This ex vivo measurement provides

a physiologic biopsy technique for evaluating the rate and total eicosanoid synthesis during clotting (16, 20, 21). As mentioned earlier, TXA_2 is quantitatively the most important serum eicosanoid. PGE_2 does not initiate platelet aggregation, but it is released during the aggregation process.

Correlations between plasma and serum eicosanoids have not been examined adequately. The Department of Food and Nutrition at Iowa State University is one of the few institutions that is currently investigating this relationship. Some studies have been conducted at by the Department of Food Science and Nutrition in Colorado State University in Fort Collins, Colorado (16, 21). The significance of endogenous eicosanoid concentrations in blood warrants verification.

Factors that Influence Eicosanoid Production

Cardiovascular disease, diabetes mellitus, and aging

One area that has received considerable attention is the role of PGI_2 and TXA_2 in cardiovascular disease and diabetes. Cardiovascular disease and diabetes are in themselves related. For example, cardiovascular disease is an important risk factor for diabetes. A 16-year follow-up study of the Framingham population (22) indicated that the mortality rate in diabetic subjects with cardiovascular disease was about three times higher than the rate in the general population. Recent research findings suggest that these disease states are accompanied by an imbalance in PGI_2 and TXA_2 production (5, 7).

Endothelial cells line the inner wall of the blood vessel. The

study of these cells is under intense investigation because they are the first tissues injured during the development of cardiovascular disease (1, 14). It has been demonstrated that PGI_2 production by the endothelial cells is decreased during the initial developmental stages of atherosclerosis (23). When PGI_2 production is insufficient, platelets attach to the cell which in turn favor TXA_2 production (2). It is apparent that an optimum balance between PGI_2 and TXA_2 must be maintained to prevent damage to the endothelium.

It has been suggested that an imbalance in eicosanoids, mainly PGI_2 and TXA_2, promotes the development of the microvascular changes observed in diabetics (8). In a diabetic condition, PGI_2 production by the endothelial cells is lower compared to the synthesis in cells from an age-matched control population (7, 24). In addition, platelets aggregate more readily and TXA_2 synthesis is greater in diabetics than in age-matched control subjects (6).

Plasma levels of Met-I_2 which reflect PGI_2 synthesis have been studied in diabetics and age-matched control groups. In general, Met-I_2 concentrations are lower in the diabetics than in the nondiabetic control populations (25, 26). Some studies, however, report no differences in the Met-I_2 concentrations between these two groups (27, 28). The lack of consistency may be due to selection of subjects and blood sampling protocol used.

The reasons offered for the PGI_2/TXA_2 imbalance in cardiovascular disease and diabetes remain speculative. Several investigators have suggested that high circulating glucose concentrations lead to the

eicosanoid imbalance (6, 9, 29, 30). Many studies, however, give conflicting results on the relationship between glucose and eicosanoid concentrations (21, 31, 32). Recently, Lasche and Larson (33) reported that hyperinsulinemia, a risk factor for atherosclerosis development (34, 35), decreases PGI_2 production by the vascular endothelium in the rat. In contrast, treatment of the aorta with insulin significantly increased PGI_2 production in diabetic and nondiabetic rats (7, 24). Other factors such as hypercholesterolemia (36, 37) and lipoprotein composition (5, 38, 39) may influence the balance between PGI_2 and TXA_2.

Some research data suggest that the PGI_2/TXA_2 imbalance may be part of the normal process of aging. Ylikorkala et al. (40) reported the effect of age on circulating PG concentrations in a normal human population. In general, Met-I_2 concentrations were similar for males and females of the same age. Plasma Met-I_2 concentrations were higher in the adolescent (10-20 years of age) than in healthy adults (30-50 years of age). Chang and Tai (41) observed that PGI_2 production by rat aortas decreased with age. Although TXA_2 production in platelets did not change with age in the latter study, the reduced PGI_2 production would decrease the PGI_2/TXA_2 ratio.

Dietary lipid intake

The consumption of polyunsaturated fatty acids affects platelet aggregation. Hornstra et al. (42) found decreased platelet aggregation following ingestion of a diet consisting of 35% of the calories as fat with 12% linoleic acid compared to 4% as linoleic acid. Fleischman et al. (43) and O'Brien et al. (44) have reported similar results of the

beneficial effect of increased dietary linoleate upon platelet function in humans.

The average consumption of linoleate in the United States is about 2 to 5% of the total calories (45). It has been recommended that intakes be increased two to three times this range (46). Results from investigations of the effect of dietary linoleate on eicosanoid production, however, are conflicting (11, 47, 48, 49) and difficult to interpret (Table 2). Differences in experimental design used in these studies may account for some of the diverse results. Thus, the effect of dietary linoleate treatment on eicosanoid production is not predictable. Data by Dupont et al. (49) indicate an increased rate of eicosanoid production with dietary linoleate above 20%; this suggests that excessive dietary linoleate may be detrimental.

Nutritional state and eicosanoid synthesis

Recently, it has been suggested that the nutritional state may affect eicosanoid synthesis. In this context, nutritional state of the experimental animal refers to the collection of blood samples for eicosanoid analysis in relation to the last meal: either under fasting (fasted) or postprandial (fed) conditions. Only a few studies have addressed specifically the effects of the nutritional state on eicosanoid production.

Sullivan and Mathias (11) conducted one of the few thorough investigations that examines the effect of nutritional state on escosanoid production. They found that the synthesis of serum TXB_2 and PGE_2 differed significantly between fasted and fed blood samples. Serum

TABLE 2. Effect of dietary linoleate treatment on eicosanoid production in the rat.

Reference	Sex & Duration	Blood Collection	Level & type of Dietary Fat[a]	Effect
Sullivan & Mathias (11)	Female 13 weeks	Fasted & Fed	No fat (0.0% linoleate) BT (0.4% linoleate) BT:CO (14% linoleate) CO (22% linoleate) SF (29% linoleate)	Serum PGE_1, PGE_2, & TXB_2 depressed at 0% linoleate; Syntheses greatest at 0.4% linoleate.
Hwang et al. (47)	Male 2 months	Fasted & Fed	40% total energy 1) BT (3.2% linoleate) 2) HVF (29% linoleate) 3) CO (63% linoleate)	Serum PGE_1 higher at 63% linoleate.
Fine et al. (48)	Male 8 & 11 weeks	Fasted	20 or 40% of total energy P/S ratios (SBO:BT): 1) 0.4 2) 0.8 3) 5.5	Serum PGE_2 not affected; serum PGE_1 higher with 20% than 40% fat calories.
Dupont et al. (49)	Female 6 months	Fed	0-27% linoleate calories 1) BT 2) SBO 3) SFO	Serum PGE_1 & PGE_2 increased at 0-2% kcal, increased at 2-7% kcal, increased at 7-27% kcal. Serum TXB_2 decreased at 2-20% kcal, increased at 27% kcal.

[a]Abbreviations for dietary fat are: BT=beef tallow; BT:CO=beff tallow, corn oil; CO=corn oil; SF=safflower oil; HVF=dhyrogenated vegetable fat; SBO=soybean oil; and SFO=sunflower oil.

TXB_2 and PGE_2 concentrations in fasted samples were 62 and 48% higher, respectively, than were eicosanoids values in fed samples. The authors explained these findings on the basis of an increased norepinephrine activity associated with fasting. In this respect, norepinephrine elevates plasma free fatty acid concentrations which in turn induce platelet aggregation. In theory, this physiological response would stimulate platelet eicosanoid synthesis. Furthermore, they suggest that increased eicosanoid production during fasting is due to complex interactions among several factors: norepinephrine, platelet lipid turnover, and plasma free fatty acid composition. In addition to the well-known phospholipid precursor pool, they suggest that plasma free fatty acids may be also an important eicosanoid precursor pool.

Sullivan and Mathias (11) reported another interesting finding in the same study. They examined concurrently the effects of dietary linoleate treatment on eicosanoid production in fasting and fed serum samples (Table 2). The effect of dietary linoleate was observed only in the fed samples. They reported that serum TXB_2 and PGE_2 synthesis were depressed on a fat-free diet in the fed samples. Synthesis of these eicosanoids was highest during a 0.4% linoleate calories treatment (beef tallow). Moreover, serum TXB_2 was depressed as the concentration of dietary linoleate increased from 14 to 29% linoleate calories. None of these changes were observed in the fasted serum samples.

Only two human studies have examined the production of eicosanoids during different nutritional states. Storms (19) examined this relationship in four insulin-dependent diabetic patients. She found

that plasma PGE_1 and PGE_2 concentrations were higher in the 12-hour fasting samples than the 2-hour or 4-hour postprandial samples. The nutritional state of her subjects, however, did not affect serum eicosanoid production. On the other hand, Greaves et al. (18) found PGE_1 concentrations in whole blood were higher in the 1-hour postprandial samples than in the 12-hour fasted samples in human subjects.

In humans, some evidence indicate that a postprandial state may stimulate eicosanoid synthesis, particularly serum TXA_2 and PGE_2. The majority of the available data is based on studies that examine platelet activation, platelet adhesiveness, and platelet survival. In general, human platelets are activated by lipids, mainly saturated fats (50); this stimulation leads to increased platelet adhesiveness, increased platelet clumping, and decreased platelet survival. In theory, these phenomena would imply enhanced platelet eicosanoid synthesis. Results from human studies support these concepts. Lowe et al. (51) studied the effect of a saturated fat meal on the induction of circulating platelet-aggregates in healthy volunteers. They found increased platelet aggregates and reduced platelet-counts 90 minutes after the meal. O'Brien et al. (44) also observed an increase in platelet adhesiveness and a decrease in platelet count following a polyunsaturated meal. Moolten et al. (52) found increased platelet adhesiveness in human subjects fed either low fat or high fat diets. The rise in adhesiveness, however, was significantly greater for the group receiving the high fat diet than for the low fat group. These findings suggest

that activation of platelets is associated with the fed condition. In turn, synthesis of serum eicosanoids, predominantly TXA_2, would be stimulated. Platelet production of PGE_2, and to a lesser degree PGE_1, would also occur.

Prostaglandins and Obesity

It is well-documented that PG play an important physiological role in the regulation of lipid mobilization from adipose tissue (53, 54). Steinberg et al. (55) and Bergstrom and Carlson (56) were the first to report that PGE_1 reduced basal and hormone-stimulated lipolysis in adipose tissue in vitro. It is generally accepted that eicosanoids, mainly PGE_1 and PGE_2, are involved in the maintenance of adipose tissue homeostasis by a negative feedback inhibition mechanism. In the normal adipose cell, cAMP formation (inducing lipolysis) and PG synthesis are in a finely balanced state. It has been hypothesized from in vitro studies that in obesity, PG synthesis is excessive causing a negative inhibition of the cAMP system (reducing lipolysis) at one or more steps. Hence, the mobilization of the triglyceride from adipose tissue is "tuned-off" and little, if any release of free fatty acids and glycerol is observed (10).

The PG hypothesis of obesity is attractive but lacks supporting evidence. This view might imply that the inhibition of PG synthesis by aspirin and other non-steroidal anti-inflammatory drugs would be useful in the treatment of obesity. Breddin et al. (57) reported that significant weight loss did not occur in humans receiving doses of

aspirin at least five times higher than that required for complete inhibition of platelet PG. A daily dose of 1800 mg of aspirin for three days will completely inhibit eicosanoid production.

Insulin and Glucose Levels in Relation to Body Weight Status

A positive relationship exists between obesity and either fasting or glucose-stimulated insulin levels (58). In this respect, increasing relative body weights are associated with increments in basal insulin secretion in normal subjects. A study result by Kaplan and Leveille (59) strengthens this concept. Fasting and postprandial blood samples of obese females had significantly more insulin and glucose than samples of normal weight females.

Recent reports showed that insulin and glucose levels are closely associated with the pattern of body fat distribution (60). Individuals with an upper-body-obesity (fat is distributed mostly above the waist) are more likely to develop diabetes than those with a lower-body-obesity (fat is predominantly on the hips and thighs). This theory is based on the insulin and glucose concentrations of women observed by Kissebah and associates (61, 62). They found that elevated insulin and glucose levels are more commonly associated with an upper-body-obesity compared to a lower-body-obesity.

Weight Reduction and Maintenance of Weight Loss

Although eicosanoid research has captured the attention of the scientific community in recent years, the preoccupation with body weight

continues to elicit universal interest and personal concern. Centuries ago, an overweight state was considered a status symbol and an object of artistic representation. But times have changed especially with the advent of Twiggy; a social stigma has become attached to obesity. Ayers (63) has presented the changing views toward overweight and obesity from the Greco-Roman times to the twentieth century. These are the esthetic views on body shape and form; health aspects are often overlooked.

The Health and Nutritional Examination Survey (HANES) data (64) indicate that a large percentage of Americans exceed the recommended weights. Approximately, one-fifth (1.7 million) of females, 20-24 years of age, are 10% or more above the suggested weights for their heights. For females in the same age group, about one-tenth are 20% or more above the recommended weights. A greater percent of women than men in the same age groups have body weight above the suggested ranges. The percent deviations from the recommended weights for either sex increase with age.

Weight reduction programs continue to proliferate into the public sector; they are lucrative business enterprises. When a new program is introduced, clientele invest hundreds and thousands of dollars in hopes that finally a "miracle" program has been discovered. Some of these "miracle" diets, such as the liquid-protein-diet, have proven lethal to some poorly-counseled patrons (65). Moreover, the dietary recommendations of these faddish programs do not produce favorable lean/fat (body compartments) ratio (66). The most undesirable outcome is that weight losses achieved are seldom maintained permanently

(67-69).

The basic goals of a sensible weight reduction program are (1) to decrease caloric intake and (2) to increase caloric expenditure (67). Both of these objectives should be met in a moderate fashion and adequate nutrient intakes must be assured. A caloric intake below 1200 kcal/day is not recommended; below this level, the Recommended Dietary Allowances (45) for essential nutrients are difficult to meet. Additionally, a daily physical exercise pattern needs to be adopted, although it need not be a strenuous regimen.

The cyclic loss and regain of body weight has been popularly termed the 'yo-yo syndrome' (70). Bray (67) has evaluated the body weight patterns of obese individuals from long-term studies. He found that 10 to 20% of the "dieters" will maintain their weight loss or continue to lose weight, but few will actually reach their recommended weights. Unfortunately, the remaining dieters will return to their pre-reduction weights, and some will exceed their initial levels. Usually, individuals are motivated to lose weight during the initial dieting period but many fail to make a lifetime commitment to modify their lifestyle. A strong individual commitment is imperative if body weight is to be properly controlled over a lifetime. One question raised is: How do you guide individuals to make such a commitment?

METHODS AND MATERIALS

Body Weight Management Program (BWMP)

The BWMP was designed to assist overweight college females lose body weight sensibly and maintain weight loss. The philosophy of the BWMP was to modify the eating habits and lifestyle of the participants while they maintained a familiar eating and living environment. This program was directed by the present investigator and offered to the female occupants in an ISU residence complex, Maple-Willow-Larch (MWL). The dietary guidance aspect was integrated into the program with the cooperation of the MWL Food Service Facility. Appendix A gives the following: a detailed description of the program, the computerization of the food service menu items, and a sample of the daily menu provided to the participants. Changes in body weights and a discussion of the cyclic weight loss patterns of these females are provided also in Appendix A.

The Iowa State University Committee on the Use of Human Subjects in Research reviewed this project and concluded that the rights and welfare of the human subjects were adequately protected, that risks were outweighed by the potential benefits and expected value of the knowledge sought, that confidentiality of data was assured, and that informed consent was obtained by appropriate procedures. Supporting data for this approval are presented in Appendix B.

Description of Participants

The BWMP for the 1982-1983 school year began in September based on the encouraging results from a pilot study in May, 1982 (Appendix A). Of the 78 applicants, 29 females were selected to participate in the dietary counseling and blood sampling collection phases of the study. Five females (Subjects B, D, BB, T, and U) dropped out of the program within a couple of months. An additional four females (Subjects G, A1, A2, and B1) enrolled in the program in November, 1982. The BWMP ended in April, 1983, after the final blood collection period.

A general description of the 33 participants in the BWMP is given in Table 3. All were Caucasian females with relative body weights ranging from 106 to 152%. The body mass index [Wt (kg)/Ht (m)1.5] ranged from 29 to 40. The subjects were in good physical health as certified by a physician at the ISU Student Health Services prior to enrollment in the program. Most of the participants were in good academic standing, maintaining at least a 2.5 grade point average out of a possible 4.0. All of the subjects were non-smokers. No one was taking any type of medication throughout the study period. Only about one-half of the participants had regular menstrual cycles during the 7-month period.

Other characteristics of the 33 subjects are shown also in Table 3. The majority of the subjects (88%) indicated in a personal interview that they had lower-body-obesity compared to upper-body-obesity. Forty-four percent reported being overweight before menarche. The remainder of the subjects (56%) had become heavier following the onset of their

TABLE 3. Description of the females in the Body Weight Management Program experimental study, 1982-1983

Subject	Age (year)	Height cm (in)	IBW[a] [kg (in)]	RBW[b] (%)	RBW[c] (%)	BMI[d]	Obesity[e]	Onset[f]	RMR[g] (kcal/24 hr)
A1	19	176.8 (69.6)	68.2 (150.0)	106	96	29	upper	post	1207
E	21	174.0 (68.5)	67.0 (142.4)	107	97	29	lower	post	1631
C	18	164.6 (64.8)	62.2 (136.8)	111	99	29	lower	post	1485
L	19	160.3 (63.1)	60.3 (132.7)	112	99	30	lower	post	1584
I	20	167.6 (65.0)	63.6 (139.9)	112	100	29	lower	post	1415
K	20	171.3 (67.4)	67.8 (149.2)	112	100	30	lower	post	1267
A2	19	174.7 (68.7)	69.9 (153.8)	112	101	30	lower	post	1111
B	19	162.8 (64.1)	62.6 (137.7)	113	101	30	lower	post	1234
B1	19	167.6 (66.0)	66.0 (145.2)	113	101	30	upper	post	1111
D	18	165.2 (65.0)	64.7 (142.3)	114	101	30	lower	post	1468
H	19	168.2 (66.2)	67.3 (148.1)	115	103	31	lower	post	1630
J	21	156.7 (61.7)	61.6 (135.5)	119	106	31	lower	post	1285
M	21	167.0 (65.7)	68.4 (150.5)	119	106	32	lower	post	1267
P	21	173.7 (68.4)	74.3 (163.5)	119	108	33	lower	post	1500
S	19	177.7 (70.0)	81.3 (178.9)	125	113	34	lower	pre	1853
FF	22	167.8 (66.1)	73.8 (162.4)	126	113	34	lower	post	1482
Q	20	161.5 (63.6)	68.7 (151.1)	126	113	34	lower	pre	1354
U	18	169.5 (66.7)	74.9 (164.8)	127	114	34	lower	pre	1593
O	20	170.7 (67.2)	77.3 (170.1)	129	115	35	lower	pre	1494
T	19	154.5 (60.8)	65.5 (144.1)	130	115	34	lower	pre	1633
R	18	161.2 (63.5)	70.5 (155.1)	130	115	35	lower	post	1350
X	20	169.5 (66.7)	76.8 (169.0)	130	116	35	lower	pre	1578
N	19	168.2 (66.2)	77.3 (170.1)	132	118	35	lower	post	1402
Y	19	169.8 (66.9)	79.0 (173.8)	132	118	36	lower	pre	1676
W	20	164.9 (64.9)	76.0 (167.2)	134	119	36	lower	post	1723
Z	21	167.6 (66.0)	79.6 (175.1)	136	122	37	upper	pre	1606
V	19	167.9 (66.1)	81.0 (178.2)	138	124	37	lower	post	1639
AA	20	164.9 (64.9)	80.8 (177.8)	142	127	38	lower	pre	1594
G	20	164.6 (64.8)	81.7 (179.7)	145	129	39	lower	post	unk

BB	19	173.4 (68.3)	91.5 (201.3)	147	132	40	lower	pre	1845
EE	19	167.6 (66.0)	87.6 (192.7)	150	134	40	lower	post	1368
CC	21	153.9 (60.6)	76.5 (168.3)	152	134	40	upper	pre	1348
DD	19	156.7 (61.7)	78.6 (172.9)	152	134	40	lower	pre	1843

[a]Initial body weights (IBW) were recorded on the first blood collection period (Sept. '82) at the Student Health Services.

