ORAL CONTRACEPTIVES, WEIGHT CONTROL, AND FAT PATTERNING IN YOUNG COLLEGE WOMEN

by

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INTRODUCTION

Since the introduction of oral contraceptives in the 1950-60's, they have become the most widely used contraceptive today. Approximately seven million women in America take 'the pill' daily (2).

Of particular interest in this study is the effect of oral contraceptive use on weight gain. In 1985 Bradley (65) asked 243 women what conditions had been associated with their weight gain, and 8.5% cited oral contraceptives as a factor. Merians et al (47) found that women using progestin-dominant oral contraceptives had more body fat, and those taking estrogen/progestin balanced pills had less body fat than women who did not take oral contraceptives. Amatayakul et al (67) observed that Thai women using the injectable contraceptive medroxyprogesterone acetate exhibited a mean weight gain of 6.05 kg. Others reported no change in weight or body composition (81,82).

Few of the studies cited above have examined the effects of oral contraceptives on fat distribution. Because female hormones are generally associated with female secondary sex characteristics and characteristic distribution of fat (68), a study or oral contraceptives seems warranted. Furthermore, there is little work on the effects of hormonal potencies in oral contraceptives. The two synthetic estrogens and five synthetic progestins currently in use (7) vary in

their potencies or biologic activities; and biologic activity is more important than the type or dosage of the hormones when considering effectiveness and side effects (7,8). Also much of the previous work was done on oral contraceptives which had higher doses of hormone than are currently prescribed (95).

The objectives of the study reported herein are to compare body weight, body fat, and fat patterning between oral contraceptive users and matched non-users. Correlations between those measurements with hormonal potencies in the combined oral contraceptives were also examined.

REVIEW OF LITERATURE

I. ORAL CONTRACEPTIVES

Types of Pills

Oral contraceptives, containing both an estrogen and progestin, were first demonstrated to be effective contraceptives in the mid to late 1950's (1). These are more commonly referred to as 'combined' oral contraceptives. In the early 1960's these combined pills contained 100-150 mcg of an estrogen and 1-10 mg of a progestin (1,2). Since the introduction of the oral contraceptives, there has been a trend towards prescribing lower doses of both the estrogen and progestin due to the reported side effects and complications with the higher doses. Combined oral contraceptives now contain 30-50 mcg of an estrogen and 1 mg or less of a progestin (1,3,4). The trend towards lower doses also brought about the development of the 'Mini-Pill' or progestin-only pill in 1973 (1). These oral contraceptives contain no estrogen and also have less than 1 mg of progestin (1,4).

Sequential pills contain both an estrogen and a progestin (4,5). The dosage of each hormone fluctuates throughout the three-week cycle so that it more closely simulates normal hormonal function. The combined and mini-pill have a constant dosage of the hormones throughout the three-week cycle.

Another type of oral contraceptive is the phasic pills-- biphasic and triphasic. These pills contain both an estrogen and progestin. In the biphasic pill, the initial low dosages of estrogen and progestin are changed after ten days to a slightly higher dosage of progestin for the remainder of the cycle. With the triphasic pill, the progestin dosage increases after the seventh and fourteenth days of the cycle (3,5).

The phasic and sequential pills have only recently been re-introduced into the market and have not gained widespread use. The 'Mini-Pill' has not gained popularity due to the spotting and breakthrough bleeding associated with its use. Because combined pills are the most widely-used pill currently, the remainder of this review will focus on their consequences.

Hormones and Potencies

The combined pill contains a synthetic estrogen and progestin similar to the hormones the ovary normally produces. Natural estrogen and progestin (estradiol and progesterone) cannot be used orally because the digestive system destroys them (5,6). This is due to the rapid, first-pass hepatic metabolism following intestinal absorption.

When comparing the effectiveness of various combined pills, it is important to note that their

biologic activity is more important than the type or dosage of the hormones (7,8). When the hormones are being prescribed for a disorder, the biologic activity is of primary importance (9). Side effects are also less dependent on the dosage than the potency (8).

There are two synthetic forms of estrogen currently in use-- ethinyl estradiol and mestranol (1,4,7,10). The structural formulas of these two hormones are found in Appendix A. The potency or biologic activity of the two hormones is essentially the same, with 30 mcg of ethinyl estradiol equivalent to the activity of 50 mcg of mestranol (8). The potency of estrogen is hard to define because it differs depending on the tissue, organ, or species in which it is tested. Various studies have examined vaginal smears, uterine growth, vaginal opening, and oviduct growth as a means of determining the potency of various estrogens (10). The anti-estrogenic and estrogenic effect of the progestins also compound the measurement of estrogenic potency for a given combined pill. However, the potency scale most widely accepted is based on the estrogenic effect on uterine growth and anti-ovulatory effect (11,12) and is shown in Table 1.

The synthetic oral progestins currently used are derivatives of testosterone (7,13). The 19-methyl group is removed from the testosterone to reduce its

Table 1. Relative potencies of synthetic hormones

	Relative Potencies			
Progestin	Pro[1]	Est[2]	Anti-Est[3]	And[4]
Norethindrone	1	0.25	2.5	1.6
Norethynodrel	1.09	1.09	0	0
Norethindrone Acetate	2	2	25	2.5
Ethynodiol Diacetate	15	0.86	1	1
Norgestrel	30	0	18.5	7.6
Estrogen				
Mestranol	-	1	-	-
Ethinyl Estradiol	-	1.7-2.0	-	-

[1]Progestinic
[2]Estrogenic
[3]Anti-Estrogenic
[4]Androgenic

androgenic properties and unveil its progestinic capabilities. Increasing progestinic potency is achieved by 17 alpha acetylation which generates a family of orally active progestins commonly referred to as the '19 NOR' steroids. Additional acetylation of the 19 NOR steroids at either the 17 beta or 3 position results in the greater progestinic potency, as seen in norethindrone, norethindrone acetate, norethynodrel, and ethynodiol diacetate (13,14). The most potent progestinic activity is achieved by adding an additional methyl group at the 18 carbon as seen in norgestrel (14). The structural formulas of these five synthetic progestins used in combined pills today are found in Appendix B. The progestinic potency of these synthetic hormones is determined by the Greenblatt-Swyer Test which uses the criteria of postponement of menstruation (9,15). Various criteria have been used to identify progestinic capabilities, such as postponement of menses, withdrawal bleeding, and vaginal smears, but it appears that the postponement of menses criteria is most reliable and valid (15). The relative progestinic potency of identical dosages of the five synthetic progestins is shown in Table 1 (9,15).

The synthetic progestins exerts not only a progestinic effect but also an estrogenic effect,

anti-estrogenic effect, and androgenic effect (1,3,4,9,10,13). The progestins also vary in potency with respect to each of these effects. The estrogenic potency scale of the various progestins used in this research was determined by using the rat vaginal epithelial assay as its criterion. This scale of relative potencies is shown in Table 2 (16).

The anti-estrogenic activity of the progestin is its ability to inhibit the increase in uterine weight induced by an estrogen. The scale of activity most widely used examines vaginal smears to determine if keratinization had occurred. This scale is shown in Table 2 (17).

The androgenic property of the various progestins is attributed to their structural resemblance of dihydrotestosterone, the most potent androgen or male sex hormone. The growth response of the ventral prostate is the criteria used to determine androgenic activity. The androgenic potency scale of various progestins is shown in Table 2 (18).

From the preceding discussion it is evident that a single estimate of combination pills is virtually impossible according to Edgren and Sturtevant (10). This is due to the complex interactions among the two hormones as well as within each hormone. Estrogenic potency is affected by the type of estrogen, amount of

estrogen, biological activity of the estrogen, type of progestin, amount of progestin, estrogenic effect of progestin, and the anti-estrogenic effect of the progestin. Progestinic potency is affected by the type of progestin, amount of progestin, biological activity of the progestin, type of estrogen, and amount of estrogen. Thus, the interactions between the hormones are numerous, and this does not even account for the variability of the reactions within individuals.