[b]Relative body weights (%) were calculated by $\frac{\text{actual body wieght}}{\text{desirable body weight}} \times 100$.
Desirable body weights were based on the Hathaway and Foard (71) measurements.

[c]Relative body weights (%) were calculated as described in footnote "b" above. Desirable body weights were based on the HANES (64) measurements which were estimated from regression equation of weight on height for women aged 20-29 years.

[d]Body Mass Index, W/H (in $Kg/m^{1.5}$).

[e]Obesity refers to pattern of body fat. Lower indicates a lower-body-obesity (fat is predominately on the hips and thighs) and upper refers to upper-body-obesity (fat is distributed mostly above the waist). This information was obtained from a personal interview with each participant of the BWMP.

[f]Onset of obesity refers to premenarche (overwieght during childhood) and postmenarche (excess weight following the onset of the menstrual cycle).

[g]RMR denotes resting metabolic rate. Standard procedures (72) were used to obtain these 2-hour postprandial measurements.

menstrual cycle (around 10 to 12 years of age). Again, this information was obtained from each participant in a personal interview by this investigator. Their resting metabolic rates (Spirometer, Warren E. Collins, Inc., Boston, MA) ranged from 1111 to 1853 kcal/24 hours. Dietary intakes were not analyzed in this study.

Most of the participants had a family history for obesity, diabetes, and hypertension (Table 4). The majority of the participants (n=21) reported that at least one immediate family member was overweight by at least 25 pounds. Diabetes (adult-onset) and hypertension were present also among the family members of a number of the participants. This information was also self-reported.

Blood Sample Collection

The original plan of the BWMP was to obtain a 12-hour fasting (fasted) blood sample and a 2-hour postprandial (fed) blood sample from the participants at various stages during their weight reduction. These samples were to be collected (1) immediately before the restricted caloric intake period, (2) when a relative weight of 125% (if relative weight was 150%) was achieved, (3) when a relative weight of 100±10% was reached, and (4) several months after a relative weight of 100±10% had been achieved and stabilized (see page 94, Appendix B).

The original blood collection schedule was not followed due to the erratic body weight changes of the participants. Instead, blood samples were obtained at the beginning (September, n=28) and end (April, n=24) of the BWMP. Blood samples were collected also in December (n=10) from

TABLE 4. Presence of obesity, adult-onset diabetes, and high blood pressure in family members of the females in the Body Weight Management Program[a]

Subject	Obesity	Diabetes	Hypertension
A1			
E	M/F/S		
C	GF		
L	F		
I			F
K	F/GF	F	
A2			F
B	M/B		B
B1			F
D			
H	F/S		M
J	GM	GM	M
M			GF/F
P	GF		GM
S	GF		GM
FF	M/F/S/B		M
Q			
U	F	U	
O		GF	
T	S		
R	M/F		
X			
N	M/F		
Y	M/F/B		
W			
Z	M/F		F
V	F		
AA	M/F/B		F
G			
BB	M/F/S/GF	GF	
EE	F	U	
CC		GF	
DD	M	A	

[a]A personal interview was conducted with each participant in the program concerning the presence of these disease states among family members. Thus, the presence of these states is subject to individual interpretation. Obesity is defined as 25 pounds or more above suggested weights for their heights. Diabetes and a hypertensive state prevailed when dietary and/or drugs were required. The following abbreviations indicate the presence of these disease states among family members: F=father, GF=grandfather, M=mother, S=sister, GM=grandmother, A=aunt, U=uncle, and B=brother.

four newly enrolled participants and six females who had lost about one-half of the weight they desired to lose.

The participants were instructed not to take any medication including aspirin 10 days prior to the blood collection day. The inhibition of eicosanoid synthesis by aspirin has been reviewed earlier. It is believed that the participants complied with this request. Many blood samples could not be collected at the specified stages due to the frequent usage of aspirin for menstrual cramps and headaches.

Blood was withdrawn by venipuncture with the use of a cuff by a medical technologist at the ISU Student Health Service. The 12-hour fasting samples (fasted) were obtained before breakfast and the 2-hour postprandial samples (fed) were collected after the noon meal on the same day.

Collection methods for plasma and serum eicosanoids have been described previously (16) and are probably the most crucial phase of eicosanoid assays. The following modified procedure was followed for the collection and the immediate processing of samples for plasma eicosanoids, serum eicosanoids, plasma insulin, and plasma glucose, respectively. First, blood for plasma eicosanoid analyses was drawn into a 10 ml vacutainer tube containing 1.0 ml of a disodium ethylenediamine tetraacetate (EDTA)-aspirin solution. This tube was inverted and placed on ice for 10 minutes. Second, blood for serum eicosanoids was drawn into vacutainer tube (5 ml) without anticoagulant-eicosanoid interfering substances and was allowed to clot for exactly 10 minutes in a water bath at 37°C. The 10-minute timing began as blood

was drawn into the tube. Serum eicosanoid production was stopped by adding 0.5 ml of 4.2 mM aspirin in a 0.1 M potassium phosphate buffer (pH 7.4). Third, blood for the plasma insulin and glucose assays was obtained in a 7 ml heparinized vacutainer tube and remained at room temperature for exactly 10 minutes. All of these three tubes were centrifuged (unrefrigerated) at 2000xg for 10 minutes.

Further processing of the samples was performed at the Student Health Services. Following the removal of platelets from the blood by centrifugation, 200 µl aliquots were pipetted for plasma PGE_1, PGE_2, and Met-I_2 analyses. A 400 µl aliquot was measured for the plasma TXB_2 assay. Aliquots for serum eicosanoid assays were not made immediately. The serum was removed by aspiration, transferred to one tube and stored at -20°C. Prior to the laboratory analyses, the serum was thawed at room temperature. Aliquots of 300 µl and 50 µl were taken for the serum PGE_1 and PGE_2 analyses, respectively. A 200 µl aliquot from a 1/10 dilution in phosphate buffered saline (PBS) - 0.1% gelatin was made for the serum TXB_2 assay. Plasma for the insulin and glucose analyses was removed and stored in two separate tubes. Aliquots of 100 µl and 10 µl were taken prior to the insulin and glucose analyses, respectively. All tubes were stored at -20°C until laboratory analysis. Assays for plasma eicosanoid, insulin, and glucose levels were performed when plasma was thawed for the first time (16). Duplicate aliquots were measured for all assays.

The incubation of blood for serum eicosanoid analysis is termed an ex vivo incubation (20). Whole blood was allowed to clot for exactly 10

minutes at 37°C. Addition of aspirin was then used to stop eicosanoid synthesis. The measurement of serum eicosanoids is the amount of eicosanoid synthesized in a 10-minute period in the absence of an exogenous substrate. It is assumed that this measurement is indicative of the ability of tissues to synthesize eicosanoids in vivo. Generally, it is believed that plasma eicosanoid measurements are indicative of endogenous vascular release of PGI_2, PGE_1, and PGE_2. As mentioned previously, aspirin is present in the vacutainer tube as the blood is drawn into the tube. This prevents further eicosanoid synthesis.

All of the samples for eicosanoid, insulin, and glucose analyses from one female were evaluated in the same assay run. Moreover, the fasted and fed samples from one subject were analyzed alternately in the assay run. When results of the duplicate samples did not agree within 10%, both the fasted and corresponding fed sample were reassayed.

Analytical Methods

Eicosanoid assay

Fasted and postprandial serum samples were assayed for PGE_1, PGE_2, and TXB_2. Likewise, plasma samples were analyzed for PGE_1, PGE_2, TXB_2, and also Met-I_2. The radioimmunoassay procedure by McCosh et al. (17) was used for the eicosanoid analyses.

This radioimmunoassay method is based upon the double-antibody procedure. Briefly, it involves the overnight pre-precipitation of anti-rabbit gamma globulin with the specific antibody. This suspended pre-precipitated antisera solution is mixed with duplicate unknown

samples and tritiated standards, and a 24-hour precipitation period is then required. Finally, the tubes are centrifuged and prepared for counting by liquid scintillation.

Results were analyzed by use of a computer program obtained from Lousiana State University (73). This program plotted the percent bound of labelled antigen as a function of the quantity of unlabelled antigen added versus a log transformation of the standard concentrations.

Insulin analysis

Insulin concentrations were determined in the fasted and fed plasma samples. An insulin kit (Code IM.78, Amersham, Arlington Heights, IL 60005) was used for this quantitative measurement. A 96-hour incubation period was used instead of the usual 3-hour incubation. This modification was necessary to obtain more sensitive measurements of the insulin concentrations below 10 microunits/ml.

In general, the modified procedure involved a 24-hour incubation of anti-insulin serum (guinea pig) with samples and standard insulin. These samples were incubated for an additional 72 hours with ^{125}I-insulin prepared from purified bovine insulin. The insoluble antigen-antibody complex was then separated from the soluble free insulin by centrifugation. By counting the radioactivity in the precipitate from both standards and sample unknowns, a standard curve was constructed and unknown insulin levels were interpolated.

Glucose analysis

Glucose concentrations were measured in the fasted and postprandial plasma samples. A method based on the use of glucose oxidase (GOD) as reported by Raabo and Terkildsen (74) was used.

Briefly, the procedure is based on the following enzymatic reactions.

$$\text{Glucose} + 2\ H_2O + O_2 \xrightarrow[\text{Glucose Oxidase}]{} \text{Gluconic Acid} + 2\ H_2O_2$$

$$H_2O_2 + \underset{\text{(Colorless)}}{\text{O-Dianisidine}} \xrightarrow[\text{Peroxidase}]{} \underset{\text{(Brown)}}{\text{Oxidized O-Dianisidine}}$$

Following a 30-minute incubation at 37°C, the samples were promptly read on a spectrometer (Gilford 240) at 450 nm.

Glucose concentrations of the samples were calculated by dividing the absorbance of the sample unknown by the absorbance of a glucose standard. This value was multiplied by 100. Absorbance of the standard solution was verified by a standard curve (slope = 1). The accuracy and precision of the the glucose oxidase method was verified by a Moni-trol I Chemistry (DADE Division, Miami, FL) control solution.

Statistical Analysis

Data were analyzed using the paired t-test, Students' t-test, analysis of variance and Duncan's Multiple Range Test with the Statistical Analysis System (SAS Institute, Cary, N.C.). Correlation coefficients were also calculated. The statistical probability of 0.05 was considered significant.

RESULTS

The effects of the nutritional state (fasted and fed) on eicosanoid, insulin and glucose concentrations are presented in Table 5. Paired t-test showed that the 2-hour postprandial serum samples contained significantly ($P \leq 0.01$) more TXB_2 than did the 12-hour fasted samples. Plasma insulin concentrations were significantly ($P \leq 0.0001$) higher in the fed state compared to the fasted condition. Plasma glucose and all of the other eicosanoid concentrations examined did not differ significantly between the two collection periods.

Several significant relationships based on correlation coefficients were found for the parameters measured in this study (Table 6). During a fasted state, more significant correlations among the various eicosanoids were obtained than in the fed state. Serum PGE_1 synthesis correlated with most of the eicosanoids measured in the fasted condition. Significant correlations of eicosanoids with both glucose and body weight were found during the fed state.

Fasted and fed eicosanoid concentrations were examined separately (paired t-test) during the three blood collection periods (September, December, and April). Except for serum TXB_2, no significant differences were found between the fasted and fed samples. The absolute concentrations of these parameters may have been higher or lower during a specific month but differences due to the nutritional states were not significant. On the other hand, serum TXB_2 concentrations in the fed samples were significantly ($P \leq 0.0001$) greater compared to fasted samples in Septemper (Table 7). In December, the serum TXB_2 concentration was

TABLE 5. Eicosanoid, insulin, and glucose concentrations[a] in fasted and fed blood samples[b] in overweight females

Parameter	Fasted	Fed	Level of Significance
Serum PGE_1	0.93 ± 0.12	0.96 ± 0.12	
Serum PGE_2	0.56 ± 0.09	0.58 ± 0.09	
Plasma PGE_1	1.18 ± 0.16	1.02 ± 0.14	
Plasma PGE_2	2.08 ± 0.31	1.96 ± 0.30	
Serum TXB_2	15.80 ± 2.77	22.18 ± 1.78	$P < 0.01$
Plasma TXB_2	4.56 ± 0.28	4.46 ± 0.28	
Plasma Met-I_2	0.19 ± 0.03	0.19 ± 0.05	
Plasma Insulin	23.5 ± 1.7	49.7 ± 4.4	$P < 0.0001$
Plasma Glucose	69.4 ± 1.5	70.3 ± 1.7	

[a]Eicosanoid, insulin, and glucose concentrations are expressed as ng/ml, µl/ml, and mg%, respectively. Data were analyzed by the paired t-test.

[b]Fasted (12-hour) and fed (2-hour; postprandial) blood samples were obtained from a total of 33 females during September and/or December and/or April. Means ± SEM are based on a total of 61 blood samples for each fasted and fed state.

greater in the fed samples than in the fasted samples but the difference was not significant ($P \leq 0.06$). In April, serum TXB_2 concentrations were similar for the two collection periods.

In general, eicosanoid concentrations were not significantly related to the three indices of body fatness: body weight, relative body weight, and body mass index based on Duncan's Multiple Range Test. However, there was a significant positive correlation between Met-I_2 concentration and body weight during the fed state (Table 6). Eicosanoid concentrations were not affected by a loss or gain in body weight or body-type-obesity (students' t-test). The onset of obesity (premenarche or postmenarche) had no effect on eicosanoid concentrations, except for serum PGE_1. Serum PGE_1 concentrations were significantly greater in the females with childhood obesity (premenarche, 1.29 ± 0.24 ng/ml) compared to a postmenarche obese individual (0.73 ± 0.10 ng/ml).

Some relationships were found between glucose and eicosanoid concentrations in this research (Table 6). The fasting glucose level was negatively correlated to serum PGE_1 concentration ($r=-0.32$, $P \leq 0.01$). During the fed state, positive correlations were found for glucose concentration with serum PGE_2 concentration and with plasma TXB_2 ($r=+0.32$, $P \leq 0.01$).

Glucose and insulin concentrations were not related to loss or gain in body weight, body-type-obesity, or onset of obesity of the participants based on the students' t-test. During a fed state, a positive relationship ($r=+0.56$, $P \leq 0.0001$) was found between glucose and

TABLE 6. Relationships between eicosanoids, insulin, and glucose concentrations in fasted and fed blood samples[a] in overweight females

Parameters	Fasted		Fed	
	r	Level of Significance	r	Level of Significance
Serum PGE_1 * Serum PGE_2	+0.41	P<0.001	+0.41	P<0.001
Serum PGE_1 * Plasma PGE_1	+0.51	P<0.0001	+0.53	P<0.0001
Serum PGE_1 * Serum TXB_2	-0.32	P<0.01		
Serum PGE_1 * Plasma PGE_2	-0.31	P<0.02		
Serum PGE_1 * Plasma TXB_2	+0.30	P<0.02		
Serum PGE_2 * Plasma TXB_2	+0.50	P<0.0001	+0.50	P<0.0001
Serum TXB_2 * Plasma PGE_1	-0.39	P<0.005		
Serum TXB_2 * Plasma PGE_2	+0.36	P<0.005		
Plasma Met-I_2* Plasma PGE_1	+0.54	P<0.0001	+0.34	P<0.02
Plasma PGE_1 * Plasma PGE_2	-0.38	P<0.005	-0.36	P<0.01
Serum PGE_1 * Plasma Glucose	-0.32	P<0.01		
Serum PGE_2 * Plasma Glucose			+0.32	P<0.01
Plasma TXB_2 * Plasma Glucose			+0.32	P<0.01
Plasma Glucose*Plasma Insulin			+0.56	P<0.0001
Plasma Glucose*RBW[b]			+0.34	P<0.01
Plasma Met-I_2* Body Weight			+0.33	P<0.01

[a]Fasted (12-hour and fed (2-hour) postprandial blood samples were obtained from a total of 33 females during September and/or December and/or April. Partial correlations were based on a total of 61 blood samples for each fasted and fed state.

[b]Relative body weights were based on the Hathaway and Foard (71) measurements.

insulin concentrations (Table 6). Additionally, glucose concentrations and relative body weights were positively correlated during a fed state (Table 6).

In the present study, a seasonal effect on eicosanoid production may be present. Analysis of variance of data from 19 females (only 19 of the 33 participants had blood samples taken in both September and April) indicated that serum TXB_2, serum PGE_1, serum PGE_2, plasma PGE_1, and plasma PGE_2 concentrations differed significantly in the September blood samples compared to the April samples. For example, September samples had significantly ($P \leq 0.0001$) less serum TXB_2 (Figure 2) and plasma PGE_2 (Figure 3) than the April samples. In fact, plasma PGE_2 concentrations in April were about 4 times higher than in September. Differences in serum TXB_2 were even more pronounced.

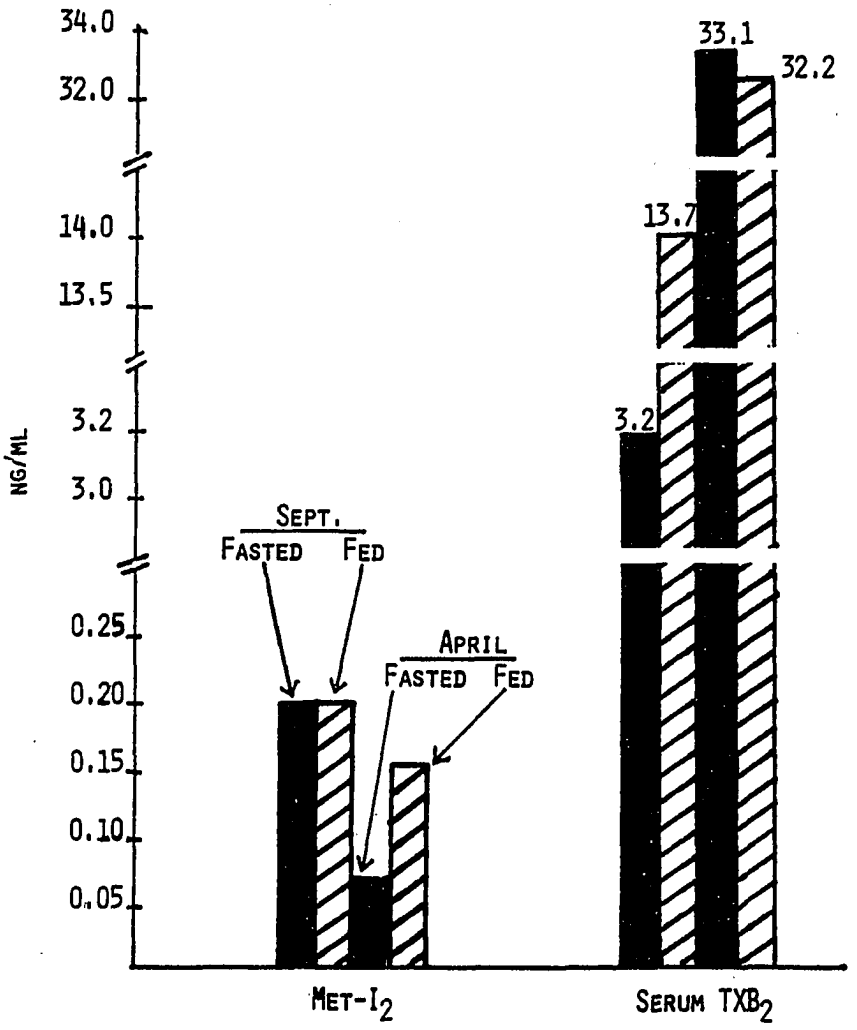
On the other hand, plasma PGE_1, serum PGE_1, and serum PGE_2 concentrations responded in an opposite fashion to serum TXB_2 and plasma PGE_2. September blood samples showed significantly ($P \leq 0.0005$) more plasma PGE_1 (Figure 3), serum PGE_1 (Figure 4), and serum PGE_2 (Figure 4) than the April samples. Although not significant, fasted and fed concentrations of Met-I_2 were higher in September than in April (Figure 2). Plasma TXB_2 concentrations in September and April were similar.

TABLE 7. Serum thromboxane B_2 concentration[a] in fasted and fed samples[b] from overweight females in three different months

Month	Fasted	Fed	Level of Significance
September, 1982 (n = 28)	4.99 ± 2.11	14.16 ± 3.25	$P < 0.0001$
December, 1982 (n = 10)	10.99 ± 3.44	22.35 ± 7.71	NS
April, 1983 (n = 24)	31.76 ± 5.42	31.87 ± 4.64	NS

[a]Synthesis of TXB_2 (Mean ± SEM; mg/ml) is during 10 minutes of ex vivo clotting in blood samples. Data were analyzed by the pair t-test.

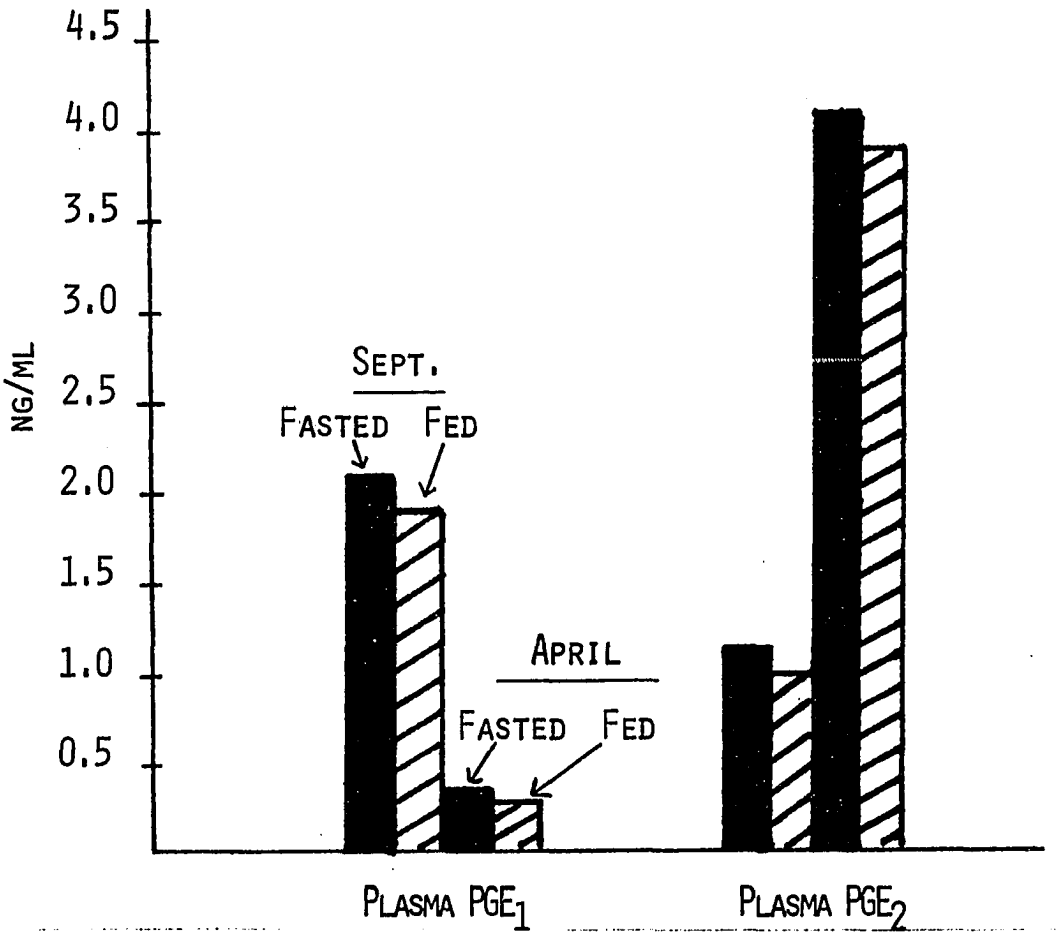
[b]Fasted (12-hour) and postprandial (2-hour) blood samples were collected.



Probability Level from 2-Way Factorial with Interaction

Season	NS	$P < 0.0001$
Nutritional State (NS)	NS	NS
Season * NS	NS	NS

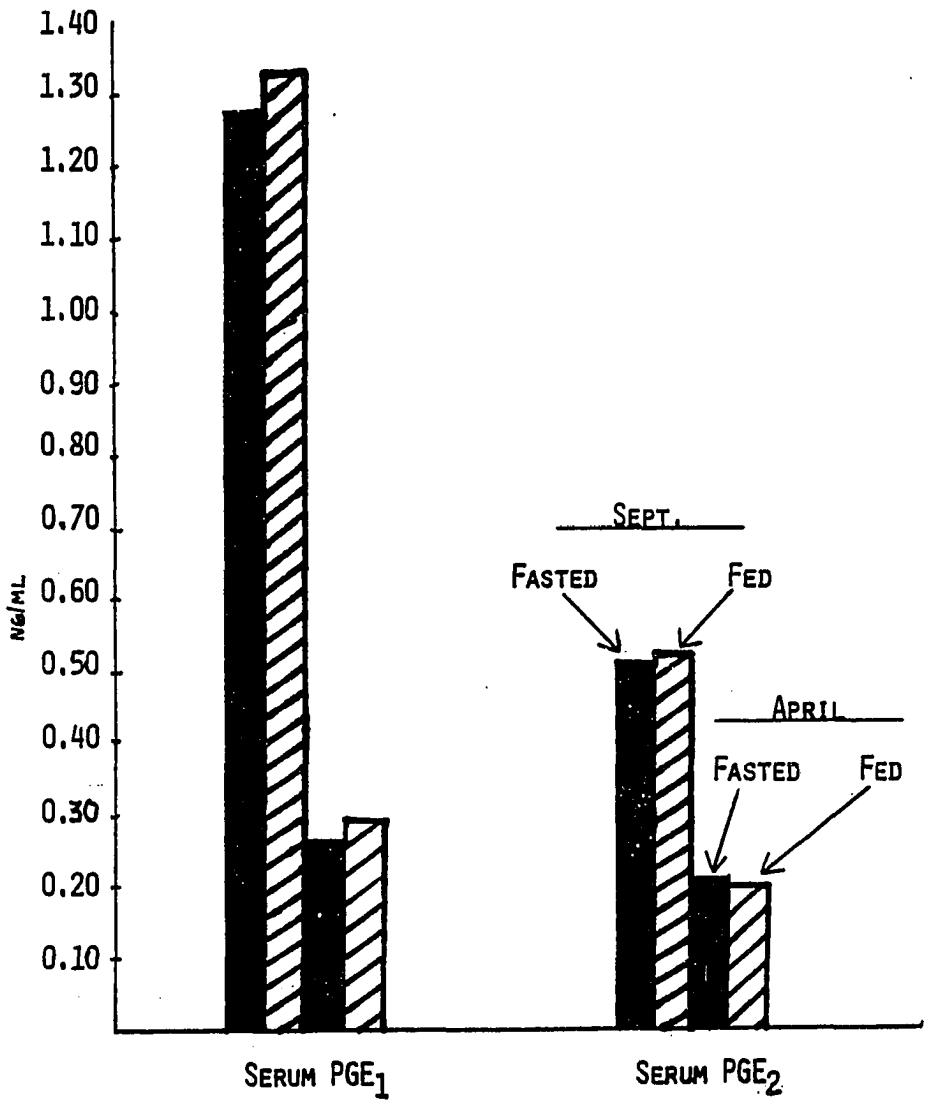
FIGURE 2. Effect of season on plasma prostacyclin (Met-I_2) and serum thromboxane (TXB_2) in fasted (12-hour) and fed (2-hour) blood samples in overweight females



PROBABILITY LEVEL FROM 2-WAY FACTORIAL WITH INTERACTION

SEASON	P<0.0001	P<0.0001
NUTRITIONAL STATE (NS)	NS	NS
SEASON * NS	NS	NS

FIGURE 3. Effect of season on plasma PGE_1 and plasma PGE_2 levels in fasted (12-hour) and fed (2-hour) blood samples in overweight females



Probability Level from 2-Way Factorial with Interaction

Season	$P < 0.0001$	$P < 0.005$
Nutritional State (NS)	NS	NS
Season * NS	NS	NS

FIGURE 4. Effect of season on serum PGE_1 and serum PGE_2 levels in fasted (12-hour) and fed (2-hour) blood samples in overweight females

DISCUSSION

Information is available on eicosanoid concentrations in body fluids, such as serum and plasma in normal, healthy individuals but the results reported are contradictory. In the present study, a number of eicosanoids in both plasma and serum samples in humans, were evaluated. Early investigations, usually on an in vitro basis, focused on prostaglandins of the E series. Since the discovery of PGI_2 and TXA_2 in the late seventies, research has been conducted mainly on these two labile eicosanoids. Most of the studies, mainly in vitro, investigated PGI_2 production in the endothelium and/or TXA_2 synthesis by the platelets (1, 3). Human studies are beginning to explore eicosanoid concentrations in serum and plasma especially in relation to diabetes and cardiovascular disease (16, 21, 25, 27, 31).

A paucity of information exists on several other aspects of the present investigation. First, only a few investigators have examined specifically the effect of the nutritional state (fasting vs fed) on eicosanoid concentration (11, 18, 19, 47). The results from these studies are contradictory. Some research results indicate that feeding, especially unsaturated fatty acids, stimulates platelet activity in humans (50, 51, 52). In contrast, plasma free fatty acid levels are elevated in a fasting state. Thus, greater amounts of free fatty acids would be available for eicosanoid synthesis. Second, the relationship between body weight and eicosanoid concentration in blood has not been studied in a human population. Based on in vitro studies, it has been suggested that PG production is excessive in the obese state (10).

Finally, the seasonal variation of eicosanoid concentrations has never been documented.

The results from the present study indicate that a fed state stimulates TXB_2 synthesis in humans. Platelet eicosanoid synthesis was responsive to the fed condition, as indicated by the significantly higher serum TXB_2 concentration in the fed samples than in the fasted samples (Table 5). The increased serum TXB_2 concentration suggests that platelet activation occurs upon feeding in humans. This activation would be associated also with an increased platelet adhesiveness and an increased platelet clumping. These in vitro observations have been verified by other investigators (44, 50, 51, 52).

Although platelets synthesize predominately TXA_2, PGE_2 and to a lesser extent PGE_1 also are released during the aggregation process (3). Mean concentrations of PGE_2 and PGE_1, however, were not increased significantly during the fed state compared to the fasted period. In humans, serum PGE_2 and serum PGE_1 synthesis do not appear to be sensitive to a nutritional state due to the selective production of TXB_2.

In this study, the observation that feeding stimulates serum TXB_2 synthesis in humans contradicts the results obtained by Sullivan and Mathias (11) in the animal model. In rats, they observed that compared to the fed state, fasting increased serum TXB_2 and PGE_2 concentrations by 62 and 48%, respectively. Moreover, the significant effects of various dietary linoleate treatments on eicosanoid concentrations were detected only in the fed samples. Due to the opposing results of these

two investigations, one might speculate that the apparent discrepancy is species specific. Lipoprotein fractions differ between the rat and human model (75, 77) and the contribution of these fractions to eicosanoid concentrations has not been elucidated.

The relationships between plasma and serum eicosanoids were interesting, but in some cases, were difficult to interpret due to the lack of previous documentation (Table 7). In this study, some correlation coefficients reflect opposing physiological effects of eicosanoids on platelet aggregation (Table 6). For example, a significant negative correlation (r=-0.38, fasted; r=-0.36, fed) between plasma PGE_1 and plasma PGE_2 was found. A significant positive correlation was found between plasma PGE_1 and Met-I_2, both of which inhibit platelet aggregation (1, 2, 14). Significant positive relationships were found in serum and/or plasma samples between PGE_2 and TXB_2, both of which promote platelet aggregation (1, 2, 14). But the same trends failed with the following positive significant correlations: between serum PGE_1 (inhibits) and serum PGE_2 (promotes), and between serum PGE_1 (inhibits) and plasma TXB_2 (promotes). These direct correlations between eicosanoids with opposing physiogical effects on platelet aggregation may be anomalous.

The energy balance status of the participants differed between the three blood collection periods (September, December, and April). The original plan of the Body Weight Management Program was to obtain a blood sample before weight loss in September (baseline samples at initial body weights). The September blood collections, however, were

obtained from the participants (n=29) during the first two weeks of a negative energy balance. This occurred because once the participants had been notified of their acceptance into the BWMP, they began immediately to restrict their caloric intake. The December blood collection (n=10) is based on six females who had lost one-half of the weight (ranging from -3.4 to -9.1 kg) they desired to lose and four females who had been in the program for about one month (enrolled in November). By this time, weight loss had slowed down possibly due to physiological adaptation to the energy deficit. In addition, the energy deficit may have been smaller in December compared to September. In April, energy balance had returned to pre-reduction levels as indicated by the body weight gain in the majority of the females (n=24) (Appendix A).

The effect of energy balance on eicosanoid production has not been documented. Although feeding may stimulate platelet activation (inducing TXB_2 production), energy balance may explain in part the different effects of the nutritional state on TXB_2 synthesis. To illustrate the effect of energy balance on serum TXB_2, fed/fasted ratios of serum TXB_2 levels were calculated (Table 7). The fed/fasted ratio approached a value of 3 during the initial phase of weight loss (September). As weight loss slowed down due to adaptation (December), the ratio decreased to a value of 2. A fed/fasted ratio of 1 was found during weight maintenance or weight gain of the females (April). Differences due to energy balance were not found for the other eicosanoids examined in the present study.

It is known that subjects in negative energy balance, lipolysis is increased in order to use fatty acids as a source of energy (68). Under these circumstances, linoleic acid is used as an energy source rather than being elongated to synthesize arachidonic acid. This might suggest that in September, a relative arachidonic acid deficiency was present, explaining the lowest TXB_2 concentration by platelets in the fasting blood samples (Table 8). After feeding (postprandial blood samples), more arachidonic acid would be available to be utilized by platelets, which may account for the increase in TXB_2 concentration in relation to fasting. After a metabolic adaptation had occurred in December, the need of linoleic acid as a source of energy would be decreased, making it more available for arachidonic acid synthesis and TXB_2 production. In April, the energy balance had returned to pre-reduction levels, and more arachidonic acid would be available.

The effect of energy balance was not observed in Met-I2 and PGE1 concentrations. These two eicosanoids produce vasodilation and both are synthesized by adipocytes (10, 78). It has been suggested that an increase in synthesis of these two eicosanoids occurs during lipolysis in order to increase the blood flow through the adipose tissue and the fatty acid transport to the liver (79). This might explain the decrease in these eicosanoid concentrations in April.

An imbalance in eicosanoid production has been associated with clinical manifestations of diabetes and cardiovascular disease. In vitro study results suggest that circulating glucose levels may promote a decreased PGI_2 production from endothelial tissue and/or an increased

TXB_2 synthesis by platelets (6, 9, 29, 30). Other results indicate that eicosanoid concentrations are not influenced by circulating glucose levels (21, 31, 32).

The observed relationships between glucose and eicosanoids in the present study reflect this complex relationship. Fasting glucose levels were inversely related to serum PGE_1 concentration (Table 6). A similar association was found by Storms (19) in diabetic patients. In contrast, postprandial glucose levels were positively correlated to serum PGE_2 and plasma TXB_2 levels. Although the mean glucose concentrations did not differ significantly between the fasted and 2-hour postprandial samples (Table 5), glucose levels after feeding might have a latent effect on serum PGE_2 and plasma TXB_2 synthesis. The increased concentrations of glucose (e.g., a 1-hour postprandial sample) may trigger a subsequent increase in the PGE_2 and TXB_2 concentrations. This particular response of eicosanoid production to increased glucose concentrations may be associated with the development of certain pathological conditions. Unfortunately, the onset of these conditions may not become apparent biochemically for some time after the initial aberration.

In addition to the 2-hour postprandial blood samples, a 1-hour fed sample was obtained from 10 of the 29 subjects in September. Regardless of the 1-hour or 2-hour sample collection, the relationships between eicosanoid, insulin, and glucose remained the same as those presented in Table 6. As expected, the 1-hour fed samples had significantly ($P \leq 0.0001$) higher glucose concentrations than the fasted samples.

A 2-hour fed sample rather than a 1-hour sample was chosen to

compare the present findings to those reported by Storms (19). Using the same antiserum to measure eicosanoid concentrations as in this study, she examined eicosanoid and free fatty acid levels in 2-hour and 4-hour postprandial samples from four insulin-dependent diabetic subjects during various dietary treatments. Plasma PGE_1 and plasma PGE_2 concentrations were greater during the fasting state compared to the fed condition in her subjects. In the present study, mean concentrations of plasma PGE_1 and PGE_2 did not differ significantly between the two collection periods. Recently, fasted and 2-hour postprandial blood samples have been collected from ten normal weight females during a metabolic balance study. The results from this particular study are forthcoming and will provide further insight into the effects of the nutritional state on eicosanoid production.

Relative body weights were significantly and positively correlated to postprandial glucose concentrations (Table 6); this result is consistent with the findings by Bagdade et al. (58). As expected, postprandial glucose concentrations were positively correlated to postprandial insulin concentrations. Mean concentrations of fasted and fed glucose and insulin for these overweight females were within a normal range (77). Kissebah and associates (61) found elevated insulin and glucose concentrations in upper-body-obese females. Relationships between body-type-obesity to insulin and glucose concentrations could not be verified in this research due to the limited number of upper-body-obese females (Subjects A1, B1, A, and C), as well as the subjective criterion used for judging body-type-obesity.

It has been suggested that prostaglandin synthesis, mainly PGE_1 and PGE_2, is excessive in obesity and may inhibit the release of free fatty acids and glycerol from adipose tissue (10). In the present in vivo study, concentrations of PGE_1, PGE_2, and also TXB_2 were not related to the indices of body fatness. It has been reported that adipose tissue produces significant amounts of PGI_2 (79). Consequently, a positive association between plasma Met-I_2 and body weight (Table 6) may be explained by an increased amount of adipose tissue and an increased production of Met-I_2 by this tissue. Moreover, the total amount of blood vessels is greater in obese people than in normal weight individuals; thus, the total amount of endothelium available for Met-I_2 production is also greater in obesity.

A seasonal effect on eicosanoid concentrations has not been reported in the literature although Dupont has observed this phenomenon (personal communication, Department of Food and Nutrition, ISU). In general, parallel trends were evident for the eicosanoids having a similar effect on platelet aggregation regardless of plasma or serum origin. Plasma PGE_1 (Figure 3) and serum PGE_1 (Figure 4) concentrations were significantly less in April than in September. Although not significant, both fasted and fed Met-I_2 concentrations (Figure 2) tend to be less in April than in September.