II. ORAL CONTRACEPTIVE MODE OF ACTION

In order to understand how oral contraceptives work it is necessary to understand the hormonal changes during a normal menstrual cycle. During the first half of the monthly cycle before the egg is released from the ovary, the ovary produces only the hormone estrogen, which is made within the follicles. These have been activated by the follicle-stimulating hormone (FSH), which is produced by the pituitary at the base of the brain.

At mid-cycle, the luteinizing hormone (LH), is released from the pituitary and causes the follicle to burst and release the egg. Once the follicle is empty, it begins to produce progesterone as well as estrogen, and both hormones travel to the uterus to thicken its lining so that it is prepared to receive a fertilized egg. After two weeks, if an egg has not been embedded

in the uterine lining, the ovaries stop producing estrogen and progesterone, and the lining of the uterus breaks down resulting in menstrual flow.

If a fertilized egg has been embedded in the lining of the uterus, another hormone is produced which works on the follicles to ensure that they keep producing the estrogen and progesterone needed for the lining of the uterus to remain and provide nourishment for the fetus. Because the levels of estrogen and progesterone remain high, the pituitary produces much less FSH and LH, and the follicles in the ovaries produce a steady amount of estrogen and progesterone. Therefore, if a pill with doses of estrogen and progestin is taken, there is a constant level of the hormones and the pituitary gets the same feedback as if one were pregnant. The pituitary stops triggering the ovary to release eggs.

When examining the mode of action of the combined pills, it becomes apparent that both hormones play separate and distinct roles. Thus, each hormone and its mode of action are discussed.

The anti-ovulatory effect of estrogenic agents is the primary contraceptive activity (10,19). The estrogenic agents exert their anti-ovulatory effect by inhibiting the release of FSH and LH from the pituitary (1,3,10,13). The estrogenic agents act on the

hypothalamus which secretes follicle-stimulating hormone-releasing hormone (FSH-RH) and luteinizing hormone-releasing hormone (LH-RH) (1,10). These releasing hormones from the hypthalamus trigger the release of FSH and LH from the pituitary. By inhibiting the pituitary from producing FSH, the estrogenic agent stops the follicle from ripening and the egg from maturing. Because there is no surge of LH, no egg is released (5). Thus, the estrogenic action actually occurs three stages away from the actual point of ovulation, since it exerts its influence on the LH-RH in the hypothalamus (10).

Estrogenic agents may also exert an anti-ovulatory effect by direct action upon the ovary. The hormone may cause an effect by influencing ovarian steroidogenesis through interference with gonadal enzyme systems (13). The estrogenic agents may also be used to deter fetus implantation. This is accomplished by administration of a high dose of estrogen after an unprotected act of intercourse. This changes the normal secretory development and causes areas of marked edema alternating with areas of dense cellularity which inhibits implantation (1).

The progestinic effects are more varied than those of the estrogen. The primary contraceptive effect of progestins is the alteration of the cervical mucus

(10). Progestins cause the cervical mucus to become scanty, thick, and cellular which creates a hostile environment for the sperm (1,2,3,5,10,13,19,20,21,22). The normal mid-cycle liquifaction of the cervical mucus is not seen. This hampers the transport of the sperm and decreases the ability of the sperm to penetrate the cervical mucus in order to reach the egg (1).

Progestinic agents also exert an influence on the development of the endometrial lining. The progestins exhibit a contraceptive effect by inhibiting nidation (the development of the uterine lining) which creates a hostile environment for fetus implantation (1,2,3,5,13,19,20,21,22). Progestins also cause a decrease in the fallopian tube contractions which decelerates ovum transport and inhibits fertilization (1,3,5,13,21). Another contraceptive effect of the progestins is the inhibition of capacitation (1). This is the activation of the hydrolytic spermatic enzymes required for the sperm to penetrate the ovum.

The progestins have a minor anti-ovulatory effect. They are the hormone which actually suppresses the secretion of LH, but they can only exert their influence on estrogen-primed tissue (3). Thus, the estrogenic agent is essential to the suppression of LH secretion. A major non-contraceptive role of the

progestins is their control of endometrial bleeding pattern (10).

To summarize, the estrogen's main mode of contraception is its anti-ovulatory effect. This is accomplished by the negative estrogen feedback (simulating pregnancy) which is an inhibiting factor to the release fo FSH and LH. The progestin contributes to the contraceptive effect, but its main role is the control of the endometrial bleeding pattern. The contraceptive effect of the progestin is exerted by creating a hostile environment for fertilization and implantation. This is accomplished by altering the cervical mucus, uterine lining, tubal contraction, and capacitation. Thus, the primary contraceptive effect of the combined pill is exerted by the anti-ovulatory effect of the estrogen which is supported by the hostile environment created by the progestin.

III. ORAL CONTRACEPTIVE SIDE EFFECTS

The side effects of the combined oral contraceptives have made them perhaps the most extensively studied medication in history. The combined pill and its effects are still not fully understood, but it has become much safer during the last twenty years of research.

Major or Life-Threatening Side Effects

Of the life threatening side effects, thromboembolic disease is the most dangerous and most widely publicized. Combined pills have been associated with pulmonary embolism, deep vein thrombosis, stroke, myocardial infarction, and postsurgical thrombosis (7). This disease involves the formation of a blood clot in a vein or artery and has been reported widely (1,2,5,7,20,21,23). Deep vein thrombosis was one of the first serious conditions to be associated with the combined pill (5). With this condition, part of the clot may break off and move along the veins to the chest and may result in pulmonary embolism.

The risk of arterial thrombosis increases as one gets older. The walls of the arteries tend to roughen and slight clotting may occur on the roughened surfaces, blocking the arterial flow (5). This process may result in stroke or myocardial infarction. The combined pill does not seem to have a cause and effect relationship on the cardiac system, but it has been suggested that it acts synergistically with the other cardiac risk factors (7). These include hypertension, smoking and hypercholesterolemia. Due the reports of increased incidence of postsurgical thrombosis, it has been suggested that women scheduled for elective surgery should be taken off the combined pill one month

prior to surgery (5,21). Emergency surgery should be preceded by treatment with drugs to prevent clots.

Combined pills have been implicated in the genesis of essential hypertension by both laboratory and epidemiologic studies (7). This condition is widely reported as a side effect of combined pill usage (1,2,5,7,20,21,24). Once the pill is discontinued though, the blood pressures tend to return to normal. It is still unclear, if an elevation in blood pressure does occur, whether it is the result of the estrogen, the progestin, or a combination of the two hormones. Preliminary studies contend that the estrogen alone or in combination with the progestin is responsible for the increase in blood pressure because there was no increase seen among progestin-only users (24,25). It has been speculated that the combined pill induces hypertension by the induction of hepatic renin substrate synthesis (7).

Another possible side effect which has received great interest, is the relationship between the combined pill and neoplasia. Breast and endometrial (uterine lining) neoplasia have been the primary forms studied in both animal studies and human epidemiologic studies. Animal studies revealing an association between the combined pill and breat carcinoma aroused interest in neoplastic research (7). The latest data

demonstrate no relationship of the incidence of breast neoplasia or deaths from breast neoplasia to the use of oral contraceptives (20). It has even been suggested that the combined pill may even exert a protective role against benign breast disease (5,26).