The lower concentrations of the anti-aggregatory eicosanoids (PGE_1 and Met-I_2) from September to April is accompanied by significantly higher concentrations of serum TXB_2 (Figure 2) and plasma PGE_2 (Figure 3). Again, it is evident that a balance is maintained for most of the

eicosanoids having opposing effects on platelet aggregation. The decline in serum PGE_2 concentration (Figure 4) in April compared to September may be anomalous. At present, no explanations can be offered for the seasonal trends observed in this study. Eicosanoid degradation during storage can be excluded since some of the levels increased during the sample storage period. Morris et al. (16) have conducted one of the more thorough investigations on the variables that affect eicosanoid concentrations. They do not indicate that the length of sample storage has any effect on eicosanoid production. Precautions suggested by these researchers include the immediate processing of samples and the evaluation of eicosanoid concentrations after the initial thawing of samples. These factors were controlled in the present study.

The relative concentrations of eicosanoids in body fluids in humans are unknown. Examination of the mean values and standard deviations from many studies indicates wide variability (27, 28, 40). Eicosanoid concentrations varied widely also in this study (Table 5). In addition, concentrations of eicosanoids in serum may not represent the true blood levels. For example, the plasma PGE_2 concentration is higher than the serum PGE_2 concentration (Table 5). As suggested by Morris and associates (16), plasma eicosanoid concentrations may be higher than serum concentrations due to "artifactual increases in plasma concentrations." Moreover, calculations of plasma PGE_2 concentrations were difficult due to this artifactual increase. In agreement with other studies (1-3), TXB_2 in platelets was the most important quantitatively (Table 5).

Many factors affect eicosanoid concentration. Methods used in the collection and processing of samples influence assay results. Eicosanoid concentrations will vary depending upon techniques used such as radioimmunoassay, gas chromatography, or mass spectrometry (16, 17). The latter two methods are not suitable for large numbers of samples. Radioimmunoassay is a precise and rapid means for quantifying eicosanoid concentrations in blood. Other factors such as exercise levels in humans (18), dietary fat (50-52), and nutritional state (11, 18, 19) have been found to affect these concentrations also.

As mentioned earlier, the time of the sample collection and eicosanoid assay were similiar for the study by Storms (19) and the present investigation. For example, (1) 12-hour fasting and 2-hour postprandial blood samples were collected, (2) serum and plasma eicosanoid concentrations were determined in the same laboratory facility using the same reagents and methodology, and (3) the first and second blood collection tubes were used for the plasma and serum eicosanoid assays, respectively. Eicosanoid results from the two studies, however, differed greatly (Table 8). It is known that diabetics have lower concentrations of Met-I_2 than normal subjects (6, 7, 8, 24). A comparison of the Met-I_2 concentrations from the diabetic patients in the study by Storms and the present investigation certainly support this assumption (Table 8). However, the differences in the Met-I_2 concentrations, as well as the other eicosanoids, between these two studies cannot be attributed to background of the subjects.

The blood drawing technique was different in the study by Storms

TABLE 8. Eicosanoid concentrations[a] in fasted blood samples from the overweight females in the present research (Struempler) and from the study by Storms

	Struempler[b] (Study 1)	Storms[c] (Study 2)	$\frac{\text{Study 1}}{\text{Study 2}}$ Ratio
Plasma TXB_2	4560	151	30.2
Plasma Met-I_2	190	2.5	74.5
Serum TXB_2	15800	41600	0.38
Serum PGE_1	930	215	4.33
Plasma PGE_1	1180	475	2.48
Serum PGE_2	560	823	0.68
Plasma PGE_2	2080	544	3.82

[a]Eicosanoid concentrations, pg/ml.

[b]Overweight college females (n=33) were the participants in Study 1 by Struempler (present investigation).

[c]The eicosanoid levels reported by Storms (19) are based on four insulin-dependent adults.

(19) than in the present investigation. The blood samples were drawn using an in-dwelling catheter that had been put in the forearm vein of the hospitalized diabetic patients examined by Storms. In contrast, the blood samples from the free-living women in the present study were obtained by venipuncture with the use of a cuff.

It has been reported that ischemia produced by a cuff during blood collection increases eicosanoid concentrations (11, 80). During the first minute of ischemia occlusion, the eicosanoid concentrations are believed to be the highest (80). In addition, Morris et al. (16) have indicated that any problems encountered during blood collection, such as slow withdrawal of blood and/or manipulation of the needle, will increase eicosanoid concentrations. These observations would explain the elevated concentrations for the plasma eicosanoids as indicated by the higher ratios in the present study compared to those reported by Storms (19) (Table 8).

In summary, the present investigation contributes valuable information related to eicosanoid concentrations. The results suggest that several factors influence eicosanoid concentrations in blood. Nutritional state and/or energy status of the individual affect these concentrations. But additional observations on normal human subjects are necessary to confirm these observations. These apparent effects emphasize the need for the adoption of a standardized procedure for the collection and processing of blood samples for eicosanoid assays. Further research on eicosanoid concentrations in serum and plasma is required. Moreover, blood collection for serial eicosanoid

concentrations in humans should be obtained within the shortest time span possible. This would minimize any possible seasonal effect on eicosanoid concentrations.

CONCLUSIONS AND RECOMMENDATIONS

Eicosanoid concentrations in serum and plasma are of recent interest especially in relation to pathological conditions, such as diabetes mellitus and cardiovascular disease. In the present study, eicosanoid concentrations in overweight college women were examined. Eicosanoid measurements included plasma PGE_1, PGE_2, TXB_2, and Met-I_2; serum levels of PGE_1, PGE_2, and TXB_2 also were evaluated. Blood samples were obtained from 33 participants in a Body Weight Management Program which emphasized weight reduction by moderate and sensible changes in dietary and lifestyle habits.

Many factors affect eicosanoid concentrations in serum and plasma. In the present study, at least three factors were found to influence eicosanoid concentration. They include:

(1) Effect of Nutritional State: Nutritional state of these females refers to the collection of blood samples for eicosanoid analysis in relation to the last meal: a 12-hour fasting (fasted) and a 2-hour postprandial (fed) blood sample. Serum TXB_2 was the only eicosanoid examined that was affected by the nutritional state. The 2-hour postprandial serum sample produced significantly more TXB_2 that did the 12-hour fasted sample. The increased serum TXB_2 concentration suggests that platelet activation occurs upon feeding in humans. This observation contradicts the effect of the nutritional state on eicosanoid production in the rat. It has been reported that serum TXB_2 and PGE_2 concentrations are greater in fasted samples compared to fed samples in the rat. One might speculate that the apparent discrepancy

is species specific. Lipoprotein fractions differ between the rat and human model and the contribution of these fractions to eicosanoid concentrations has not been elucidated.

(2) Effect of Energy Balance: Energy balance was based on body weight changes of the females and for different periods, three patterns were observed: (1) an initial weight reduction period when weight loss was most rapid (September), (2) an intermediate weight period when weight loss slowed down possibly due to adaptation (December), and (3) the final phase when body weights for the majority of the females had returned to or exceeded pre-reduction levels (April). In addition to the nutritional state effect, energy balance may influence serum TXB_2 levels (based on fed/fasted ratios). For serum TXB_2 concentrations, the fed/fasted ratios were 2.8, 2, and 1 for the September, December, and April samples, respectively. In this respect, the difference between the fed and fasted serum TXB_2 levels was more pronounced during a negative energy balance period. As energy balance approached or exceeded pre-reduction levels (positive), serum TXB_2 concentrations did not differ significantly between fed and fasted samples. This observation was not found for the other eicosanoids measured in this study. The effect of energy balance on eicosanoid concentrations has not been documented in the literature.

(3) Seasonal Effect: Eicosanoid concentrations from the September and April blood samples were significantly different in the present study. In general, parallel trends were evident for the eicosanoids having a similar effect on platelet aggregation. The

concentrations of the anti-aggregatory eicosanoids (PGE_1 and $Met\text{-}I_2$) were higher in September than in April. In contrast, the concentrations of the pro-aggregatory eicosanoids, PGE_2 (in plasma) and TXB_2 (in serum), were significantly lower in September than in April. These results may suggest a seasonal effect upon eicosanoid concentrations, although an interaction between energy balance and season should not be ruled out. A seasonal effect on eicosanoid concentrations has not been reported in the literature and requires verification. To control for. any possible seasonal effect on eicosanoids, it might be recommended that blood collection for serial eicosanoid assays in humans should be obtained within the shortest time span possible.

These results suggest that several factors may influence eicosanoid concentrations in blood. Nutritional state or possibly the energy balance status of the individual affects these concentrations. But additional observations on normal human subjects are necessary to confirm these observations. These apparent effects on eicosanoid concentrations emphasize the need for the adoption of a standardized procedure for the collection and processing of blood samples for eicosanoid assays. More data are needed on eicosanoid concentrations in serum and plasma.

Several significant correlations were found for the parameters evaluated in this investigation. Numerous relationships were found among the eicosanoid family members but their physiological significance is not known. In addition, body weights and blood glucose concentrations of the females were related to some of the eicosanoid

levels. Two correlations found were:

(1) Met-I_2 Concentrations and Body Weights: A positive relationship was found between Met-I_2 concentrations and body weights of the overweight and obese women. The greater amounts of adipose tissue and blood vessels (both of which product Met-I_2) in a obese person compared to a normal weight individual justify this relationship. Body weight was not significantly related to the other eicosanoids.

(2) Eicosanoid and Glucose Concentrations: Fasting glucose concentrations were inversely related to serum PGE_1 concentrations (inhibits platelet aggregation). Postprandial glucose concentrations were positively associated to serum PGE_2 and plasma TXB_2 concentrations (promotes platelet aggregation). In this respect, elevated circulating glucose concentrations may be associated with increased eicosanoids concentration, and these in turn, with the development of certain disease states such as diabetes and cardiovascular disease. Studies have addressed the effect of blood glucose on eicosanoid production but the results are complex. Further in vivo and in vitro investigations are needed.

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This research would not be possible without the female volunteers in the Body Weight Management Program. Remember, do it in moderation.

APPENDIX A: BODY WEIGHT MANAGEMENT PROGRAM (BWMP)

The Body Weight Management Program (BWMP) was designed to assist overweight college females lose body weight sensibly and maintain weight loss. The philosophy of the BWMP was to modify the eating habits and lifestyle of the participants while they maintained a familiar eating and living environment. This program was directed by the present investigator and offered to the female occupants in an Iowa State University (ISU) residence complex, Maple-Willow-Larch (MWL). The dietary guidance aspect was integrated into the program with the cooperation of the MWL Food Service Facility. The initiation and evaluation of the BWMP were two of the objectives for the dissertation research of this investigator. A third objective was to obtain blood samples from the females for the analyses of prostaglandins (eicosanoids), insulin, and glucose.

The Iowa State University Committee on the Use of Human Subjects in Research reviewed this project and concluded that the rights and welfare of the human subjects were adequately protected, that risks were outweighed by the potential benefits and expected value of the knowledge sought, that confidentiality of data was assured, and that informed consent was obtained by appropriate procedures. Supporting data for this approval are presented in Appendix B.

The integration of the BWMP into a food service facility was unique and had several advantages. The main advantage was that the participants of the program used meal tickets which they had purchased. Essentially, the program provided guidance to a group of free-living

students at no cost to the participants. Many weight reduction programs charge fees for the counseling and monitoring of body weight changes. Also, the University encourages the development of health programs that would benefit both students and employees.

A food service facility offered also a method of controlling food quality and food quantity. These aspects were evaluated during the developmental phase of the BWMP by this investigator. This phase involved the development of a computerized data bank for all of the food items available from the MWL Food Service Facility.

Development of Computerized Data Bank

Computerization of the food choices offered by the food service involved the following protocol. First, the food purchasing facility at ISU identified the food purchased for the food service system at ISU. Once acquainted with this information, it was relatively simple, but tedious, to determine the quality (caloric density) of the food items. For example, the caloric density of meats is based on the lean-to-fat ratio and must be qualified for a weight reduction program. Second, main entree food items are prepared from standardized recipes in the food service. These recipes were obtained and coded accordingly based on data provided in Handbook 456 (1). Third, portion control is well-regulated in a food service facility. For example, a 10x12 inch pan of meatloaf is divided into a specific number of individual servings. Based on these data, a computer printout provided the energy and nutrient contents for individual servings of the food choices available

in the food service. These data were then entered into the WYLBUR computer system.

Daily menus were prepared from the WYLBUR data bank and provided to the participants of the BWMP. An example of a daily menu is presented in the following three pages. The menus listed all of the food choices available from the food service facility for a specific day. Daily food choices with the corresponding caloric density are listed on the menu. Daily intakes ranging from 1200 to 1700 kilocalories were recommended. Suggestions were provided on the menus to assist the participants in maintaining this caloric range. See samples on pages 72, 73, and 74.

Food groups were assigned also to the food choices on the menu. Food groups were determined subjectively based on the amount of carbohydrate, fat, and protein as a percent of the total energy. This information was obtained from the computer printout. The subjects were encouraged to follow a basic four food group plan plus an empty-calorie allowance. The square boxes at the end of the menu indicate the recommended number of servings from each group. In addition, the participants were encouraged to anticipate and annotate the following day's intake before the actual consumption. For example, the dietary intake for Wednesday should be planned on Tuesday. Columns indicating the planned and actual dietary intakes are provided also on the menu. A more complete description of the menu system used in the BWMP is provided in Appendix C.

WEEK: Sample Menu SUBJECT LETTER _____

Code No.	Planned Intake	Menu Item	Food Group	Actual Intake
		B R E A K F A S T		
		Chilled Fruit Juice		
2288	_____	1 serving, Tomato (30 kcal)	Fc	_____
2288	_____	1 serving, V-8 (30 kcal)	Fc	_____
1071	_____	1 serving, Grapefruit (60 kcal)	Fc	_____
27	_____	1 serving, Apple (75 kcal)	Fc	_____
1433	_____	1 serving, Orange (80 kcal)	Fc	_____
		Bacon		
126	_____	2 sl. bacon (100 kcal)	OO	_____
126	_____	1 sl. bacon (50 kcal)	O	_____
		Pancakes w/ Syrup		
59R	_____	1 ladle, 1 pancake (210 kcal)	O & C	_____
1317	_____	Margarine, 1 pat (35 kcal)	O	_____
2049	_____	1 level tbsp. Syrup (50 kcal)	S	_____
		or		
		Toast/English Muffin		
461	_____	1 sl (65 kcal)	C	_____
461	_____	2 sl (130 kcal)	CC	_____
1317	_____	Margarine, 1 pat (35 kcal)	O	_____
1149	_____	Jelly/Jam, 1 tbsp. (50 kcal)	S	_____
		Cereal		
	_____	1 box, Cold (100 kcal)	C	_____
	_____	4 oz., 1 ladle, Hot (100 kcal)	C	_____
		L U N C H		
		Potato Soup		
20R	_____	4 oz., 1 ladle (90 kcal)	1/4C & 1/4D	_____
		Chipped Beef and Cheese Sandwich		
461	_____	2 sl buttered bread (130 kcal)	CC & OO	_____
1314				
653	_____	1 sl cheese (80 kcal)	1/2M or 1/2D	_____
56R	_____	1 oz., 1 scoop C.B. Mix (45 kcal)	1/4M & 1/2O	_____
		or		
		Pork Chow Mein on Rice		
32	_____	1/4 c., 1 scoop rice (70 kcal)	1/2C	_____
93R	_____	1 ladle chow mein (235 kcal)	M & O	_____
	_____	Salad Bar		_____

WEEK: SUBJECT LETTER _____

Code No.	Planned Intake	Menu Item	Food Group	Actual Intake
		D I N N E R		
		Salisbury Steak - Bacon Wrapped (NO GRAVY)		
370	_____	4 oz., 1 serving (310 kcal)	M & O	_____
126	_____	1 sl bacon (50 kcal)	O	_____
		or		
		Veal Parmegiana - Breaded Veal Cutlet w/ Sauce and Cheese		
2394	_____	2.8 oz., cooked veal (175 kcal)	M & O	_____
		(REMOVE CHEESE, BREADING & CHEESE)		
90R	_____	Veal w/ sauce and cheese (310 kcal)	M & OO & S	_____
		O'Brien Potatoes (OMIT)		
	_____	1 serving (300 kcal)	1/2C & OOOO	_____
		Green Bean Casserole		
33R	_____	1/4 c., 1 scoop (75 kcal)	1/2V & 1/2O	_____
		Cooked Beets		
	_____	1/4 c., 1 scoop (15 kcal)	1/2V	_____
	_____	Salad Bar		_____
		Milk, skim		
1322	_____	4 oz., (1/2 c) (45 kcal)	1/2D	_____
	_____	8 oz., (1 c.)(90 kcal)	D	_____
		Other Additions to Menu/Snacks/Alcohol		

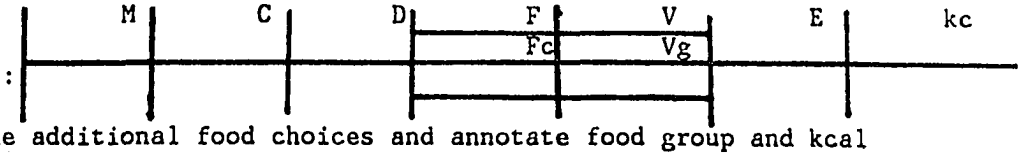
Serving Difficulties:

Comments:

Procedure for Planning and Calculating the Dietary Intake

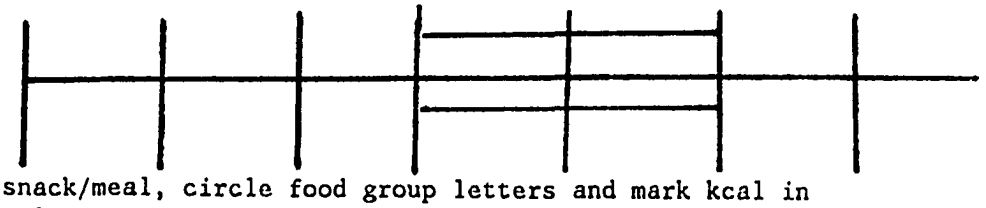
Step 1: Review menu.
Step 2: Decide on food choices and annotate food group and kcal in Plan. Int. column

Step 3: Totals:
Step 4: Subtract:



Step 5: Determine additional food choices and annotate food group and kcal in Plan. Int. column.

Step 6: Add steps 3 & 5:



Step 7: Following snack/meal, circle food group letters and mark kcal in Act. Int. col.

Daily Totals

Meat	Cereal	Dairy	Vege.	Vege.-gr.	Fruit	Fruit-C	Empty	Kcal
(2)	(4)	(2)	(1)	(1) (3-4x/wk)	(1)	(1)	(max 4)	1200-1700

Breakfast Weigh-in Meetings

The body weights of the participants were monitored on a weekly basis at Andrews House, the metabolic unit at ISU. After the weights of the females had been recorded, a self-service continental breakfast was provided. In addition to the emphasis on weight reduction, the BWMP included an educational program, primarily stressing health and wellness. The breakfast weigh-in meetings provided the opportunity to discuss a variety of topics on a group basis (Table A-1). Individual counseling sessions were available to the females on a weekly basis.

Pilot Study

A pilot study was conducted to determine the effectiveness of the dietary phase of the BWMP. Ten, moderately overweight females volunteered to participate in the 4-week study (April-May, 1982). Changes in the body weights of the females are presented in Table A-2. Weight loss ranged from -0.6 to -5.8 kg with a mean weight loss of -3.4 kg during the 4-week period. Five of the females participated in the long-term BWMP experimental study, 1982-1983.