Evidence supporting the theory that the combined pill causes endometrial cancer at this time is inconclusive. During the mid 1970's several reports showed an increased risk associated with the combined pill. Further analysis revealed that the increased risk of endometrial cancer was primarily related to sequential pill preparations, and combination pills were actually beneficial in preventing it (5,27). This is may be attributed to the higher dosage of progestin used in the combined pill. Progestin may exert a protective effect because: 1. progestins cause regression or disappearance of endometrial hyperplasia and carcinoma in situ; and 2. women with corpora lutea failure (insufficient progesterone) are more prone to develop carcinoma (28). It is believed that the periodic shedding of the endometrium may serve as a "physiological eraser of endometrial neoplasia" (29). Because of the seemingly protective action of progestin it may be concluded that estrogen is the potential causative agent.

The combined pill has also been associated with hepatic neoplasms and abnormalities (1,7,21). These can cause rupture of the capsule of the liver and extensive bleeding (1). Other hepatic abnormalities linked to the combined pill use include decreased sulfobromophthalien transport, increased cholesterol saturation of bile, cholestatic jaundice, changes in cholesterol and triglycerides, and a rise in serum glutamic-oxaloacetic transaminase and/or alkaline phosphatase (7,21). The hepatic abnormalities may be due to the metabolism of the sythetic hormones which occurs in the liver. These abnormalities of the liver, the center of metabolism in the body, are the precursors to the high blood sugar (1,7,21,30), high blood triglycerides and cholesterol (1,7,21,30), gallbladder disease (1,7,21,30), and altered carbohydrate metabolism (1,7,21), related to the combined pill usage.

Minor or Nuisance Side Effects

The less serious side effects of the combined pill are generally referred to as minor or nuisances. These side effects are not life threatening and tend to be those that are associated with the first months of pregnancy, caused by an estrogen excess (7). These include tender breasts, nausea/vomiting, dizziness, weight gain, breakthrough bleeding and spotting,

bloating/edema, and an increase in breast size. These side effects have been observed in several studies (1,5,7,8,21,30,31). An estrogen deficiency may cause the symptoms seen in the premenopausal and menopause years (7). These include early and midcycle spotting, decreased amount of menstrual flow, hot flashes, no withdrawal bleeding and depression (1,3,5,7,8,21,31). The side effects caused by a progestin excess are generally attributed to the androgenic or anabolic properties of the progestins (7). These symptoms include noncyclic weight gain, increased libido and cholestatic jaundice (1,3,7,8,21,23,31). A progestin deficiency is characterized by late cycle breakthrough bleeding, heavy menstrual flow and clotting, delayed bleeding and decreased breast size (1,7,8,31).

A complete listing of the hormone etiology of the combined pill's side effects appears in Appendix C (1,7,8). Although all of these side effects have been given a negative connotation, this depends on the individual, and in some cases are considered positive. It is clearly evident that the majority of the side effects are associated with the excess estrogen dose, most notably the life threatening complications. It is for this reason that the dosages of the estrogens have been reduced dramatically since the introduction of the first combined pill in the 1950's.

Clinical Applications

The side effects of the combined pill have been studied not only to determine their adverse effects, but also to determine their clinical applications. The clinical applications of the combined pill generally refer to the progestinic activity, thus the mini-pill is usually used in clinical applications. The clinical uses of progestin include the treatment of amenorrheas, nymphomania, delay of menstruation, cycle regularization, fluid retention, cramps, acne, pelvic infections, rheumatoid arthritis and hirsutism (3,5,9).

Due to the complexity of the hormone interactions and the resulting side effects, it is essential that one has a proper medical examination done prior to taking the combined pill. Some physicians are utilizing the 'hormone profile' (8), to prescribe a combined pill that will most accurately meet the individual's hormonal needs and avoid the bothersome side effects. Contraindications and relative contraindications to taking the combined pill have been developed to decrease the number of life threatening side effects occurring as a result of combined pill usage (1,5,7,21,31). These appear in Appendix D.

IV. ORAL CONTRACEPTIVES AND CARBOHYDRATE METABOLISM

The use of the combined pill alters carbohydrate metabolism in a manner similar to that observed during

pregnancy. Abnormal glucose tolerance curves are commonly seen in normal pregnancies. The change in carbohydrate metabolism is generally manifested by decreased glucose tolerance curves, oral, intravenous, and cortisone-stimulated (13,32). It is generally found with normal fasting blood glucose levels and elevated fasting insulin levels which suggests an increased peripheral resistance to insulin (13). This is the type of abnormality seen in Type II - adult onset diabetes and is commonly associated with obesity. The type and dosage of the hormones both play a role in determining the change that occurs in carbohydrate metabolism.

It appears that the estrogen component has a biphasic effect on carbohydrate metabolism: higher doses cause a deterioration in carbohydrate metabolism, while lower doses tend to increase efficiency (33). However, ethinyl estradiol, the estrogen used in most combined pills, does not significantly alter carbohydrate tolerance when administered alone (34). The biphasic effect appears to only occur with the administration of both an estrogen and a progestin.

The synthetic progestins used in the combined pill, specifically the 19-NOR steroids have been implicated as altering carbohydrate metabolism. The effect seems to be dose-related and is seen with a

greater degree in norgestrel than norethindrone (35). This fact is supported by the findings of Spellacy et al (33). They found that women using .075 mg norgestrel alone had significantly higher blood glucose levels, plasma insulin, and weight gain after eighteen months. Another study by Spellacy et al (34), reported that using .35 mg norethindrone alone had significantly higher plasma insulin levels, but no change in the glucose tolerance curve. Thus, it has been interpreted that the major problems of carbohydrate metabolism occur with the high dose combined pills and those containing the progestin norgestrel (36). This is supported by the findings of Wynn and Doar (37). They found that the greatest deterioration in oral glucose tolerance was associated with the highest estrogen dose (75 mcg - 100 mcg mestranol), and the greatest increase in insulin secretion was seen with norgestrel. Another finding of this study was that when given a constant dose of progestin, glucose intolerance decreased with decreasing levels of estrogen. Thus, it may be possible that due to the biphasic effect of estrogen, a low dose of estrogen could counterbalance the adverse effect of the progestin (38).

Haller (13), presents a hypothesis regarding the diabetogenic action of the combined pill. He proposes that the plasma binding of insulin, similar to that of

thyroxin, is elevated particularly by estrogens when using hormone combinations. This reduces the percentage portion of biologically active circulating estrogen. This hypothesis explains the elevated plasma insulin values reported in other studies (33,34,37).

Another hypothesis explaining the diabetogenic effect of the combined pill has been presented by Briggs (39). He proposed that the Vitamin B_6 deficiency observed in women taking the combined pill use affects tryptophan catabolism. This leads to an acculumulation of xanthurenate or quinolinate which are potential diabetogenic agents.

As with other side effects, the alteration in carbohydrate metabolism has been reported primarily with the initial high dose combined pill. The current literature confirms the advantages of the new, low dose combined pills. With a dose of .035 mg ethinyl estradiol and .4 - .5 mg of norethindrone, no adverse effects on carbohydrate metabolism were found (36). This study also reported no significant change in plasma insulin levels, a decrease in the fasting blood glucose level of normal women, and no associated weight gain. A dosage of .03 mg ethinyl estradiol and .15 mg levonorgestrel introduced to women for six months also revealed no deterioration in fasting or plasma glucose values (40).

The most common symptom of changes in carbohydrate metabolism is the annoying occurence of the common vaginal infection Monilia Vaginitis (1,7,8,21). This symptom is linked to progestin excess.

Szarthmary and Holt (41), have reported a correlation between hyperglycemia and fat patterning. Vague et al (42), found an increased centripetality of fat associated with hyperinsulinemia in Caucasians. Centripetal fat patterning is centralized about the trunk of the body. These reports are consistent with the findings of Smith et al (43). He found that abdominal adipocytes were more responsive to insulin and epinephrine. Although the evidence demonstrates differences in glucose uptake and insulin responsiveness according to location of fat cells, it is not known how these differences relate to insulin resistance or diabetes. Thus, the elevated glucose and insulin levels observed in combined pill users may be the result of body fat distribution.