Long-Term Experimental Study, 1982-1983

Description of participants

The BWMP for the 1982-1983 school year began in September based on the encouraging results from a pilot study in May. Of the 78 applicants, 29 females were selected to participate in the dietary counseling and blood sampling collection phases of the study. Five

TABLE 9. Discussion and workshop sessions presented during the Body Weight Management Program

Anthropometric Measurements
Body Composition During Body Weight Changes
Body Frame Assessment
Recommended Dietary Allowances
Height and Weight Tables
Basal Metabolic Rate
Exercise
Snacking
Caloric Density
Eating Behavior Modification
Mental Modification
Cooking/Shopping Tips
Vitamin/Mineral Supplements
Hypertension
General Health and Wellness

females (Subjects B, D, BB, T, and U) dropped out of the program within a couple of months. An additional four females (Subjects G, A1, A2, and B1) enrolled in the program in November, 1982. The BWMP ended in April, 1983, after the final blood collection period.

A general description of the 33 participants in the BWMP is given in Table A-3. All were Caucasian females with relative body weights ranging from 106 to 152%. The body mass index [Wt (kg)/Ht (m)1.5] ranged from 29 to 40. The subjects were in good physical health as certified by a physician at the ISU Student Health Services prior to enrollment in the program. Most of the participants were in good academic standing, maintaining at least a 2.5 grade point average out of a possible 4.0. All of the subjects were non-smokers. No one was taking any type of medication throughout the study period. Only about one-half of the participants had regular menstrual cycles during the 7-month period.

Other characteristics of the 33 subjects are shown also in Table A-3. The majority of the subjects (88%) indicated in a personal interview that they had lower-body-obesity compared to upper-body-obesity. Forty-four percent reported being overweight before menarche. The remainder of the subjects (56%) had become heavier following the onset of their menstrual cycle (around 10 to 12 years of age). Again, this information was obtained from each participant in a personal interview by this investigator. Their resting metabolic rates (Spirometer, Warren E. Collins, Inc., Boston, MA) ranged from 1111 to 1853 kcal/24 hours. Dietary intakes were not analyzed in this study.

TABLE 10. Body weight changes for ten females in the Body Weight Management Program four-week pilot study (April - May, 1982)

Subject	Age (years)	RBW[a] (%)	Body Weights (Kg)		
			Initial (I)	Final (II)	Change (I-II)
100 (DD)[b]	19	162	83.8	78.0	- 5.8
101	21	111	67.3	63.8	- 3.5
103 (FF)	21	129	75.8	71.3	- 2.5
104 (Y)	19	135	80.1	75.9	- 4.2
105	19	112	63.3	61.8	- 1.5
106	20	119	62.7	62.1	- 0.6
107	20	114	67.9	64.1	- 3.8
109 (E)	20	114	71.2	67.0	- 4.2
110 (N)	19	132	77.2	74.8	- 2.4
111	21	118	78.2	75.9	- 2.3
$\bar{X}$	20	125	72.8	69.5	- 3.1
SEM	0.32	5.06	2.31	2.02	0.47

[a]Relative body weights (%) were calculated by $\frac{\text{actual body weight}}{\text{desirable body weight}} \times 100$. Desirable body weights were based on the Hathaway and Foard (2) measurements.

[b]Letter designations in parentheses indicate the respective participant who continued in the long-term experimental study (September, 1982 to May, 1983).

Many of the participants had a family history for obesity (n=21), diabetes (n=4), and hypertension (n=12) (Table A-4). The majority of the participants (n=21) reported that at least one immediate family member was overweight by at least 25 pounds. Diabetes (adult-onset) and hypertension were present also among the family members of a number of the participants. This information was also self-reported.

Changes in body weight

Changes in the body weights of the participants in the BWMP are presented in Table A-5. Initial body weights in September ranged from 60.3 to 91.5 kg with a mean weight of 72.8 kg. Thirteen weeks later in December, six females had lost about one-half of the weight they had desired. These weight losses ranged from -3.4 to -9.1 kg. Upon completion of the BWMP in April, body weights ranged from 56.1 to 96.2 kg with a mean weight of 71.9 kg. This resulted in only a mean weight loss of 0.9 kg for the entire study period. These weights were recorded on the blood collection days at the Student Health Services.

The lowest body weights achieved by the participants during the BWMP are included also in Table A-5. These body weights were recorded at the weekly breakfast weigh-in meetings. It is evident that all of the participants except one female (subject E) lost weight at some point during the program. For the majority of the females, these losses were not maintained.

TABLE 11. Description of females in the Body Weight Management Program experimental study, 1982-1983.

Subject	Age (year)	Height cm (in)	IBW[a] [kg (in)]	RBW[b] (%)	RBW[c] (%)	BMI[d]	Obesity[e]	Onset[f]	RMR[g] (kcal/ 24 hr)
A1	19	176.8 (69.6)	68.2 (150.0)	106	96	29	upper	post	1207
E	21	174.0 (68.5)	67.0 (142.4)	107	97	29	lower	post	1631
C	18	164.6 (64.8)	62.2 (136.8)	111	99	29	lower	post	1485
L	19	160.3 (63.1)	60.3 (132.7)	112	99	30	lower	post	1584
I	20	167.6 (65.0)	63.6 (139.9)	112	100	29	lower	post	1415
K	20	171.3 (67.4)	67.8 (149.2)	112	100	30	lower	post	1267
A2	19	174.7 (68.7)	69.9 (153.8)	112	101	30	lower	post	1111
B	19	162.8 (64.1)	62.6 (137.7)	113	101	30	lower	post	1234
B1	19	167.6 (66.0)	66.0 (145.2)	113	101	30	upper	post	1111
D	18	165.2 (65.0)	64.7 (142.3)	114	101	30	lower	post	1468
H	19	168.2 (66.2)	67.3 (148.1)	115	103	31	lower	post	1630
J	21	156.7 (61.7)	61.6 (135.5)	119	106	31	lower	post	1285
M	21	167.0 (65.7)	68.4 (150.5)	119	106	32	lower	post	1267
P	21	173.7 (68.4)	74.3 (163.5)	119	108	33	lower	post	1500
S	19	177.7 (70.0)	81.3 (178.9)	125	113	34	lower	pre	1853
FF	22	167.8 (66.1)	73.8 (162.4)	126	113	34	lower	post	1482
Q	20	161.5 (63.6)	68.7 (151.1)	126	113	34	lower	pre	1354
U	18	169.5 (66.7)	74.9 (164.8)	127	114	34	lower	pre	1593
O	20	170.7 (67.2)	77.3 (170.1)	129	115	35	lower	pre	1494
T	19	154.5 (60.8)	65.5 (144.1)	130	115	34	lower	pre	1633
R	18	161.2 (63.5)	70.5 (155.1)	130	115	35	lower	post	1350
X	20	169.5 (66.7)	76.8 (169.0)	130	116	35	lower	pre	1578
N	19	168.2 (66.2)	77.3 (170.1)	132	118	35	lower	post	1402
Y	19	169.8 (66.9)	79.0 (173.8)	132	118	36	lower	pre	1676
W	20	164.9 (64.9)	76.0 (167.2)	134	119	36	lower	post	1723
Z	21	167.6 (66.0)	79.6 (175.1)	136	122	37	upper	pre	1606
V	19	167.9 (66.1)	81.0 (178.2)	138	124	37	lower	post	1639
AA	20	164.9 (64.9)	80.8 (177.8)	142	127	38	lower	pre	1594
G	20	164.6 (64.8)	81.7 (179.7)	145	129	39	lower	post	unk

BB	19	173.4 (68.3)	91.5 (201.3)	147	132	40	lower	pre	1845
EE	19	167.6 (66.0)	87.6 (192.7)	150	134	40	lower	post	1368
CC	21	153.9 (60.6)	76.5 (168.3)	152	134	40	upper	pre	1348
DD	19	156.7 (61.7)	78.6 (172.9)	152	134	40	lower	pre	1843

[a]Initial body weights (IBW) were recorded on the first blood collection period (Sept. '82) at the Student Health Services.

[b]Relative body weights (%) were calculated by $\frac{\text{actual body weight}}{\text{desirable body wieght}}$ X 100. Desirable body weights were based on the Hathaway and Foard (2) measurements.

[c]Relative body weights (%) were calcualted as described in footnote "b" above. Desirable body weights were based on the HANES (3) measurements which were estimated from regression equation of weight on height for women aged 20-29 years.

[d]Body Mass Index, W/H (in $Kg/m^{1.5}$).

[e]Obesity refers to pattern of body fat. Lower indicates a lower-body-obesity (fat is predominantly on the hips and thighs) and upper refers to upper-body-obesity (fat is distributed mostly above the waist). This information was obtained from a personal interview with each participant of the BWMP.

[f]Onset refers to premenarche (overweight during childhood) and post menarche (excess weight following the onset of the menstrual cycle).

[g]RMR denotes resting metabolic rate. Standard procedures (4) were used to obtain these 2-hour postprandial measurements.

Discussion

The preoccupation with body weight continues to elicit universal interest and personal concern. Centuries ago, an overweight state was considered a status symbol and an object of artistic representation. But times have changed especially with the advent of Twiggy; a social stigma has become attached to obesity. Ayers (5) has presented the changing views toward overweight and obesity from the Greco-Roman times to the twentieth century. These are the esthetic views on body shape and form; health aspects are often overlooked.

The Health and Nutritional Examination Survey (HANES) data (3) indicate that a large percentage of Americans exceed the recommended weights. Approximately, one-fifth (1.7 million) of females, 20-24 years of age, are 10% or more above the suggested weights for their heights. For females in the same age group, about one-tenth are 20% or more above the recommended weights. A greater percent of women than men in the same age groups have body weight above the suggested ranges. The percent deviations from the recommended weights for either sex increase with age.

Weight reduction programs continue to proliferate into the public sector; they are lucrative business enterprises. When a new program is introduced, clientele invest hundreds and thousands of dollars in hopes that finally a "miracle" program has been discovered. Some of these "miracle" diets, such as the liquid-protein-diet, have provend lethal to some poorly-counseled patrons (6). Moreover, the dietary recommendations of these faddish programs do not produce favorable

TABLE 12. Presence of obesity, adult-onset diabetes, and high blood pressure in family members of the females in the Body Weight Management Program[a]

Subject	Obesity	Diabetes	Hypertension
A1			
E	M/F/S		
C	GF		
L	F		
I			F
K	F/GF	F	
A2			F
B	M/B		B
B1			F
D			
H	F/S		M
J	GM	GM	M
M			GF/F
P	GF		GM
S	GF		GM
FF	M/F/S/B		M
Q			
U	F	U	
O		GF	
T	S		
R	M/F		
X			
N	M/F		
Y	M/F/B		
W			
Z	M/F		F
V	F		
AA	M/F/B		F
G			
BB	M/F/S/GF	GF	
EE	F	U	
CC		GF	
DD	M	A	

[a]A personal interview was conducted with each participant in the program concerning the presence of these disease states among family members. Thus, the presence of these states is subject to individual interpretation. Obesity was defined as 25 pounds or more. Diabetes and a hypertensive state prevailed when dietary and/or drug treatments were required. The following abbreviations indicate the presence of these disease states among family members: F=father, GF=grandfather, M=mother, S=sister, GM=grandmother, A=aunt, U=uncle, and B=brother.

lean/fat (body compartments) ratio (7). The most undesirable outcome is that weight losses achieved are seldom maintained permanently (8-10).

The basic goals of a sensible weight reduction program are (1) to decrease caloric intake, (2) to increase caloric expenditure, and (3) to establish new dietary and lifestyle habits that will maintain the body weight loss. The first two objective should be met in a moderate fashion and adequate nutrient intakes must be assured. A caloric intake below 1200 kcal/day is not recommended; below this level, the Recommended Dietary Allowances (11) for essential nutrients are difficult to meet. Additionally, a daily physical exercise pattern needs to be adopted, although it need not be a strenuous regimen. During the actual weight reduction period, the individual should be acquiring new dietary and lifestyle habits to ensure maintenance of body weight loss. Most dieters fail in this area (9, 10). Usually, individuals are motivated to lose weight during the initial dieting period but many fail to make a lifetime commitment to modify their lifestyle. A strong individual commitment is imperative if body weight is to be properly controlled over a lifetime. One question raised is: How do you guide individuals to make such a commitment?

The weight loss patterns for the majority of the females in this study are disappointing. They are, however, realistic and similar to those observed in other weight reduction programs. Bray (8) has evaluated the body weight patterns of obese individuals from long-term studies. He found that 10 to 20% of the "dieters" will maintain their weight loss or continue to lose weight, but few will actually reach

TABLE 13. Changes in body weight[a] and lowest body weight achieved[b] for the females in the Body Weight Management Program

Subject	Body Weight (Kg)						
	Initial (Sept. '82; I)	Intermediate (Dec. '82; II)	Change (I-II)	Final (Apr. '83; III)	Change (I-III)	Lowest (IV)	Change (I-IV)
A1	68.2			67.7	- 0.5	66.9	- 1.3
E	67.0			74.8	+ 7.8	67.5	+ 0.5
C	62.2			62.0	- 0.2	56.1	- 6.1
L	60.3	55.8	- 4.5	56.1	- 4.2	55.8	- 4.5
I	63.6	59.7	- 3.9	61.0	- 2.6	59.5	- 4.1
K	67.8	64.0	- 3.4	68.8	+ 1.0	63.8	- 4.0
A2	69.9			67.8	- 2.1	65.5	- 4.4
B	62.6					59.9	- 2.7
B1	66.0			69.4	+ 3.4	65.5	- 0.5
D	64.7					54.6	-10.6
H	67.3					66.4	- 0.9
J	61.6			58.2	- 3.4	55.6	- 6.0
M	67.4					65.7	- 1.7
P	74.3			72.9	- 1.4	67.6	- 6.7
S	81.3			81.9	+ 0.6	75.3	- 6.0
FF	73.8					69.3	- 4.5
Q	68.7	63.0	- 5.7	64.5	- 4.2	61.8	- 6.9
U	74.9					72.9	- 2.0
O	77.3	68.2	- 9.1	80.0	+ 2.7	66.6	-10.7
T	65.5					63.9	- 1.6
R	70.5			66.7	- 3.8	64.4	- 6.1
X	76.8	70.3	- 6.3	74.2	- 2.6	69.3	- 7.5
N	77.3			76.1	- 1.2	72.4	- 4.9
Y	79.0					75.8	- 3.2
W	76.0					69.5	- 6.5
Z	79.6			75.5	- 4.1	73.9	- 5.7
V	81.0			75.7	- 5.3	73.6	- 7.4

AA	80.8			79.5	- 1.3
C	81.9	86.7	+ 5.0	80.4	- 1.3
BB	91.5			89.0	- 2.5
EE	87.6	96.2	+ 8.6	87.1	- 0.5
CC	76.5			70.7	- 5.8
DD	78.6	72.7	- 5.9	71.7	- 6.9
$\bar{X}$	72.8 ±	71.9 ±		68.4 ±	
SEM	1.38	1.66		1.45	

[a]Body weights (periods I, II, and III) were recorded during a fasting state on the blood collection days at the Student Health Services.

[b]Lowest refers to the lowest body weight achieved (period IV) by each female during the entire Body Weight Management Program. These weights were recorded at Andrews House during the weekly breakfast weigh-in meetings.

their recommended weights. Unfortunately, the remaining dieters will return to their pre-reduction weights, and some will exceed their initial levels.

At the onset of the study, the participants were enthusiastic and highly motivated to lose weight. Most of the participants had tried other weight reduction programs, such as Weight Watchers or some faddish dietary regimen. Their earlier attempts to lose weight and most important, to maintain the weight loss, were unsuccessful. The students enrolled in the BWMP with the idea of 'alas, this one (program) might work.' They had, however, been warned against this type of attitude. The philosophy of the BWMP was based on weight reduction by moderate and sensible changes in dietary and lifestyle habits. It was repeatedly stressed that these modifications would need to be continued for a lifetime in order to maintain a desirable body weight.

The lowest body weight achieved for most of the participants occurred before the Christmas school break in December. During this 30-day break, these young women were encouraged to maintain their weight losses and not attempt further weight reduction. A mean body weight gain of about 2 kg (about 4.5 lb) occurred during the break which was fairly encouraging. The body weight changes, however, ranged from 0.0 kg to +9.6 kg (21.14 lb).

After the holiday break, further weight loss was not achieved by the majority of the females. In fact, most of the body weights returned to the pre-reduction levels (Table A-5). In three cases (Subjects E, G, and EE), the final weights exceeded the pre-reduction body weight by at

least 5.0 kg (11 lb) (Table A-5, Change I-III column).

Despite attempts to motivate the participants, many of the females lost their drive to lose weight after the first couple of months in the program. Several women requested that daily exercise classes be initiated into the program. Some of the participants took advantage of these free classes under the direction of the present researcher. Most of the women, however, found excuses for not participating; these same individuals were the ones who had requested that a scheduled exercise class be incorporated into the program. Not only were the participants to modify dietary intakes, but the BWMP encouraged them to adopt a daily exercise activity pattern. As mentioned previously, no charges were incurred by the participants in the BWMP. All of the services including dietary guidance on an individual and a group basis, weekly breakfast weigh-in meetings, daily computerized menus, and exercise classes were free-of-charge for the females.

Generally, the lighter-weight-females were more difficult to motivate than the heavier participants. This observation is in agreement with an evaluation conducted by Seaton and Rose (12). They found that the frequency of defaulters from weight reduction clinics was greater among the lighter-weight individuals compared to the heavier subjects.

The body weight changes for Subject O (relative body weight of 129%) were particularly disappointing (Figure A-1). During the 13 weeks prior to Christmas break, this female lost 10.4 kg (23.8 lb). This weight loss was achieved by the combination of a decreased energy intake

and an increased energy expediture. During the 30-day term break, she gained 9.6 kg (21.1 lb). She stated that she was making up for "lost time," although she did feel badly about the weight gain. At the end of the BWMP, she weighed 78.2 kg (172 lb), which was +0.8 kg (1.8 lb) more than her initial body weight.

One objective of this research was to evaluate the effectiveness of a nutritionally sensible weight reduction program for young college women. On a group basis, weight loss did not appear to be a good criterion to use. A mean weight loss of only 0.9 kg was calculated for the entire group (n=21); final body weight changes ranged from -5.9 kg (13.0 lb) to +8.6 kg (18.9 lb) (Table A-5, Change I-III column). For the majority of the females, the weight loss was of short duration (September through December). These body weight losses ranged from -10.7 kg (23.5 lb) to -0.5 kg (1.1 lb). (Table A-5, Change I-IV column).

The cyclic loss and regain of body weight has been popularly termed the 'yo-yo syndrome.' The mechanisms underlying the cyclic weight patterns are unknown (13). Granted, everyone can lose weight to varying degrees. What is the merit, however, of weight reduction without maintenance of the weight loss? The maintenance of a desirable weight level is achieved only by a continual modification of lifestyle habits (dietary and physical activity) throughout a lifetime. It should be mentioned, however, an irreversible increase of adipocyte number in some obese individuals might make it difficult to maintain weight loss (13).