V. ORAL CONTRACEPTIVES AND LIPID METABOLISM

The alteration of lipid metabolism incurred as a result of combined pill usage has received much attention due to its close relation to coronary heart disease. One of the primary risk factors associated with coronary heart disease is an abnormal blood lipid profile. This refers to the lipoprotein constituents

of the blood which are divided into four classes. Two classes of primary concern when discussing coronary heart disease are, low-density lipoproteins (LDL) and high-density lipoproteins (HDL). LDL's have been positively correlated with coronary heart disease, whereas HDL's have been negatively associated with the disease.

Elevated plasma triglyceride and cholesterol concentrations have been reported among combined pill users (44,45,46,47). The elevated triglycerides, reflected by an increased concentration of very-low-density lipoproteins, are generally related to estrogen dose (46). The elevation in total serum cholesterol is manifested by an increase in the LDL-cholesterol level (48). Leuven et al (49), reported small to moderate increases in serum cholesterol, serum trglyceride, and apolipoprotein levels, and large decreases in liver lipase activities. There are numerous alterations in the lipid metabolism which bring about these general effects and these vary according to the dosage and potency of the hormones (44,46,47,48,50,51).

Estrogen has been linked to an increased concentration of HDL-cholesterol among its users, both alone (46,51,52,53), and as a component of the combined pills (46,47,49,51). Progestin use (46,48,51,52,53,54)

and progestin-dominant combined pills (46,48,51), on the other hand, have been associated with a lower concentration of HDL-cholesterol. The use of estrogen has also been found to decrease the concentration of LDL-cholesterol (46,48,51), while progestin (46,48,51), and progestin-dominant combined pills (47,48), increase the concentration of LDL-cholesterol. Thus, the total effect of combined pill use on HDL's and LDL's is dependent on the relative amounts and potencies of the hormones used due to their synergistic effects (46,48,51). A study by Merians et al (47), reported findings consistent with the stated changes occurring in the LDL's and HDL's. This study reported that users of a progestin-dominant combined pill had the highest mean LDL/HDL ratio (a high mean LDL concentration), and the lowest mean HDL concentration. Those using a balanced combined pill had the lowest LDL/HDL ratio and the highest mean HDL concentration. It was also found that the progestin dominant combined pill users had lower triglyceride levels than those using the balanced combined pill (47). These findings are consistent with earlier reports that estrogen increases triglyceride production and progestin increases the rate of triglyceride removal (55,56,57).

Merians et al (47), introduced another factor, body fat, and its relation to lipid metabolism. It was

reported that after adjustment for body fat, the association between the combined pill and plasma triglycerides, as well as the LDL/HDL ratio, remained significant (47). The association between progestin and decreased HDL, as well as estrogen and increased HDL on the contrary, became non-significant when adjusted for body fat (47). Thus, this could mean that HDL levels are related to body fat.

Another factor which Merian included in this study was exercise. Exercise is known to decrease triglyceride levels and increase HDL concentrations. Exercise also decreases body fat. After adjustment for body fat, Merians et al found no significant association between exercise and lowered plasma triglycerides (47). Thus, the lower body fat may have more of an effect on triglyceride levels than the exercise. It has been postulated by Williams et al (58), that the exercise-induced weight loss mediates changes in HDL's through processes associated with decreasing body fat. Thus, exercise, together with reduced body fat, was associated with favorable plasma lipid and lipoprotein concentrations, and partially compensated for the lipid changes associated with oral contraceptive use (47).

The estrogenic and anti-estrogenic properties of progestins may also play a role in lipid metabolism.

The anti-estrogenic progestins counteract the elevated triglyceride levels linked to estrogen (48). Norethynodrel, a progestin with no anti-estrogenic effect and moderate estrogenic effect, has been reported to cause elevated HDL-cholesterol concentrations whereas norethindrone acetate and norgestrel, progestins with strong anti-estrogenic effects, have been reported to decrease HDL-cholesterol concentrations (51).

Again, the new low dose combination pills have minimized the adverse effects on lipid metabolism. Briggs and Briggs (59), reported that there was little change to the HDL-cholesterol concentration associated with a dose of 30 mcg ethinyl estradiol and .5 mg norethindrone.

VI. ORAL CONTRACEPTIVES AND APPETITE, WEIGHT GAIN, AND BODY FAT

Appetite

Combined pills are classified as oretic drugs, or appetite-inducing drugs by some sources (60). Various sources have stated that the hormones contained in combined pills have a direct effect on the appetite center of the brain (61,62). The hormones exert an influence at the hypothalamic level in the release of FSH and LH. Since the appetite center is also located in the hypothalamus, it is believed that this area of

the hypothalamus may also be affected. This increased appetite has been implicated as one of the factors reponsible for weight gain observed in combined pill users. The synthetic progestin is the hormone implicated as the precipitator of the increased appetite due to the anabolic (androgenic) properties of the hormone.

Merians et al (47), reported no significant difference in combined pill users versus non-users in relation to total kilocalories, saturated fat intake, cholesterol intake, PUFA/SFA ratio or percent of kilocalories from protein, carbohydrate and alcohol. There was a significant difference between users and non-users in their percent of kilocalories consumed from fat. The pill users consumed a lower percent of kilocalories from fat than the non-users.

A case of binge eating associated with combined pill use has been reported (63). In this case, the binge eating was linked with the consumption of sweets and starches. This is consistent with the findings of Dippel and Elias (64). They found that women using low progestinic potency combined pills preferred very sweet solutions compared to those who used high progestinic potency combined pills.

Weight Gain

Weight gain is a common complaint among combined pill users (21). Due to the increased appetite induced by progestin, this is one of the major implications of weight gain. It was reported in a study by Bradley (65), that combined pill use is a condition associated with weight gain in adulthood. In this study, 8.5% of the subjects reported weight gain associated with combined pill use. The mean weight gain was 9.6 kg and accounted for 3.2% of the total weight gained. Bakker and Dightman (66), reported that combined pill users in this study experienced an increase abdominal girth but no significant trend towards weight gain.

The weight gain seen in combined pill users has been classified as cyclic and non-cyclic. The cyclic weight gain is characterized by fluid retention, bloating, and edema which are believed to be a result of excess estrogen (1,14,23). Tyrer (23), claims that estrogen induced weight gain is associated with an increase in subcutaneous fat in the breasts and hips with no increase in appetite. These characteristics have not been reported elsewhere.

Non-cyclic weight gain is believed to be a result of progestin excess (1,14,23,47). This is the result of the anabolic (androgenic) property of the hormone which increases appetite. Tyrer (23), also attributes

the weight gain, as a result of progestin excess, to the altered carbohydrate metabolism. Progestin can also cause weight gain as a result of fluid retention. Weight gain in medroxyprogesterone acetate (an injectible progestin contraceptive) has been attributed to fat deposition rather than anabolic or fluid retaining properties (67). In the study by Amatayakul et al 50% of the users' weight remained constant, 25% experienced weight loss, and 25% experienced weight gain. Of those who experienced weight gain, there were significant changes in the tricep, sub-scapular, and anterior abdominal wall skinfolds. Positive correlations between weight, mid-upper arm circumference, and skinfolds were reported. The weight gain and fat deposition observed in this study was again attributed to the anabolic (androgenic) property of progestin which affects the appetite center of the hypothalamus.

Body Fat

Prior to discussing the possible alterations in body fat composition caused by combined pill use, it is pertinant to review the literature in relation to normal fat patterning. Females have greater subcutaneous fat thickness than males (68,69). Garn (68) reported that the total fat of males and females was not notably different. Thus the sex difference is

in the proportion of outer and inner fat. Women carry more fat externally and less internally (68). Sjostrom et al (69), claims that the greater amount of subcutaneous fat in the female is due to an increased number of adipocytes in several subcutaneous regions. The gluteal region, however, differed because of larger fat cells rather than number (69). The difference in fat patterning between the sexes is believed to be hormonally controlled.

The average female has a fat distribution which is gynoid or peripheral (68). Gynoid obesity is characterized by excess adipose in the lower body, including the hip area, and has poor muscle-blood development (70,71). Complications associated with this type of obesity are those dealing only with the excess adiposity, such as locomotor difficulty, abdominal pressure, slowing of circulation, and limited respiratory movement. This type of obesity generally begins in the younger years since it is hyperplastic. This type of fat patterning was also reported by Young et al(72). She found the thickest fat pads on young women were on the lower trunk, especially on the abdomen midline half-way between the umbilicus and pubis. This study also reported the upper legs and upper arms as other major areas of fat deposition.

Android obesity is characterized by excess adipose in the upper body, centralized, and has pronounced muscle-blood development (70,71). Complications associated with this type of obesity are diabetes, gout, urinary calculous disease, and atherosclerosis. Ashwell et al (73), contends that android subjects tend to be heavier than gynoid subjects. This type of obesity generally begins in the older years because it is hypertrophic.

It is evident from the preceding discussion and the literature that although excess fat is associated with some serious diseases, it is necessary to consider the anatomic location and clinical characteristics (71,74). This is evidenced by the findings of Hartz (74). Although relative weight, waist girth, and hip girth were significantly correlated with diabetes, gallbladder disease, arthritis, hirsutism, and menstrual abnormalities, waist girth had a stronger association with disease. Hip girth actually had a negative association with disease.

Age and obesity are two important determinants of body fat distribution (70,71,73). At the younger ages adipose tissue is deposited preferentially in the hip area (gynoid) whereas in the older ages the adipose tissue is deposited in the stomach area (android). The obese deposit more of their adipose tissue in the

stomach area. The findings of Lanska et al (70), support this conclusion. In this study it was reported that relative weight, age, and a waist girth to hip girth ratio (WHR), were positively correlated. This reveals that weight put on during adulthood is androidal (abdominal). A waist diameter to thigh diameter ratio developed by Ashwell et al (73), also results in a positive correlation between actual weight, relative weight, and age. This ratio referred to as the fat distribution (FD) score was also positively correlated with the size of fat cells in the arm and waist. The score was developed to distinguish android and gynoid obesity in women. After weight loss, the FD score was found to remain constant. This implies that the fat distribution pattern is constant with weight loss and is probably genetically determined. Ashwell et al (75) also developed a fat distribution (circumference) score (CFD), to classify female fat distribution. This score utilized a ratio of waist circumference to thigh circumference and correlated positively with the FD score.

Hormonal Influence on Body Fat

Because estrogens have been reported to be taken up by adipose tissue (76,77,78), it is suggested that they may influence adipose growth and distribution. Roncari and Van (79), found that 17 beta-estradiol

increased the number of adipocyte precursors in culture. The size of the cell was not altered and 17 alpha-estradiol had no effect on the cells. Although this has not been proven in vivo, it is possible that this hormone can influence adipose growth. Edwards (80), reported that a previous pregnancy did not alter the pattern of fat distribution but postmenopausal women had an increased proportion of fat on their anterior trunk (android) as assessed by skinfolds. This second finding also suggests that estrogens play a role in adipose growth and distribution. The previous findings are in contrast with those of Lanska et al (70), who found no change in body fat among postmenopausal women.

Combined pill use was significantly associated with body fat in the study by Merians et al (47). It was reported that progestin dominant pill users had the greatest amount of body fat and the hormone balanced pill users had less body fat the the non-users. It was proposed that the anabolic (androgenic) properties of progestin stimulated appetite accounting for the increased body fat. The relation between progestin and appetite has been discussed previously. The main effect of progestin on body fat appears to be its appetite inducing effect. This results in excessive

caloric intake and results in an increase in adipose tissue growth.

Two studies investigating the relationship between combined pill use and weight and body composition have reported that there is no significant relationship (81,82). Both studies utilized a control group which had not been done previously. Goldzieher et al (82), studied 400 women and found that the percent of subjects who gained five pounds or more over the four month period was essentially the same whether they were using the combined pill or were on a placebo. Four different combined pill formulas were studied in this research. Kudzma et al (81), studied four women with one control cycle and one cycle using a combined pill. Only one combined pill was studied and no weight gain or change in body fat was reported. Thus, none of the studies have investigated the relationship between the biologic activity or potency of the varying combined pills and weight gain or body fat distribution.

MATERIALS AND METHODS

Subjects

The subjects were a sub-population of 90 white female college students selected from two undergraduate introductory courses (nutrition, health) at Kansas State University, Manhattan. All subjects were 18-26 years of age without any reported physical disorder known to affect appetite or body weight.

Of the women, 30 were identified as oral contraceptive (OCA) users. The OCA users had been using the same brand of oral contraceptive for at least three months prior to the study and had not ever used a different brand. Each OCA user was matched at the beginning of the study to two non-users, based on weight (within 4.55 kg) and height (5.08 cm). In preliminary studies it was found that OCA users weighed less than the non-users. Consequently, we matched the users and non-users to properly examine weight gain and weight control behaviors. The non-users had never taken oral contraceptives.

The students were told that the purpose of the study was to identify factors influencing body fat and weight control behavior, but were not told that the effect of oral contraceptives was the primary research interest. This study was conducted in accordance with

the policies established by the Subcommittee on Research Involving Human Subjects, Kansas State University, Manhattan. Prior to the study, the participants signed a consent form in which the procedures, risks, and benefits were explained. The study was conducted between February 3 and March 4, 1986. Copies of the application and approval letters are in Appendix E and F respectively.

Anthropometric measurements

Each student attended a private individual measurement session where an examiner measured height (without shoes) to the nearest cm and weight (in underwear or very light clothing) to the nearest 0.1 kg by procedures outlined in the 1971-74 Health and Nutrition Examination Survey (NHANES I) (83). A Detecto sliding-weight balance (Detecto Scales, Inc., Brooklyn, NY) was used to measure body weights.

Seven skinfold measurements were taken on the subjects' right side to assess fat distribution as well as percent body fat. The skinfold measurements included triceps, subscapular, axilla, chest, suprailium, abdomen, and thigh, as described by Pollock et al (84). A trained examiner took three measurements at each site using a Lange skinfold caliper (Cambridge Scientific Industries, Cambridge, MD). The measurements were used to determine body density as

described by Jackson (85) where Density = 1.0970 - $0.0004697(X_1) + 0.00000056(X_1)^2 - 0.00012828(X_4)$. X_1 is the sum of the seven skinfolds, and X_4 is the age in years. The percent body fat was determined using the Siri equation (86) where percent body fat = ((4.95/Density) - 4.5) X 100. Obesity was classified as having greater than 30% body fat.

Circumference measurements were also taken to determine body shape and fat distribution. Subjects were measured in the upright position in front of a full-length mirror. All circumference measurements were taken in the horizontal plane using a thin (6mm) flexible steel metric tape held close to the body but not tight enough to indent the skin. Three chest measurements were taken: chest-high (under arms and above the bust), chest-middle (largest part of bust), and chest-low (directly under bust). The mid-arm circumference was taken on the right arm halfway between the shoulder and elbow with the arm relaxed. The hip measurement was taken at the largest circumference around the buttocks. The right thigh was measured just below the gluteal fold. These methods for measurement are suggested in Pollock et al (84). The waist measurement was taken at the minimum circumference between the rib cage and iliac crest as described by Ashwell et al (75).