Most of the students had modified some aspect of their eating

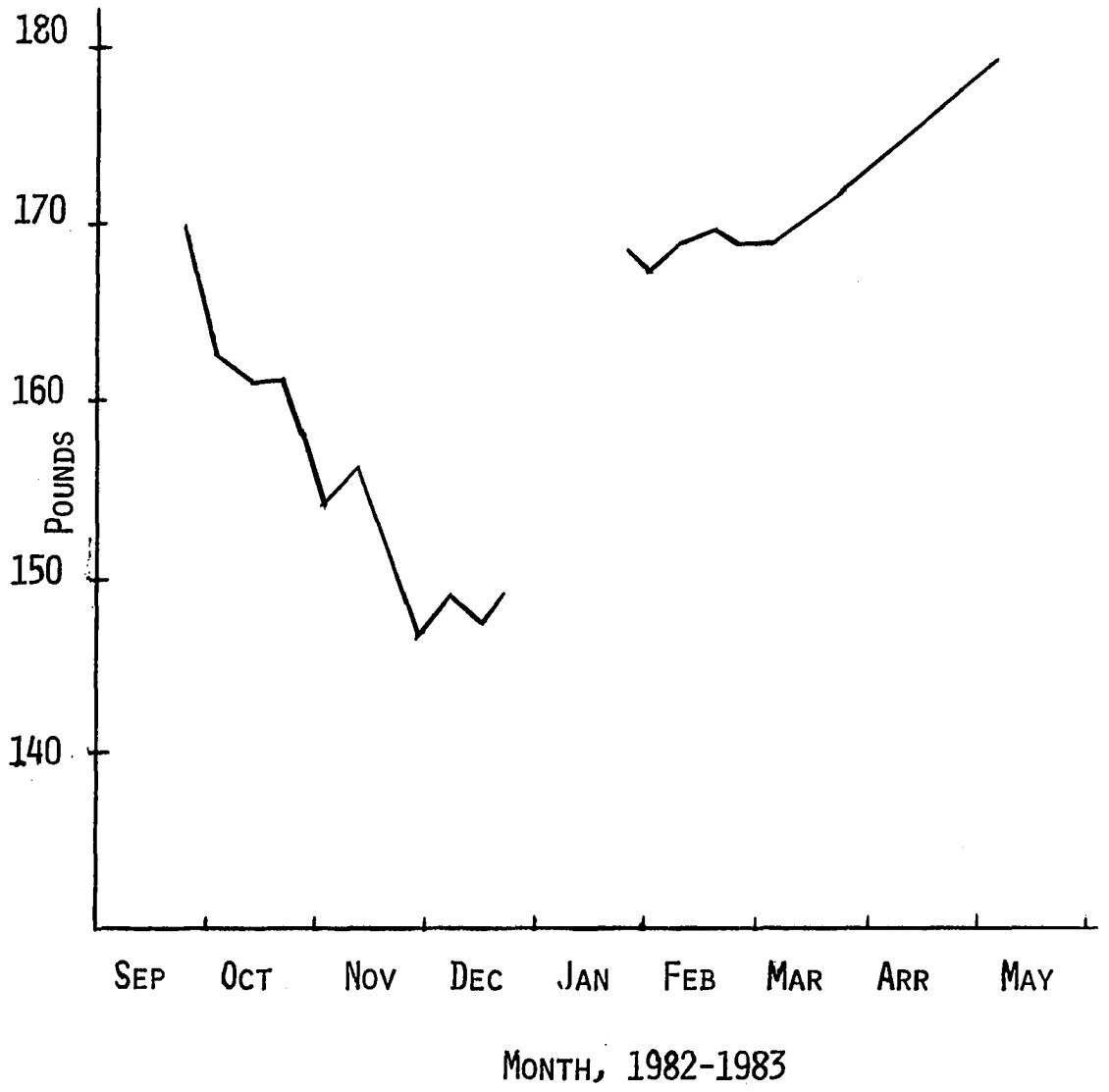


FIGURE 5. Body weight pattern for Subject O (initial relative body weight of 129%) in the Body Weight Management Program

pattern during the BWMP. A questionnaire at the end of the study indicated that the most common modifications were the omission of (1) salad dressings, (2) fried foods, and (3) margarine on potatoes and vegetables. In addition, most of the participants learned to use a trade-off system for their dietary intake selections. In this sense, you cannot have your cake and beer(s) too.

Techniques are not available that predict those individuals who will lose weight successfully in weight reduction program. In addition to caloric restriction with or without exercise, there are many different types of treatment for obesity. Some of these include total starvation, jaw wiring, behavior techniques, and surgical intervention. It is, however, not known which approach would be the best method for a specific individual.

The body weight reduction results of the BWMP appear dismal on a group basis. About 30% of the females (Subjects L, J, R, V, Z, DD, Q, T, W, Z), however, did achieve a weight loss of about 4.0 kg or more (about 9 lb). Most notably was the weight loss of Subject DD, one of the most overweight females (relative body weight of 162%). She was one of the original participants in the BWMP pilot study and elected to continue in the experimental study phase. During this one-year period, she lost 11.1 kg (about 25 lb) (Figure A-2). This decrease in weight was certainly a rewarding result and served to justify the BWMP. Moreover, she has maintained this weight loss for about 6 months (personal communication, ISU). The maintenance of weight loss can be achieved by modification of lifestyle, such as regulation of both

dietary intake and physical activity. Only a few, however, are successful.

In summary, this program illustrated realistic body weight loss patterns for females involved in a weight reduction program. The females who did achieve a weight loss and the maintenance of the loss made the BWMP a successful endeavor. More important, the participants realized the need for nutritionally sensible eating habits.

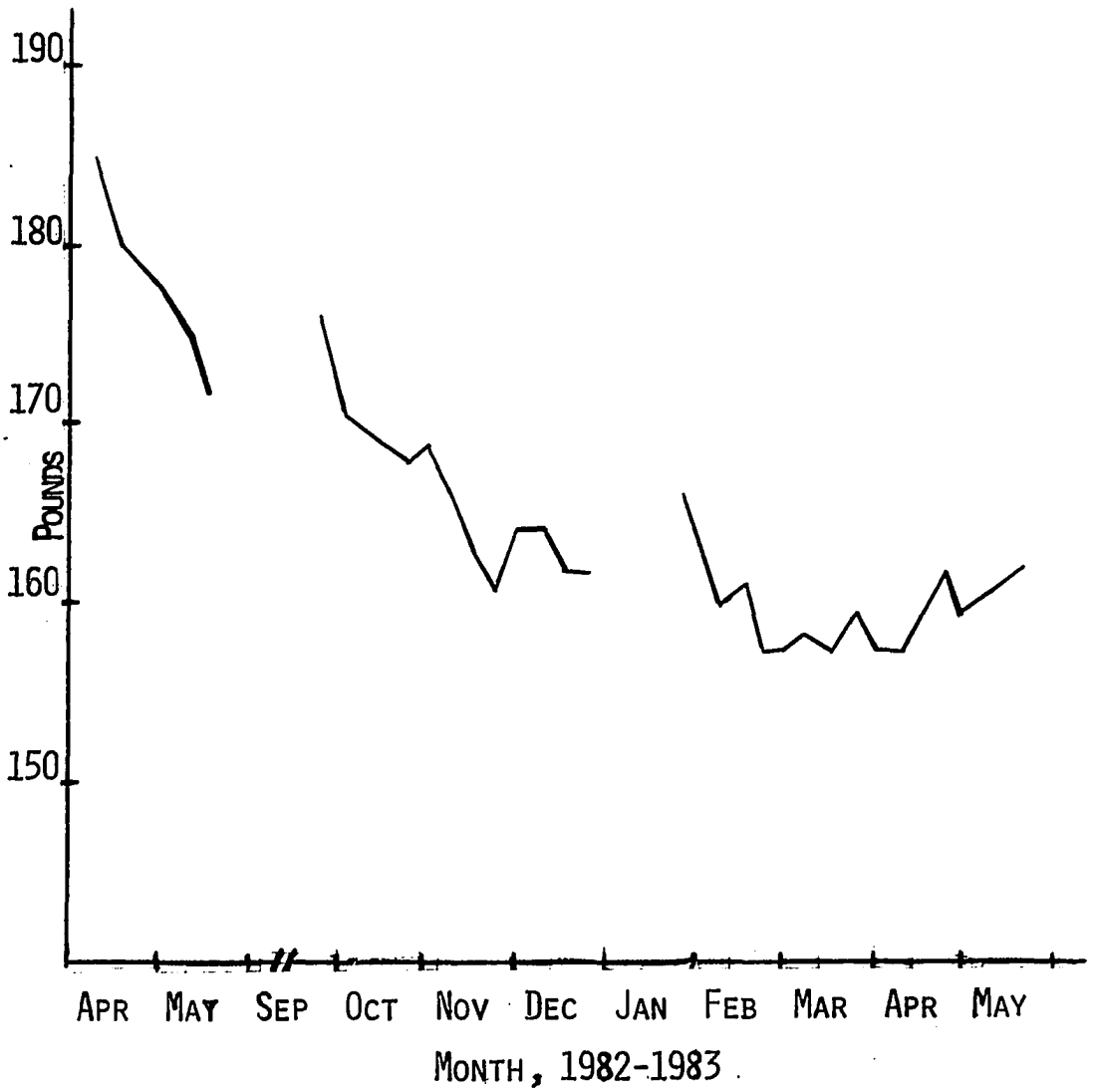


FIGURE 6. Body weight pattern for Subject DD (initial relative body weight of 162%) in the Body Weight Management Program

APPENDIX B. INFORMATION ON THE USE OF HUMAN SUBJECTS IN RESEARCH

INFORMATION ON THE USE OF HUMAN SUBJECTS IN RESEARCH
IOWA STATE UNIVERSITY

(Please follow the accompanying instructions for completing this form.)

1. Title of project (please type): Body Weight Management Program for Moderately Obese Young Women

2. I agree to provide the proper surveillance of this project to insure that the rights and welfare of the human subjects are properly protected. Additions to or changes in procedures affecting the subjects after the project has been approved will be submitted to the committee for review.

Barbara Struempler	3/11/82	Barbara Struempler
Typed Named of Principal Investigator	Date	Signature of Principal Investigator

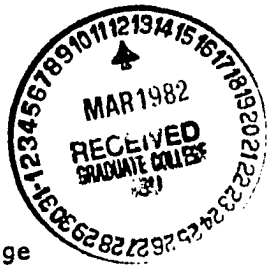
106 MacKay Hall	294-7316
Campus Address	Campus Telephone

3. Signatures of others (if any)

Signatures of others (if any)	Date	Relationship to Principal Investigator
Allen G. Garcia	3/10/82	Major Professor

4. ATTACH an additional page(s) (A) describing your proposed research and (B) the subjects to be used, (C) indicating any risks or discomforts to the subjects, and (D) covering any topics checked below. CHECK all boxes applicable.

- [X] Medical clearance necessary before subjects can participate
- [X] Samples (blood, tissue, etc.) from subjects
- [ ] Administration of substances (foods, drugs, etc.) to subjects
- [ ] Physical exercise or conditioning for subjects
- [ ] Deception of subjects
- [ ] Subjects under 14 years of age and(or) [ ] Subjects 14-17 years of age
- [ ] Subjects in Institutions
- [ ] Research must be approved by another institution or agency



5. ATTACH an example of the material to be used to obtain informed consent and CHECK which type will be used.

- [X] Signed informed consent will be obtained.
- [ ] Modified informed consent will be obtained.

6. Anticipated date on which subjects will be first contacted: Month March, Day 22, Year 1982

Anticipated date for last contact with subjects: Month May, Day 15, Year 1983

7. If Applicable: Anticipated date on which audio or visual tapes will be erased and(or) identifiers will be removed from completed survey instruments: Month ____ Day ____ Year ____

8.

Signature of Head or Chairperson	Date	Department or Administrative Unit
Jacqueline Vaughn	3/11/82	Food and Nutrition

9. Decision of the University Committee on the Use of Human Subjects in Research:

- [ ] Project Approved
- [ ] Project not approved
- [ ] No action required

George G. Karas		
Name of Committee Chairperson	Date	Signature of Committee Chairperson

Revised 6/78

Year - 1981-83

INFORMATION TO SUBJECTS

IOWA AGRICULTURE AND HOME ECONOMICS EXPERIMENT STATION

Project 2213: Metabolic Patterns of Obese Women

Body Weight Management Program for Moderately Obese Young Women

Objectives: The purposes of this study are:

(a) to set-up and assess the effectiveness of a body weight management program in an ISU residence hall for moderately obese college females

(b) to study the relationship of fasting (12 hours after food consumption) and postprandial (1 hour after food consumption) levels of plasma and serum prostaglandins and plasma insulin and glucose levels among the program participants.

Principal Investigators:

Barbara Struempler, program director;
Pilar A. Garcia, project leader;
Assisted by Kathy Morrison and Ruth Smith, Human Nutrition Research Staff and Food and Nutrition students (enrolled in practicum courses).

Office Phones: MacKay Hall 34A 4-7316
Andrews House 4-7336

Department: Food and Nutrition, Human Nutrition Research

Cooperation: Dr. Donald Hotchkiss, statistical consultant, Statistical Laboratory
Dr. Katherine Schneider, Consultant on eating behavior, ISU Student Counseling Service
Dr. L.Z. Furman, M.D., medical consultant, ISU Student Health Service

Description of the Body Weight Management Program Study

I. Background: Current dietary guidelines for Americans emphasize the attainment and maintenance of desirable weight. Excess fat or obesity in many people is assoicated with increased incidence of degenerative disease. Evidence suggest that for the obese, the best preventive measure against cardiovascular disease is weight reduction. Recent work has also suggested an imbalance in prostaglandin production in obesity which may further increase the risk of developing atherosclerosis and adult-onset diabetes in the obese individual. Prostaglandins are derivatives of essential fatty acids and they are important regulators of many biological processes such as blood pressure, smooth muscle contractility and blood clotting. It has been reported that prostaglandins negatively feedback in the glucose-induced insulin secretion in humans and that an obese individual may overproduce or be hypersensitive to prostaglandins. A study of this nature should increase our understanding of the role of the prostaglandins in the management of obesity and weight maintenance following weight loss.

II. Subjects: Subjects participating in the body weight management program must meet the following criteria: (a) female, (b) 18-22 years of age, (c) body weight of 120-150% relative weight, (d) good health except for moderate obesity, as evidenced by a physical examination at the University Student Health Service, (e) no record of use of oral contraceptives or other medications, such as aspirin which would confound research data, (e) resident of Maple-Willow-Larch (M-W-L) dormitory complex and will consume meals that are served in the M-W-L Food Service facility, (f) good academic standing to manage additional load imposed by the study.

A pre-study physical examination will be given and the program will be under continuous medical supervision.

III. Period of Study:

Preliminary Study:

March - May 14, 1982 Observation Period and recruitment.
May - August 1982 Recruitment continued; further work on methodology.

The main objective of the preliminary study is to monitor the feeding phase of the body weight management program.

Ten moderately obese female volunteers will participate in the body weight management program. Anthropometric measurements and blood samples will not be obtained during this period.

Experimental Study: September 1982-May 1983

The feeding phase of the body weight management program will consist of a 5-day orientation period, followed by a restricted caloric intake (approximately 1000 kcal/day deficit) period. The restricted dietary intakes will range from 1200-1700 kcal/day. It is hoped that enough subjects achieve relative weights equivalent to one-half of the

initial value and can be monitored for approximately three months during a weight maintenance period. The physical activity pattern adopted during this period will be monitored.

1. Dietary Regime: The participants in the program will continue to consume meals that are served in the Maple-Willow-Larch Food Service facility throughout the study. During the weight reduction period, the calorie intake of each subject will be adjusted to provide an approximate 1000 kcal deficit per day. Following the weight reduction period, the calorie intake of each subject will be adjusted to permit weight maintenance. Food choices will be closely monitored to help participants achieve their desirable weight goals.

   During university holidays, term breaks, and/or weekends : when meals are not eaten at the M-W-L Food Service facility, the subjects will record dietary intakes according to the instructions provided by the Food and Nutrition research staff.

2. Blood Prostaglandins (PG), Insulin and Glucose: The blood samples (20 ml each time) will be drawn by a registered medical technologist at the Student Health Service under stringent aseptic conditions.

   Fasting (12 hours after food consumption) and postprandial (1 hour after food consumption) blood samples will be obtained five times during the study for PG, glucose, and insulin analysis: (1) during the orientation period, (2) during the first week of caloric restriction, (3) at a relative weight of 125%, (if initial relative weight is 150%), (4) at a relative weight of 100±10%, and again (5) three months after a relative weight of 100±10%. Arrangements will be made to eat breakfast at the Student Health Service following the fasting blood sample collection.

3. Skinfold Thickness and Anthropometric Measurements: These measurements will be taken concurrently with the blood samples at the Andrews House by a research staff member.

   Skinfold thickness estimates body fat because a large proportion of the fat in an adult body is located under the skin. A constant pressure skinfold caliper is used for measuring skinfold thickness.

   Anthropometric measurements include height, weight, other indices of body frame and musculature. Tools used: platform scale, upright board with metric scale, sliding caliper, stainless steel tape, dynamometer.

4. Resting Metabolism: This standard measurement will be taken during the orientation period at the Andrews House. Measurements of resting metabolism provide estimates of the amount of energy needed by the body in a resting state. The tests will be given about 3 hours before the evening meanl.

5. Physical Activity: Each subject will be encouraged to choose and adopt a pattern of physical activity as part of her program. She will keep a record at specified time intervals.

IV. Risks: Minimum risks are involved in the drawing of blood including possible hematoma formation (similar to a bruise) and infection. Blood will be drawn by a registered medical technologist at the Student Health Service under stringent aseptic conditions minimizing any chance of the above. Some minimal pain is involved in blood drawing but this should be no greater than for any other venipuncture (blood tests) you may have had in past medical work. If a prospective participant has ever fainted or experienced dizziness when blood was drawn she should not volunteer for this study.

V. Benefits: A program participant will have the opportunity to lose weight at a moderate rate while consuming nutritious food chosen on an individual basis. The ability to lose weight while participating in the program should help establish an eating and activity pattern that will allow you to maintain the weight loss. Additionally, the changes in the prostaglandins, glucose and insulin levels during and following the weight reduction period will increase our understanding of the role of the prostaglandins in the management of energy balance. The participant will be given access to results from this study for your own information and benefit.

Withdrawal from the study: A participant may withdraw from the study at any time of her own free will, without giving a reason, or in any open or subtle way, being coerced to remain on the study.

Procedures to Which you as a Subject in this Study Agree -- In addition to the conditions of participation implied or stated in the attached "Information to Subject" I specifically understand and agree to the following:

1. To cooperate willingly, of my own free will, and with a realization of the importance of complete honesty in adherence to all instructions and requirements.

2. To realize that my own personal safety, health and well-being is of the upper most importance to me, and to you, and that we both expect this to supersede all aspects of the study, and that to protect me and for purpose of the study, disclosure of complete medical information to the Physician-in-Charge or her/his associates is necessary. This information will be used by the investigators in the study with every effort made to prevent undue disclosure about me as an individual.

3. If selected as a subject, I will make every effort to adhere to the dietary recommendations on selection of food service menu items provided by the research staff in the study.

4. I will allow blood samples to be drawn by venipuncture from the arm, by a registered medical technician at the Student Health Service at five different occasions (20 ml each time) during the study. Each of the five occasions will consist of two blood drawings, a fasting and postprandial sample.

5. I agree to avoid taking any hormonal preparation during the study; I will not take other medications especially aspirin unless I consult the program director and the Physician-in-Charge or her/his associates. Aspirin inhibits the synthesis of prostaglandins.

6. During the study I agree to have any medical examinations which the Physician-in-Charge may deem prudent or necessary to avoid risk to me from participation in the study.

7. In consenting to participate in this study I agree to have read the "Information to Subjects", understand its contents, and have had it contents explained to me orally. I agree to participate fully and of my own free will in this study as indicated by my signautre on this and the attached "Informed Consent" form.