The waist girth to hip ratio (WHR) was calculated as suggested by Lanska (70) where WHR = waist circumference/hip circumference. The fat distribution (circumference) score (CFD score) as described by Ashwell et al (75) was calculated as: CFD score = $29\log_{10}$(waist circumference) - $36\log_{10}$(thigh circumference) + 10.5. Body mass index was calculated as: BMI = weight / (height)^2 where weight is reported as kilograms and height is in meters (87).

Arm muscle diameter was calculated as $(c/\pi) - S$, where c is the upper arm circumference (mm), π is 3.1429, and S is the triceps skinfold thickness (mm) (83).

Questionnaire

The women received a two-part questionnaire. In the first part, oral contraceptive users could be identified and matched to the non-users. Additionally, the OCA users were asked how long they had taken oral contraceptives so that a time frame could be applied in the second part of the questionnaire. The time frame was expressed in months and was written individually onto each questionnaire. The second part of the questionnaire included questions on weight control behavior and history, health habits, and physical symptoms which are frequently side effects of oral contraceptives. Because the time frame for OCA users

was similar to that for their paired non-users, their responses could be matched for similar time intervals. This part of the questionnaire also included an assessment tool to identify restraint eaters introduced by Herman and Polivy (88).

An initial version of the questionnaire had been pilot-tested during the previous semester on a group of 50 women having similar backgrounds to the study population.

A food frequency checklist was also administered with the initial questionnaire. Subjects were asked to check how frequently they consumed 95 foods from nine different categories. The categories were: 1) dairy products, 2) meat, poultry, and fish, 3) breads and cereals, 4) fruits and vegetables, 5) nuts and snacks, 6) candies or sweet desserts, 7) non-alcoholic beverages, 8) alcoholic beverages, and 9) miscellaneous. These 95 foods were found to be the most commonly and frequently consumed foods according to the NHANES I data (89). The serving size of each food was estimated based on age and sex according to previous data (90). Nutrient analyses were based on data from USDA Agricultural Handbook 8.

The food frequency questionnaire also queried the students on their amount of physical activity. Students were asked to indicate the number of hours

during an average 24-hour day spent in each of five different activity levels. These values were used to estimate the caloric expenditure of the subjects.

A copy of the initial questionnaire including the food frequency checklist, physical measurement recording form, and final questionnaire are in Appendix G.

Data Analysis

Data were then coded into a computer as three separate files (demographic and physical data, weight control data, food frequency checklist data) which were then merged during analysis. Data for each file were entered twice and then compared to ensure accuracy of data entry. All statistical tests were conducted using Statistical Analysis Systems (SAS) computerized programs (91).

Statistical tests used to compare continuous and ordinal categorical data of OCA users and non-users were Analysis of Variance (ANOVA) procedures, reporting F-tests and the square root of the mean square error at a significance level of $p<.05$. The ANOVA F-test procedure was chosen because of the two-group paired block design of the study. The ANOVA F-test procedure analyzes pooled data, thus rather than reporting a standard deviation of the individual treatments the pooled standard deviation (square root of the mean

square error) was reported. The square root of the mean square error is the estimate of standard deviation of experimental error (92). Additionally, hormonal potencies (estrogen, progestational) as calculated by Dickey (93) were correlated with other variables using Spearman correlations (91). Correlations between hormonal potencies and other variables were performed only on women using combined OCAs; they could not be calculated for women on sequential OCAs because the amount of hormones in those preparations varies with the menstrual cycle. A copy of the program used in the ANOVA and Spearman correlation tests is in Appendix H.

RESULTS

A description of the oral contraceptive agents (OCAs) used by the college women is shown in Table 2. Combined OCAs were used by 22 of the women and the sequential brands were used by 8 women. Over 75% of the women used one of five brands which were Ortho-Novum 1+35 (Ortho), Norinyl 1+35 (Syntex), Nordette (Wyeth), Modicon (Ortho), and Ortho-Novum 7-7-7 (Ortho).

Descriptive characteristics of the 30 OCA users and 60 matched non-users were determined from the questionnaire responses. The average ages of the OCA users and non-users were 20.8 and 20.3 years respectively. Women in both groups began menstruating at an average age of 13.0 years of age and 3.3% of both groups were smokers. The OCA users had used oral contraceptives for an average 17.7 months (range 3-36 months). The subjects were also asked if they had sexually active within the previous 3 months. Most of the OCA users, 93.3%, reported that they had been sexually active compared to only 21.7% of the non-users (data not shown).

Physical characteristics of the 30 OCA users and 60 non-users are shown in Table 3. Data are reported as the mean $\pm$ the square root of the mean squared error

Table 2. Combination and sequential oral contraceptive agents (OCAs) used by subjects*

name (manufacturer)	no. of subjects	estrogen (mcg/tablet)	progestin (mg/tablet)	estrogen potency	progestational potency
combined OCAs					
Ortho-Novum 1+35 (Ortho)	4	ethinyl estradiol (35)	norethindrone (1)	38	.38
Norinyl 1+35 (Syntex)	4	ethinyl estradiol (35)	norethindrone (1)	38	.38
Nordette (Wyeth)	4	ethinyl estradiol (30)	levonorgestrel (0.15)	25	.30
Modicon (Ortho)	3	ethinyl estradiol (35)	norethindrone (0.5)	42	.19
Lo/Ovral (Wyeth)	3	ethinyl estradiol (30)	norgestrel (0.3)	25	.30
Loestrin 1/20 (Parke-Davis)	1	ethinyl estradiol (20)	norethindrone acetate (1)	13	.44
Norinyl 1+50 (Syntex)	1	mestranol (50)	norethindrone (1)	32	.38
Loestrin 1.5/30 (Parke-Davis)	1	ethinyl estradiol (30)	norethindrone acetate (1.5)	14	.65
Demulen 1/35 (Searle)	1	ethinyl estradiol (35)	ethynodiol diacetate (1)	19	.53
sequential OCAs					
Ortho-Novum 7-7-7 (Ortho)	5	ethinyl estradiol (35)	norethindrone (0.5-1.0)	variable	variable
Ortho-Novum 10/11 (Ortho)	2	ethinyl estradiol (35)	norethindrone (0.5-1.0)	variable	variable
Tri-Norinyl (Syntex)	1	ethinyl estradiol (35)	norethindrone (0.5-1.0)	variable	variable

* Twenty-eight day regimen includes 7 placebo tablets. Information taken from (91) except for Ortho-Novum 7-7-7 and Tri-Norinyl which were taken from (4).

Table 3. Physical characteristics of oral contraceptive (OCA) users and non-users

measurement	comparison of OCA users and non-users* non-users (no.=60)	OCA users (no.=30)	correlation with hormonal potencies in oral contraceptives† estrogen (no.=22)	progestational (no.=22)
height (without shoes) (cm)	165.0 ±3.6	163.5 ±3.6	0.30	-0.01
weight (in light clothing) (kg)	59.9 ±2.3	59.6 ±2.3	0.50#	-0.03
skinfold (mm)				
tricep	18.2 ±3.6	18.3 ±3.6	0.25	0.12
chest	13.7 ±4.1	13.7 ±4.1	-0.07	0.17
axilla	10.3 ±3.3	12.0 ±3.3+	0.04	0.01
subscapular	13.0 ±4.5	14.8 ±4.5	0.07	-0.08
abdomen	21.0 ±5.1	18.9 ±5.1	0.02	0.13
suprailium	11.1 ±3.8	11.6 ±3.8	0.02	-0.01
thigh	25.6 ±4.3	26.8 ±4.3	0.17	0.35
circumference (cm)				
arm	26.8 ±1.8	27.0 ±1.8	0.45#	-0.09
high chest	84.2 ±2.5	83.6 ±2.5	0.28	0.29
bust	88.2 ±3.2	88.1 ±3.2	0.22	0.15
low chest	75.5 ±2.5	75.6 ±2.5	0.39	0.18
waist	68.8 ±2.9	69.6 ±2.9	0.10	0.15
hip	98.3 ±3.5	97.8 ±3.5	0.41	0.18
thigh	57.3 ±2.4	57.2 ±2.4	0.47#	0.02
calculated measurements				
body mass index (BMI)	22.0 ±1.2	22.3 ±1.2	0.52#	-0.13
waist-hip ratio (WHR)	0.7 ±0.1	0.7 ±0.1	-0.24	-0.17
circumference fat distribution score (CFD)	0.5 ±0.6	0.7 ±0.6	-0.45#	0.03
arm muscle diameter (mm)	67.2 ±4.5	67.4 ±4.5	0.40	-0.12
body fat (%)	21.9 ±3.4	22.3 ±3.4	0.06	0.17
percent of subjects obese	6.7	10.0	-0.14	-0.13

*Each value is mean $\pm\sqrt{MSE}$.
†Spearman's correlation coefficients for women on combined OCAs.
+Significantly different from controls, $p<.05$ (F-test).
#Significant correlation, $p<.05$ (Spearman's test).

(MSE) computed from the F-test. There were no significant differences when comparing height and weight between the two groups. However, among the OCA users estrogen potency was positively correlated with measured weight. In other words, the greater the estrogen potency the more the OCA users weighed. The axilla skinfold measurements of oral contraceptive users were significantly greater than those of the non-users, but there were no differences when comparing tricep, subscapular, abdomen, suprailium, and thigh skinfolds. There were also no differences when comparing arm, high chest, bust, low chest, waist, hip and thigh circumference measurements. There were no significant correlations between the skinfolds and hormonal potencies, but estrogen potency was positively correlated with arm and thigh circumferences.

Six physical indices were computed from the physical measurements and also are shown in Table 3. There were no significant differences between OCA users and non-users when comparing body mass index, waist-hip ratio, circumference fat distribution score, arm muscle diameter, percent body fat, and percent of obese subjects. Estrogen potency was positively correlated with circumference fat distribution score, which means that women using the higher estrogen pills were more likely to have peripheral fat distribution.

The physical measurements described above were taken at the time of the study, but we were also interested in the women's self-reported changes over the period of OCA use. Subjects were asked to report perceived changes in their circumference measurements, desire for foods, and weight loss behaviors during oral contraceptive use and a matched period of time for the non-users. Responses were based on a 5-point scale where 1=definitely less and 5=definitely more for the period of time specified in their questionnaires. These are shown in Table 4. No significant differences were found between OCA users and non-users when comparing their reported changes in circumference measurements, desire for foods, and weight loss behaviors. However, a positive correlation between estrogen potency and self-reported change in bust circumference approached statistical significance ($p<.10$). Estrogen potency was negatively correlated with the desire for dairy products.

The weight control history and practices of the 30 OCA users and non-users is shown in Table 5. Women were asked to report what they weighed before the period of OCA use or a matched period of time for non-users. Differences between these values and measured weight were used to estimate weight gain. There were no differences in weight gain when comparing

Table 4. Self-reported changes during oral contraceptive (OCA) use or a matched period of time for non-users

change reported	comparison of OCA users and non-users* non-users (no.=60)	OCA users (no.=30)	correlation with hormonal potencies in oral contraceptives† estrogen (no.=22)	progestational (no.=22)
circumference measurements				
arm	3.0 ±0.6	3.1 ±0.6	0.26	0.06
bust	3.2 ±0.7	3.2 ±0.7	0.36	-0.22
waist	3.3 ±0.9	3.3 ±0.9	0.35	-0.12
hip	3.4 ±1.0	3.4 ±1.0	0.21	-0.02
thigh	3.3 ±1.0	3.4 ±1.0	0.06	0.12
desire for foods				
dairy products	3.3 ±0.9	3.5 ±0.9	-0.43 ‡	0.20
meat, poultry, fish	3.0 ±0.7	2.9 ±0.7	-0.11	-0.12
breads and cereals	3.4 ±0.8	3.3 ±0.8	-0.05	0.10
vegetables	3.4 ±0.8	3.3 ±0.8	-0.11	0.20
fruits	3.4 ±0.7	3.5 ±0.7	-0.34	0.01
snacks (chips and nuts)	2.9 ±0.9	2.9 ±0.9	0.30	-0.42
candy or sweet desserts	2.9 ±1.0	2.9 ±1.0	-0.04	-0.22
alcoholic beverages	3.1 ±0.9	2.9 ±0.9	-0.20	0.04
overall appetite	3.1 ±0.8	3.5 ±0.8	0.27	-0.03
weight loss behaviors				
time spent trying to lose weight	3.3 ±0.9	3.5 ±0.9	0.21	-0.12
time on weight loss diets	3.2 ±0.8	3.3 ±0.8	0.22	-0.14
time spent exercising	3.1 ±1.3	3.5 ±1.3	0.20	0.01

*Each value is mean $\pm\sqrt{MSE}$. Values are the degree of change reported using a 5-point scale where 1=definitely less and 5=definitely more.
†Spearman's correlation coefficients for women on combined OCAs.
‡Significant correlation, $p<.05$ (Spearman's test).

Table 5. Weight control history and practices of oral contraceptive (OCA) users and non-users

	comparison of OCA users and non-users*		correlation with hormonal potencies in oral contraceptives†	
measurement	non-users (no.=60)	OCA users (no.=30)	estrogen (no.=22)	progestational (no.=22)
measured weight (kg)	59.9 ±2.3	59.6 ±2.3	0.50#	-0.03
reported weight before OCA use (kg) §	58.9 ±4.2	58.1 ±4.2	0.11	0.23
weight difference (kg)	1.0 ±4.3	1.5 ±4.3	0.51#	-0.42
preferred weight (kg)	55.2 ±2.6	53.8 ±2.6 ≠	0.47#	-0.01
on weight loss program at time of study (% of subjects)	30.0	33.3	-0.04	-0.09
tried to lose weight during OCA use (% of subjects) §	73.3	76.7	0.06	0.02
weight loss method tried¶				
moderate caloric restriction	93.2	87.0	0.08	0.11
exercise	93.2	87.0	-0.06	-0.04
diet pills (prescribed by physician)	0.0	0.0	0.0	0.0
diet pills (over-the-counter)	4.5	21.7 ≠	0.14	0.13
fasting or starvation	20.4	17.4	-0.03	-0.03
skipping meals	56.8	60.9	0.11	-0.14
Weight Watchers	4.5	4.3	0.14	0.13
Nutri/Systems	0.0	0.0	0.0	0.0
The Diet Center	0.0	0.0	0.0	0.0
vegetarianism	4.5	0.0	0.0	0.0
Herbalife	6.8	4.3	-0.38	0.31
liquid diet	6.8	4.3	0.34	-0.34
Ayds	0.0	4.3	0.0	0.0
low carbohydrate diet	2.3	4.3	0.0	0.0
figure salons	2.3	0.0	0.0	0.0
self-induced vomiting	2.3	4.3	0.14	0.13
laxatives	2.3	4.3	-0.16	-0.16

*Each value is mean $\pm\sqrt{MSE}$.
†Spearman's correlation coefficients for women on combined OCAs.
≠Significantly different from controls, $p<.05$ (F-test).
#Significant correlation, $p<.05$ (Spearman's test).
§Non-users matched for a similar period of time.
¶Each value is the % of subjects who had tried the weight loss method within the time frame specified.