_____ _____

Signature Witness

_____

Date

INFORMED CONSENT

I, _____, have been informed orally and in writing of the purpose, benefits, and potential hazards of the research project entitled "Body Weight Management Program for Moderately Obese Young Women" which is under the guidance of Barbara Struempler, program director and Pilar A. Garcia, project leader.

I volunteer of my own free will to participate fully in this project. I understand that I will be given further explanation of the project and of specific procedures, if I so desire. I also understand that I may terminate my participation in the project at any time and that I am not waiving my legal rights.

I understand that my records will be treated in a confidential manner as medical records, and that I will be given access to my records during and at the end of this study, if I so desire.

_____
Signature

_____
Witness

_____
Date

MEDICAL CLEARANCE STATEMENT

102

To: Dr. L.Z. Furman, Examining Physician
Student Health Service
Iowa State University
Ames, Iowa 50011

From: Barbara Struempler, Program Director
Body Weight Management Program
106 MacKay Hall
Iowa State University
Ames, Iowa 50011

Date:

We have initiated a body weight management program in an ISU residence hall as part of a research project on moderately obese college females. The feeding phase of the research project has been integrated into the program with the cooperation of the Maple-Willow-Larch (M-W-L) Food Service facility at ISU. Following an orientation period, each subject will adjust her calorie intake to provide an approximate 1000 kcal deficit per day. The restricted dietary intakes will range from 1200-1700 kcal per day. Subjects will remain on the restricted dietary intakes until desired body weight is achieved. Thereafter, the calorie intake of each subject will be adjusted to permit weight maintenance. The body weight management program, its purpose, and procedures to be used are described in the attached statement of Information to Subjects together with the Informed Consent.

To participate in our metabolic study, the female student must meet the following criteria: (a) 18-22 years of age, (b) body weight of 120-150% relative weight, (c) good health, except for moderate obesity, as evidenced by a physical examination at the University Student Health Service, (d) no record of use of oral contraceptives or other medications which would confound research data, (e) resident of Maple-Willow-Larch and uses the M-W-L Food Service facility, (f) good academic standing to manage additional load imposed by the study. Individual will maintain their physical activity pattern during the study and participants will monitor their activities.

_____ meets the criteria given above except
Name of student

for (c) and she has been referred to you for the physical examination.

Please give us your medical recommendation by completing the following:

Please check:

_____ I have no reservation regarding this student's participation in your study.

_____ I have reservations regarding this student's participation in your study. My reservations are as follows:

a. _____

b. _____

c. _____

_____ _____
Signature of Physician Date

Dissertation Research Proposal

Barb Struempler
March 1982
Food and Nutrition Department
Doctor of Philosophy

Proposed Title: Blood concentrations of prostaglandins, insulin, and glucose in overweight college women in a body weight management program

Justification: The discovery of prostaglandins (PGs) has caused researchers to reevaluate neurological and metabolic aspects of many diseases. It is well documented that PGs play an important physiological role in the regulation of lipid mobilization from adipose tissue. Steinberg et al. (1) and Bergström et al. (2) were the first to observe that PGE_1 reduced basal or hormone-stimulated lipolysis in adipose tissue in vitro. It is generally accepted that PGs are involved in the maintenance of adipose tissue homeostasis by a negative feedback inhibition mechanism. In the normal adipose cell, cyclic AMP formation (inducing lipolysis) and PG synthesis are in a finely balanced state. In obesity, PG synthesis is excessive causing a negative inhibition on the cyclic AMP system (reducing lipolysis) at one or more steps. Hence, the mobilization of triglycerides from adipose tissue is "tuned-off" and little, if any release of free fatty acids and glycerol is observed (3).

The insulin-like activities of PGs and their influence on blood insulin and glucose levels in human need evaluation. The majority of the data involving PGs and insulin (excluding diabetes research) consists of studies in vitro of adipose tissue or islets in the presence of a PG and/or glucose media (4, 5). Studies in vivo involve mainly animals such as dogs (6, 7) and sheep(8). The results from these studies are conflicting due to the methodology, the species variability, the concentration of PGs and glucose used in the studies, and the determination of a specific PG series. Extrapolation of data from these studies for human application is difficult but does provide a useful baseline for PG research in obese humans.

The purposes of this research are: (a) to establish and assess the effectiveness of a body weight management program in an ISU residence hall for moderately obese college females, (b) to study the relationship of fasting (12 hours after food consumption) and postprandial (1 hour after food consumption) levels of plasma and serum PGs (E_1, E_2, Thromboxane B_2 and 6-keto-PGF 1α) and plasma insulin and glucose levels among the program participants. It has been reported that prostaglandins negatively feedback in the glucose-induced insulin secretion in humans and that an obese individual may overproduce and be hypersensitive to prostaglandins. Moreover, only 5-25% of individuals on long-term weight reduction maintain weight loss (9). The changes in the PGs, glucose and insulin levels during and following an approximate energy deficit of 1000 kcal/day would increase our understanding of the role of the PGs in the management of obesity and weight maintenance.

Plan of Study: The body weight management program will be initiated in an ISU residence hall. The feeding phase of the research project will be integrated into the program with the cooperation of the Maple-Willow-Larch (MWL) Food Service facility at ISU.

(over)

Thirty overweight females , 18-22 years of age, will be recruited as subjects. The females will be students at ISU and free-living in the MWL dormitory complex. The subjects must consume their meals at the MWL Food Service facility. The subjects will be less or equal to 150% relative weight at the beginning of the study. The body mass index (Wt_{Kg}/Ht_{m} 1.5) will be used to assess body fat (10).

The feeding phase of the research project will consist of an orientation period followed by a restricted caloric intake (approximately 1000 kcal/day deficit) period. The restricted dietary intakes will range from 1200-1700 kcal/day. It is hoped that enough subjects achieve relative weights equivalent to one-half of the initial value and can be monitored for approximately three months during a weight maintenance period. The physical activity pattern adopted during the period will be monitored.

Fasting and postprandial blood samples will be obtained five times (see attached sheet) during the study for PG, glucose, and insulin analysis: (1) during the orientation period, (2) during the first week of caloric restriction, (3) at a relative weight of 125%, (if initial relative weight is 150%), (4) at a relative weight of 100±10%, and again (5) three months after a relative weight of 100±10%. Anthropometric measurements (skinfold and girth) will be measured concurrently.

Data on fasting and postprandial serum PGs and plasma insulin and glucose will be analyzed statistically with regard to the changes in body composition in terms of fat and lean body mass.

Literature Cited

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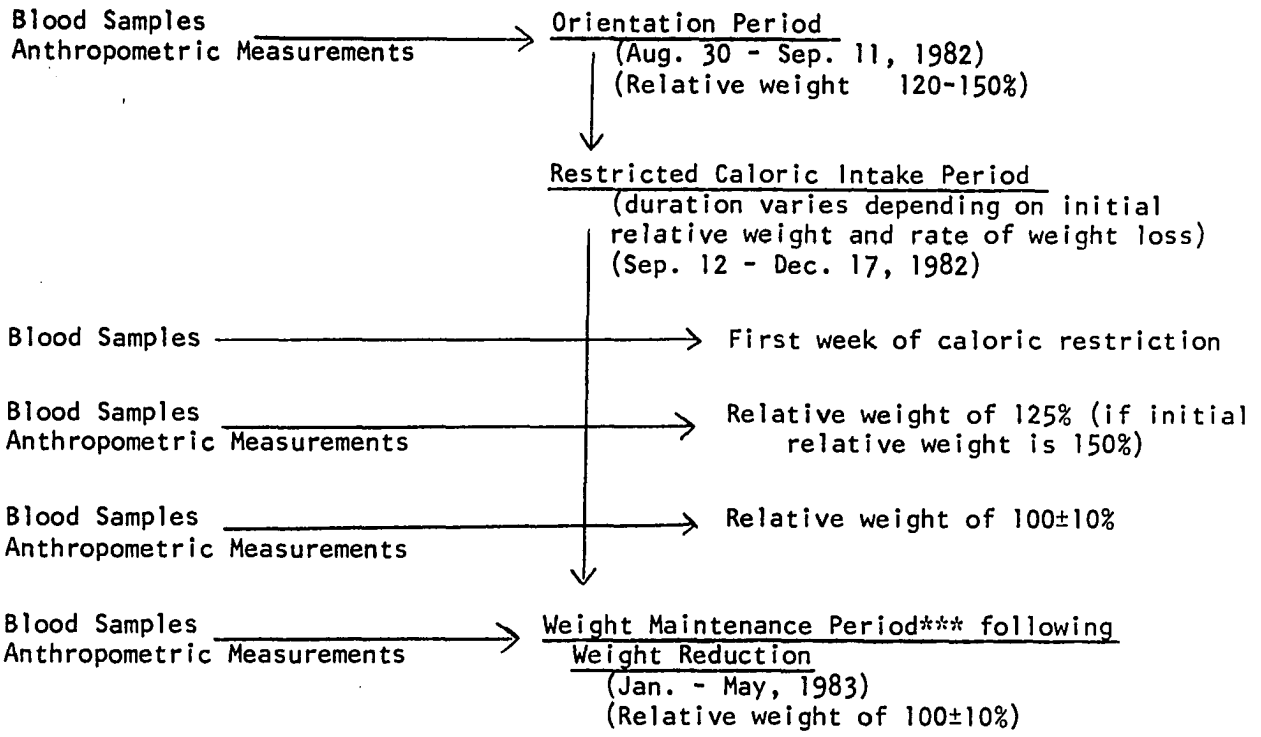
10. Ibid. pg. 5-6.

Tentative Time Schedule of Activities: Blood Concentrations of prostaglandins, insulin and glucose in overweight college women in a body weight maintenance program

Dates	Activity	Brief Description
Mar. 15 - Aug., 1982	Preliminary Study	A) Set-up mechanics for (1) recruitment, (2) a monitored feeding system in the residence dining hall (with obese volunteers, n=11), and (3) monitoring activites of participants during study. B) Set-up analytic procedure for serum PGs and plasma insulin and glucose.
Aug. 1982-May, 1983	Experimental Study	
Aug. 23 - Aug. 31, 1982	1) Program Orientation Target sample size: 30 individuals. Criteria for participation (a) female, (b) 18-22 years of age, (c) body weight 120-150% relative weight, (d) consume meals at residence dining hall, and (e) free-living in ISU dormitory.	A) Medical examination at ISU Health Service facility and completion of forms (participant approval forms) B) Meetings at Andrews House with participants for (1) progress report on program activites, (2) discussion of study protocol, (3) participant/staff interaction.
Aug. 30 - Dec. 17 1982	2) Feeding Phase at Residence Dining Hall	
Aug. 30 - Sept. 11, 1982		A) Orientation periods (see attached sheet)
Sept. 12 - Dec. 17, 1982		B) Restricted caloric intake (a deficit of approximately 1000 kcal/day) period (see attached sheet)
Jan. - May 1983 (because of staggered participation into program)	3) Weight Maintenance Period following Weight Reduction	A) (see attached sheet)

Collection of postprandial* and fasting blood samples* for PG, insulin and glucose analysis and anthropometric measurements**



* Postprandial blood sample (1 hour after food consumption)
Fasting blood sample (12 hours after food consumption)

** Anthropometric measurements will be obtained on four different occasions; blood samples will be obtained on five different occasions.

*** Subjects who regain body wt. will remain in the study for observation.

AUGUST						
S	M	T	W	T	F	S
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	(25)	26	27	28
29	30	31				

SEPTEMBER						
S	M	T	W	T	F	S
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30		

OCTOBER						
S	M	T	W	T	F	S
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24, 31	25	26	27	28	29	30

NOVEMBER						
S	M	T	W	T	F	S
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30				

DECEMBER						
S	M	T	W	T	F	S
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	(17)	18
19	20	21	22	23	24	25
26	27	28	29	30	31	

APPENDIX C. GENERAL INSTRUCTIONS ON DIETARY INTAKES DURING THE BODY WEIGHT MANAGEMENT PROGRAM

GENERAL INSTRUCTIONS ON DIETARY INTAKES DURING THE BODY WEIGHT MANAGEMENT PROGRAM

Staff members to be contacted for information regarding meals, snacks, and food service:

Barb Struempler
Andrews House
294-7336
292-9647 (Home): until 9 P.M.

Ruth Smith
34A MacKay Hall
294-7316

RATIONAL OF THE RESTRICTED DIETARY INTAKE

Commonly eaten foods are categorized according to their nutritional value. You can easily plan your restricted dietary intake by dividing foods into five groups:

1) Dairy (D)
2) Meats (M)
3) Fruits (F)/Vegetables (V):
   Includes: Citrus Fruit (Fc)/Dark-green Vegetable (Vg)
4) Cereals (C)
5) Empty:
   Includes: Fats (F)/Oils (O)/Sugar (S)/Alcohol (A)

When food choices are based on the five groups, you will be able to choose foods that decrease your caloric intake but provide adequate vitamins, minerals, and protein.

The foundation diet of the five groups (as follows) consists of a recommended number of the servings for the first four food groups and specifies a maximum number of servings for the last group (Empty). This diet provides approximately 1200 kcal (calories) and allows you to make food choices to fit your eating style. You should make an effort to follow the recommended number of servings on a daily basis; however, a weekly balance is more meaningful than a daily balance. It is important to remember that with any dietary plan, a variety of food choices will help to provide a nutritious diet.

Food Group	Recommended No. of Servings	One Serving is Equivalent to:	Important Nutrients
Dairy (D)	2	1 8-oz. c. skim milk 1 c. yogurt 1 sl. cheese 1-1/2 c. ice cream or ice milk 2 c. cottage cheese	Calcium Riboflavin Vitamin B_6 Vitamin B_{12}
Meat (M)	2	2-3 oz. lean, cooked meat 2-3 oz. poultry 2-3 oz. fish 1 oz. of meat is equal to: 1 egg 1/2-3/4 c. cooked dry beans 2 tbsp. peanut butter 1/4-1/2 c. nuts 1 sl. of cheese	Protein Phosphorus Vitamin B_6 Vitamin B_{12} Vitmain A: (liver & egg yolks)
Fruits (F)	2 (include one Fc daily).	Includes all fruits: 1/2 c. cooked or juice 1 fresh fruit (apple, orange) 1/2 med. grapefruit One serving must include a citrus fruit (Fc): oranges, grapefruit, melons, berries, and tomatoes	Vitamin C Vitamin C
Vegetables (V)	2 (include one Vg 3-4 times/week).	Includes all vegetables: wedge of lettuce bowl of salad 1 med. potato One serving must include deep-yellow or dark-green vegetables (Vg), 3-4 times a week: 1/2 c. carrots Sweet potatoes Greens	Vitamin A Vitamin A Riboflavin Folacin Iron

Food Group	Recommended No. of Servings	One Serving is Equivalent to:	Important Nutrients
Cereal (C)	4	Includes all products made with whole grains or enriched flour or meal: 1 sl. bread 1/2-3/4 c. cooked cereal 1 oz. ready-to-eat cereal 1/2-3/4 c. cooked rice, macaroni, noodles, spaghetti 5 saltine crackers 2 graham crackers 1 med. potato 1 muffin or biscuit (2" dia.) 1/3 c. peas or cooked-dried beans 1/3 c. corn	Thiamin (B_1) Niacin Iron
Empty (E):			
Fats/Oils (O)	Max. of 4 for total group.	Includes foods like butter, margarine, mayonnaise and other salad dressings and other salad dressings and other fats and oils. Serving size is difficult to define, but in general: 1/2 tbsp, butter, margarine, vegetable oil to other fats 1 level tbsp. salad dressing 5 olives 1 sl. drained, crisp bacon	
Sugars (S)		Includes foods like candy, sugar, jams, jellies, syrups, sweet toppings, and other sweets; soft drinks and other highly sugared beverages. Serving size is difficult to define, but in general: 1 tbsp. jams/jellies 1 tbsp. syrups	
Alcohol (A)		Includes beer, wine, and hard liquor. Cut down on or eliminate alcholic drinks.	

PROCEDURE FOR THE DETERMINATION OF THE CALORIE-RESTRICTED DIETARY INTAKE

The menus for the coming week will be distributed on either Tuesday or Thursday morning at the weigh-in and breakfast meeting (7:00 A.M.) at Andrews House. Recommendations on food choices will be provided by the FN research staff, although your dietary intake may be selected from a wide variety of food service menu items.

Before planning your dietary intake, let us examine a sample menu (attached). The food items have been calculated to reflect the serving portion used in the M-W-L Food Service facility and its corresponding caloric (kcal) value. The main entree has been subdivided to reflect the ingredients of the item whenever possible. This should allow you enough flexibility to omit and combine foods as you wish. The food and menu items have been assigned a code number. The code number allows the research staff to identify the items. The food group letters correspond to the food groups which have been previously defined.

The procedure for planning your dietary intake is simple and will be easy to use once you have become accustomed to the system. The following day's dietary intake should be planned (anticipated intake) the previous day. For example, anticipate and plan Wednesday's dietary intake on Tuesday. The following steps should be used in planning and calculating the dietary intake:

Step 1: Review the daily menu.

Step 2: Select the food/main entree items that you prefer to eat and annotate the food group(s) and the caloric (kcal) value of these foods in the Planned Intake column.

Step 3: Determine (by addition) the number of servings from each food group and the kcal that have been marked in the Planned Intake column. In many situations, the food/menu items consist of one or more food groups. For example:

M/O = 1 serving of Meat and 1 serving of Oil

OO = 2 servings of Oil

M/O/C = 1 serving of Meat and 1 serving of Oil and 1 serving of Cereal

½M or ½D = 1/2 serving of Meat or 1/2 serving of Dairy

½M/OO = 1/2 serving of Meat and 2 servings of Oil

V/S = 1 serving of Vegetables and 1 serving of Sugar

½ Fc = 1/2 serving of Citrus Fruit

Step 4: Subtract the planned number of servings of each food group from the recommended number of servings. This provides you with the number of servings that are further required to meet the recommended number of servings for each group.

Step 5: Review the menu items again and select food items that provide the additional number of servings from the food groups and annotate the food groups and kcal in the Planned Intake column.

Step 6: Total (by the addition of Steps 3 and 5) the number of servings from each food group and the kcal. Again, compare this amount with the recommended number of servings. When you need to select additional foods to fulfill the recommended number of servings, repeat Steps 5 and 6. Your planned caloric intake should range from 1200 to 1700 kcal. When your planned caloric intake exceeds 1700 kcal, reselect menu items to decrease the caloric intake but still provide the recommended number of servings of the food groups. The easiest method to decrease the caloric intake is to select a main entree that has less calories than your previous (first) choice.

Step 7: Immediately following the consumption of a snack or meal, circle the food group letter in the Food Group column and annotate the caloric value in the Actual Intake column. At the end of the day, annotate the total number of servings from the food groups and the total caloric intake in the boxes located at the bottom of the menu. Space is provided on the menu to describe any serving difficulties and for other comments.

Food Substitutions by Food Service: Occasionally, a food item that appears on the menu will be replaced/substituted with another food item. When you consume the substituted food item, cross out the original food item and enter the actual (substituted) food item consumed below the original food item. Food substitutions will usually be of comparable food groups and caloric value. Therefore, add the food group(s) and the caloric value of the original food item into your total dietary intake. Food substitutions will be less of a problem as you become more familiar with the menu items.

Salad Bar Selection: A list of Salad Bar Food Items has been given to you. You should check the food group and caloric value of a salad bar item before its consumption. The salad bar food items from Table A can be consumed in generous amounts, whereas, those foods from Table B should be eaten in limited amounts. The salad bar food items from Table C should not be consumed. As a general rule,

a tossed salad that consists of generous amounts of food items from Table A, such as lettuce, cabbage, carrots, celery, mushrooms, onions and tomatoes, will have approximately 50 kcal and provide one serving from the Vegetable group. When you prepare a tossed salad similar to the above from food items listed in Table A only, simply write "tossed salad from Table A, V (indicates the food group) and 50 kcal" in the space provided under Salad Bar on the menus. However, accurately indicate the code number, menu item, serving size, food group(s) and caloric value for a menu item from Table B.

The low-calorie tossed salad will be a great source of calories when heaped with salad dressing. One level tablespoon of salad dressing has about 50 kcal and is one serving from the Fat/Oil group. As you realize, we seldom use this amount of dressing on a tossed salad. Try to learn to like vinegar on your tossed salad. Another option to the high-calorie salad dressing is to purchase a low-calorie salad dressing from the grocer and bring the bottle with you to the dining hall.

Condiments: A list of condiments available in the food service can be found following Table C in the Salad Bar Food Items list. Condiments that are added to a hamburger or other menu items contribute "empty" calories to the food item. In general, avoid mayonnaise, catsup, and sweet pickles/relish.

Sauces/Gravies/Breading: These items contribute "hidden" calories to the diet and should be omitted. Several meat items on the menu are breaded, then cooked, and you will be served the breaded portion of meat. However, the food group(s) and caloric value of the breaded items have been calculated without the breading. You must remove the breading from these meat items. The menus will usually specify that the breading should be removed, but if in doubt, always remove the breading.

Snacks: Several food items available from the food service can be used as snacks and provide a serving from a food group (see list). A space for snack items is provided on the menu. You will need to annotate the code number, menu item (snack), serving size, food group(s) and caloric value on the menu. On occasion, a bag of popcorn will be provided to you by the research staff.

Beverages: Skim milk has essentially the same nutrients as whole milk but fewer calories. You must omit whole or 2% milks from your diet and drink skim milk. Fruit drinks (Hi-C, Tang) are not substitutes for fruit juices and should not be consumed. Coffee and tea (without sugar) may be consumed as desired.

Alcohol: Alcohol consumption is permitted during the restricted dietary intake period but in moderation only. Alcohol has approximately twice the calories per ounce as protein, starches, or sugars. Regardless of the type of alcohol, assume that a 12 ounce beer, or 1/2 cup of wine, or one jigger of hard liquor has 130 kcal and is equivalent to one serving from the Alcohol group. When hard liquor is mixed with 6 ounces of a carbonated beverage (Cola, 7-Up), an additional 70 kcal should be added to the 130 kcal from the hard liquor (200 kcal total per drink) and this is equivalent to one serving from the Alcohol group and one serving from the Sugar group.

Sunday Night Dinners: Since this meal is not available from the food service, you will be entirely responsible for the selection of food items. But beware, this meal could drastically increase your caloric intake. It is always fun for "the group" to go out on Sunday nights for the thick, cheesy pizza (about 600 kcal per slice) or the Big Mac Attack (about 600 kcal per serving). Moreover, your caloric intake from the Sunday lunch may be greater than for other lunches or dinners due to the larger portion sizes and the limited food choices. You have been careful to restrict your caloric intake for the entire week, so why blow it now?? Regardless of your food choices for this meal, you must record the food items in the space provided under Dinner on the Sunday menu. Attempt to annotate the code numbers, menu items, servings sizes, food groups and caloric intakes. Most information is available from the daily menus.

Two suggestions for low-calorie ideas for the Sunday night dinner are:

1) A group of participants in the Body Weight Management Program can pool their funds and purchase containers of yogurt or cottage cheese, packages of sliced cheese or luncheon meats, a loaf of bread, a box of saltine/graham crackers, fruits and vegetables, skim milk, or diet pop. This potluck meal could be eaten in a dormitory room, lounge, or at Andrews House providing prior arrangements have been made with the research staff. (Popcorn would be made available by the research staff. This would also be a good time to complete your weekly forms).

2) Eat a chef's salad in a food establish. (Code the food items from the Salad Bar Food Items list).

ACTUAL DETERMINATION OF THE RESTRICTED DIETARY INTAKE FROM A SAMPLE MENU

We will now plan and calculate a dietary intake from a sample menu (attached). Refer to Steps 1-7 as previously discussed. The steps are also outlined on the bottom of your menu.

Step 1: Review menu.

Step 2: Decide on food choices and annotate the food group(s) and kcal in the Planned Intake column.

Food Choices:

Windsor Chop (dinner)
Rice (dinner)
Chicken Noodle Soup (lunch)
Fresh Fruit Plate w/Amer. Cheese (lunch)
Tossed Salad from Table A (lunch)
Skim Milk (lunch - 8 oz.)
Orange Juice (breakfast)
1 Fried Egg (breakfast)
1 sl. English Muffin (breakfast)
1 tbsp. Jam (breakfast)

Step 3: Add the number of servings from each food group and kcal marked in the Planned Intake column.

Step 4: Subtract the planned number of food groups from the recommended number of servings. The recommended number of servings for each food group is located directly above the boxes at the end of the menu.

Step 5: Review the menu items again and select foods to complete the recommended number of servings for each food group and annotate the food group(s) and kcal in the Planned Intake column.

Food Choices:

1 sl. Bread (lunch) -- See "Other Additions to Menu" on menu
1/2 c. Skim Milk (dinner)
Tossed Salad from Table A (dinner)
5 Saltine Crackers (snack)
1 Apple (snack)

Step 6: Add together the number of servings from each food group and kcal (Steps 3 and 5).

Step 7: At the end of the day, annotate the total number of servings and kcal in the boxes located at the bottom of the menu.

Based on the sample menu, the recommended number of servings for each food group was met, except for one-half of a portion of Meat and one Green-Vegetable. These groups may be used in another day's dietary intake. Remember, a weekly balance is more important than a daily balance.

The planned and actual dietary intakes from the sample menu illustrate how different foods can be combined to supply a nutritious low-calorie diet. The following menu can be constructed from the actual dietary intake from the sample menu.

Breakfast	Lunch	Dinner
5 oz. Orange Juice	4 oz. Chicken Noodle Soup	3 oz. Windsor Chop
1 Fried Egg	Fresh Fruit Plate w/Amer. Cheese	1/4 c. White Rice
1/2 English Muffin	1 sl. Bread	Tossed Green Salad
1 tbsp. Jam	Tossed Green Salad	4 oz. Skim Milk
Coffee/Tea	8 oz. Skim Milk	Coffee/Tea
	Coffee/Tea	

Snacks

Fresh Apple, 1 med.
5 Saltine Crackers

DISTRIBUTION AND PORTION CONTROL OF MENU ITEMS

You will continue to eat your meals at the M-W-I.- dining hall and use your meal ticket in the usual manner.

The caloric values of the food items on the menus are based on normal portions served in the food service. Most menu items are served as a standardized portion, such as meats, soups, whipped potatoes, fruit juices, rice, vegetables and scrambled eggs. Some menu items are not served as a standardized (nonstandardized) portion, such as salad bar items.

You will be responsible for the serving of menu items from the salad bar. In general, one heaped tablespoon and one level tablespoon will provide 1/4 cup of Salad Bar items from Table B.

Tongs will be used to serve the shredded lettuce, cabbage, (Table A food items), but as previously emphasized, one cup of lettuce when compared to three cups of lettuce will not greatly affect your overall caloric intake.

Immediately following a snack or meal, you should circle the appropriate food group and indicate the caloric value of the food in the Actual Intake column. Left-overs should be subtracted from the actual serving portion.

RESPONSIBILITIES OF THE PARTICIPANT IN THE BODY WEIGHT MANAGEMENT PROGRAM

I. Food Selection

You will be responsible for the selection of food items from the menus based on the five food groups. Your food choices should be varied to: (1) provide the essential nutrients; and (2) prevent a monotonous dietary regime.

II. Maintenance of Records

You will be responsible for the maintenance of several records which include:

(1) the anticipated daily dietary intake: This involves a preliminary examination of the menus and selection of an anticipated dietary intake based on: (a) the five food groups, and (b) the menu items available from the food service.

(2) the actual daily dietary intake: This requires that you appropriately annotate your actual intake on the menus immediately following its consumption. At the end of the day, total your actual consumption and complete the information on the menus. The completed menus for seven days are to be submitted to the research staff during the breakfast meetings on Monday at Andrews House.

(3) the weekly dietary summary worksheet: This involves the composition of the weekly number of servings from each food group and a calculated weekly average caloric intake. The description and directions are provided on the worksheet. The worksheet is to be submitted with the completed daily menus to the research staff during the breakfast meetings at Andrews House.

(4) the dietary intakes during University holidays, term breaks, and/or weekends: When meals are not eaten at the M-W-L dining facility, you will record your dietary intakes according to the instructions provided by the research staff.

(5) the physical activity record (see below).

III. Adoption of a Physical Activity Pattern

In addition to decreasing your caloric intake, you should increase your energy expenditure. Your exercise regime does not need to be a strenous activity such as jogging. A brisk walk on a daily basis will "burn-off" the calories and "tone-up" the muscles. You will be encouraged to adopt an exercise pattern of your choice and keep a record of this activity. The description and directions are provided on the Physical Activity Pattern Chart. The chart is to be submitted with the completed daily menus and dietary summary worksheet to the research staff during the breakfast meetings at Andrews House.

IV. Compliance with the Body Weight Management Program

You should make an effort to comply with the dietary recommendations of the research staff. It is also important that you are honest with yourself in recording your actual daily intake. Do not become a "closet muncher." The research staff realize that there are certain foods that you like that are not considered nutritious, low-calorie foods. However, it is important that you inform the staff of these foods, so that your intakes can be modified to allow for these foods (on a limited basis). The research staff is here to assist and guide you in losing weight and the maintenance of the weight loss.

Salad Bar Food Items

Table A

Many food items are available from the salad bar in any amount that you wish. They contain few calories, provide roughage to the diet and supply many essential nutrients.

Code No.	Food Item	Amount	Food Group
486	Broccoli	1/2 c. (25 kcal)	Vg
	Carrots:		
619	fresh	Strip 1/4" wide, 3" long (5 strip = 10 kcal)	V
622	canned	1/2 c. = 25 kcal	V
632	Cauliflower	1/2 c. = 15 kcal	V
637	Celery	3 stalks, 5" long, 3/4" wide = 10 kcal	V
942	Cucumbers	5 slices, 1/8" thick = 5 kcal	V
1415	Green Onions	1/4 c., chopped = 10 kcal	V
1545	Green Peppers	1/4 c., sliced = 5 kcal	V
1354	Mushrooms	1/2 c. = 10 kcal	V
1258	Lettuce	1 c. shredded = 10 kcal	V
1412	Onions	1/4 c., chopped = 15 kcal	V
1844	Radishes	5 med. = 5 kcal	V
512	Red Cabbage	1 c., shredded = 20 kcal	V
1977	Sauerkraut	1/2 c. =20 kcal	V
2177	Spinach	1/2 c. = 25 kcal	Vg
2282	Tomatoes	1 tomato = 20 kcal	Fc
	Zucchini		

Table B

Many food items are available from the salad bar that should not be consumed in large amounts. The fruits or vegetables in the menu items are nutritious but either they contain a lot of calories for a small serving portion, or they have been mixed with sugar or oil which would greatly increase the caloric value of the food.

Code No.	Food Item	Amount	Food Group
1529	Peas	1/2 c. = 55 kcal	C
856	Corn	1/2 c. = 70 kcal	C
4R	Cucumbers/Onions Sweet/Sour	1/4 c. = 45 kcal	¼V/½S
2R	Potato Salad	1/4 c. = 135 kcal	OO/½C
3R	Kidney Bean Salad	1/4 c. = 100 kcal	¼P/¼O/¼C
6R	Pea Salad	1/4 c. = 110 kcal	¼P/¼O/¼C
8R	Tomato/Cucumber/ Onion Salad	1/4 c. = 25 kcal	¼V/½S
9R	Marinated Chinese Salad	1/4 c. = 50 kcal	½C/½S
10R	Macaroni Salad	1/4 c. = 96 kcal	O/½C
11R	Sprout-kraut	1/4 c. = 90 kcal	½V/S
12R	3-Bean Salad	1/4 c. = 190 kcal	½C/S
13R	Creamy Coleslaw	1/4 c. = 35 kcal	½V/O
15R	Sweet/Sour Beets	1/4 c. = 115 kcal	½V/S
16R	Carrot-Raisin Salad	1/4 c. = 70 kcal	½V/O
653	Cheese	1/4 c., shredded = 105 kcal	½M or ½D
14R	Devilled Eggs	1/2 egg = 55 kcal	¼M/¼O
974	Egg	1/4 c., chopped = 55 kcal	½M/½O
461	Croutons	1/2 c. = 50 kcal	¼C/¼O
1507	Pear, canned	1/2 pear (no syrup) = 80 kcal	½F/½S
1483	Peach, canned	1/2 peach (no syrup) = 80 kcal	½F/½S
29	Applesauce	1/4 c. = 60 kcal	½F/S
2005	Luncheon Meats	1/8 c., cubed = 35 kcal	¼M/¼O
2236	Sunflower Seeds	1 tsp. = 100 kcal	OO
132R	24-Hour Salad	1 serving = 90 kcal	F/S
133R	Fruit Mixture	1/4 c. = 30 kcal	F/S
1499	Peanut Butter	1 level tbsp = 95 kcal	½M/O
1381	Chow Mein Noodles	1/4c., = 55 kcal	¼C/O
1408	Black Olives	5 olives = 1/4c. sliced = 40 kcal	O

Table C

There are only a couple of food items available from the salad bar that should not be consumed at any time. These foods are "loaded" with calories and have little, if any, nutritional value:

Baco-bits
Onion Rings
Jello

Condiments

Code No.	Food Item	Amount	Food Group
1938	Mayonnaise	1 tbsp. = 100 kcal	00
1373	Mustard	1 tsp. (pouch) = 5 kcal	
	Pickles		
1558	Dill	(sliced lengthwise, 1/3) = 5 kcal	
1561	Sweet	(2½" long, 3/4" dia.) = 25 kcal	1/2 S
1565	Relish	1 tbsp. = 25 kcal	1/2 S
		1 packet = 15 kcal	
2286	Catsup	1 tbsp./1 packet = 15 kcal	1/2 S
2287	Taco Sauce	1 tbsp. = 16 kcal	

Snack Items

Many foods make excellent snacks and provide a serving portion from a food group. A list of recommended snack items is below.

Code No.	Snack Item	Food Group
	1 fruit = 80 kcal	F
916	5 saltine crackers (2" sq.) = 10 kcal/cracker	C
913	1 graham cracker (2½" long, 2" wide, ¼" thick) = 55 kcal	1/2C
1814	30 pretzels (sticks, 3" long, 1/8" dia.) = 75 kcal	C
	4 oz. (1/2 c.) fruit juice = 50 kcal	Fc
1846	2 level tbsp. raisins = 50 kcal	F
653	1 sl. cheese = 80 kcal	1/2M or 1/2D
974	1 hard boiled egg = 70 kcal	½M/½O
	1 box cold cereal = 100 kcal	C
1654	3 c. popcorn (air-popped) = 150 kcal	C
2481	1 c. calorie yogurt - 210 kcal	D or M
	carrots (any amount)	
	celery (any amount)	
	radishes (any amount)	
1655	Popcorn w/ oil 1 c. = 75 (3 c. = 225 kcal)	C & 1½O

Week

Subject No. Sample

	Code No.	Planned Intake	Menu Item	Food Group	Actual Intake
			Chilled Fruit Juice		
	2288		1 serving, Tomato (30 kcal)	Fc	
	2288		1 serving, V-8 (30 kcal)	Fc	
B	1071		1 serving, Grapefruit (60 kcal)	Fc	
	27		1 serving, Apple (75 kcal)	Fc	
R	1433	Fc 80	1 serving, Orange (80 kcal)	(Fc)	80
E			**Cereal**		
			1 box, Cold (100 kcal)	C	
A			4 oz., 1 ladle, Hot (100 kcal)	C	
K			**Toast/English Muffin**		
	461	C 65	1 sl (65 kcal)	(C)	65
F	461		2 sl (130 kcal)	CC	
	1317		Margarine, 1 pat (35 kcal)	O	
A	1149	S 50	Jelly/Jam, 1 tbsp. (50 kcal)	(S)	50
S			**Fried Eggs**		
	973		2 eggs (170 kcal)	M/O	
T	973	½M/½O 85	1 egg (85 kcal)	(½M/½O)	85
			Chicken Noodle Soup		
	19R	25	4 oz., 1 ladle (25 kcal)		25
			Hamburger on Bun		
	1902		1 whole bun (120 kcal)	CC	
	1902		1/2 bun (60 kcal)	C	
	370		2.3 oz., 1 cooked Hb (185 kcal)	M/O	
			Fresh Fruit Plate w/Amer. Cheese		
L	13c	½F 40	1/2 apple (40 kcal)	(1/2 F	40
	1420b	½F 40	1/2 orange (40 kcal)	1/2 Fc)	40
U	141c	½F 40	1/2 banana (40 kcal)	1/2 F	40
		—	1/2 Other _____	1/2 F	
N	653	½D 80	1 sl. cheese (80 kcal)	½M or (½D)	80
C			**French Fried Potatoes** (OMIT)		
H			**Fruit** (80 kcal)	F	
			Salad Bar		
		V 50	Tossed Salad from Table A	(V)	50
	1322	½D 40	**Milk, skim** 4 oz., (¼ c.)(45 kcal)	(½D)	40
		D 80	8 oz., (1 c.)(90 kcal)	(D)	80

Week

Subject No. Sample

	Code No.	Planned Intake	Menu Item	Food Group	Actual Intake
D I N N E R	1716	M/O 310	Windsor Chop 3.0 oz., 1 cooked portion (310 kcal)	(M/O)	310
	79R	_____	Beef Stroganoff 1/4 c., 1 ladle (70 kcal)	M	_____
	1872	C 70	Rice 1/4 c., 1 scoop (70 kcal)	(C)	70
	1530	_____	Peas 1/2 c., 1 scoop (55 kcal)	C	_____
	_____		Fruit (80 kcal)	F	_____
	87R	_____	Mashed Squash 1 scoop (80 kcal)	Vg	_____
		V 50	Salad Bar Tossed Salad from Table A →	(V)	50

Other Additions to Menu/Snacks/Alcohol

461	C 65	1 bread (lunch) →	(C)	65
916	C 50	5 saltine crackers (snack) →	(C)	50
13C	F 80	1 apple (snack) →	(F)	80

Procedure for Planning and Calculating the Dietary Intake

Step 1: Review menu.

Step 2: Decide on food choices and annotate food group and kcal in Plan. Int. column.

		M	C	D	F	V	E	kc
Step 3:	Totals:	1½,1	11	1½,1	2½,½ Fc 1,½	1 Vg 0	1,1½,1	280 B) 305 L) 965 430 D)
Step 4:	Subtract:	½	2	½	0 +½	0 0	1½	—
Step 5:					1	1		65,80 50,50 40 285
		0	11	½	0	0	0	
Step 6:	Add steps 3 & 5:	1½	4	2	2 ½	2 0	2½	1250

Step 7: Following snack/meal, circle food group letters and mark kcal in Act. Int. col.

Daily Totals

Meat (2)	Cereal (4)	Dairy (2)	Vege. (1)	Vege.-gr. (1) (3-4x/wk)	Fruit (1)	Fruit-C (1)	Empty (max 4)	Kcal 1200-1700
1½	4	2	2	0	2	1½	2½	1250

Serving Difficulties: _____

Comments: _____

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