OCA users and non-users. However when observing women on the combined OCAs, there was a positive correlation between estrogen potency and weight gain. Estrogen potency was also positively correlated with weight gain. When the women were asked what they preferred to weigh, the OCA users wanted to weigh less than the non-users, but among women using the combined OCAs, higher estrogen potency was correlated with higher preferred weight.

Weight loss practices were also studied. There were no significant differences between OCA users and non-users when comparing the number of women who were on a weight loss program at the time of the study or had tried to lose weight during OCA use or a matched period of time. The women were also asked to check if they had tried any of 17 different weight loss methods during that time, even if they had tried it only once. There were no significant differences between the two groups except that more of the OCA users had tried over-the-counter diet pills than the non-users (21.7% vs. 4.5%). Clearly, the most popular weight loss methods for both groups were moderate caloric restriction, exercise, skipping meals, and fasting or starvation.

The food frequency checklist included in the questionnaire grouped the 95 foods into nine

categories. The reported frequency of consumption was calculated on a daily basis and totaled for each category to examine food consumption patterns. The food group consumption was expressed as centigrams per day for the 30 OCA users and 60 non-users and is shown in Table 6. There were no significant differences in the food group consumption between the two groups. However, estrogen potency was negatively correlated with the consumption of fruit.

Daily nutrient intake was calculated from the food frequency checklist after converting consumption frequency (e.g. times per day, week, month, or year) to a daily basis. The energy, protein, fat, and carbohydrate intake of the 30 OCA users and 60 non-users is shown in Table 7. There were no significant differences or correlations between the two groups when comparing energy, protein, fat, and carbohydrate intake.

The vitamin and mineral intake of the 30 OCA users and 60 non-users is shown in Table 8. There were no significant differences between the two groups in vitamin and mineral intake or the percent of their Recommended Dietary Allowances (RDA) for these vitamins and minerals. Progestational potency was positively correlated with vitamin A intake and the percent of the

Table 6. Foods consumed by oral contraceptive (OCA) users and non-users

	comparison of OCA users and non-users*		correlation with hormonal potencies in oral contraceptives †	
food group ‡	non-users (no.=60)	OCA users (no.=30)	estrogen (no.=22)	progestational (no.=22)
dairy	5.3 ±4.3	5.6 ±4.3	-0.17	0.35
meat	1.6 ±1.4	1.7 ±1.4	0.05	0.04
starch	2.1 ±1.2	2.3 ±1.2	0.11	0.34
vegetable	1.8 ±1.5	1.3 ±1.5	-0.20	0.28
fruit	1.5 ±1.1	1.6 ±1.1	-0.56#	0.32
miscellaneous	1.6 ±1.4	1.8 ±1.4	0.12	-0.15
snacks	0.9 ±0.7	0.7 ±0.7	-0.26	0.15
drink	7.6 ±4.8	8.0 ±4.8	0.09	0.11
alcohol	1.4 ±4.3	2.5 ±4.3	-0.22	0.05

*Each value is mean $\pm\sqrt{MSE}$ expressed in centigrams per day.
†Spearman's correlation coefficients for women on combined OCAs.
‡Food groups are summation of individual food values for each food group in the questionnaire food frequency checklist, Appendix G.
#Significant correlation, $p<.05$ (Spearman's test).

Table 7. Daily energy, protein, fat, carbohydrate and electrolyte intake of oral contraceptive (OCA) users and non-users

	comparison of OCA users and non-users*		correlation with hormonal potencies in oral contraceptives†	
nutrient	non-users (no.=60)	OCA users (no.=30)	estrogen (no.=22)	progestational (no.=22)
energy (kcal)	2280.7 ±1128.8	2339.0 ±1128.8	-0.17	0.31
energy % of RDA	86.2 ±36.2	85.2 ±36.2	-0.13	0.27
protein (g)	91.9 ±51.3	94.3 ±51.3	-0.14	0.32
protein (% of RDA)	207.8 ±115.7	212.7 ±115.7	-0.13	0.33
protein (% of kcal)	15.9 ±2.4	16.6 ±2.4	0.34	-0.13
fat (g)	93.7 ±51.1	95.2 ±51.1	-0.12	0.15
fat (% of kcal)	36.5 ±5.1	36.4 ±5.1	0.27	-0.46
saturated fat (g)	34.3 ±19.5	36.0 ±19.5	-0.15	0.26
oleic acid (g)	32.8 ±13.8	31.8 ±13.8	-0.11	0.14
linoleic acid (g)	15.0 ±7.8	14.4 ±7.8	-0.10	0.02
cholesterol (mg)	363.2 ±266.5	353.6 ±266.5	0.11	0.14
carbohydrate (g)	260.5 ±120.4	258.3 ±120.4	-0.26	0.36
carbohydrate (% of kcal)	46.3 ±5.5	44.8 ±5.5	-0.27	0.40
sodium (mg)	3831.5 ±1341.4	3735.0 ±1341.4	0.01	0.19
potassium (mg)	3008.1 ±1066.3	3280.9 ±1066.3	-0.28	0.55

*Each value is mean $\pm\sqrt{MSE}$.
†Spearman's correlation coefficients for women on combined OCAs.

Table 8. Daily vitamin and mineral intake of oral contraceptive (OCA) users and non-users

	comparison of OCA users and non-users*		correlation with hormonal potencies in oral contraceptives†	
measurement	non-users (no.=60)	OCA users (no.=30)	estrogen (no.=22)	progestational (no.=22)
nutrient intake				
vitamin A (IU)	9059.6 ±8172.3	8483.1 ±8172.3	-0.25	0.37
vitamin C (mg)	159.9 ±89.4	136.7 ±89.4	-0.38	0.47*
thiamin (mg)	1.6 ±0.8	1.6 ±0.8	-0.11	0.29
riboflavin (mg)	2.5 ±1.5	2.6 ±1.5	-0.19	0.41
niacin (mg)	22.3 ±13.1	24.2 ±13.1	-0.03	0.28
calcium (mg)	1197.5 ±683.8	1242.7 ±683.8	-0.28	0.37
phosphorous (mg)	1708.6 ±894.6	1776.6 ±894.6	-0.17	0.39
iron (mg)	15.4 ±8.4	16.1 ±8.4	-0.06	0.29
% RDA met				
vitamin A	226.4 ±204.3	212.0 ±204.3	-0.25	0.37
vitamin C	266.5 ±148.9	227.8 ±148.9	-0.38	0.47*
thiamin	147.1 ±73.6	148.5 ±73.6	-0.11	0.34
riboflavin	191.0 ±116.6	202.4 ±116.6	-0.22	0.43*
niacin	159.9 ±93.8	174.8 ±93.8	-0.03	0.33
calcium	143.6 ±83.8	148.3 ±83.8	-0.23	0.42*
phosphorous	205.5 ±108.1	209.4 ±108.1	-0.09	0.44*
iron	85.3 ±46.8	89.1 ±46.8	-0.06	0.30

*Each value is mean $\pm\sqrt{MSE}$.
†Spearman's correlation coefficients for women on combined OCAs.
*Significant correlation, $p<.05$ (Spearman's test).

RDA met for vitamin C, riboflavin, calcium, and phosphorous.

DISCUSSION

Although several studies have implicated OCAs as an etiological factor in weight gain (33,67), the weight gain during OCA use was usually not matched for a similar period in time to that of non-users. Consequently, it is possible that the women may have gained weight over time regardless of whether they used the contraceptives. Also, several of the earlier studies involved oral contraceptives which had different formulations than the ones currently in use.

In our study the OCA users were carefully matched to non-users for weight and height. This was done because we found in two preliminary studies at this institution on a similar population that OCA users tended to weigh less than non-users (unpublished data). Thus we believed that weight control practices might be different. Also the women who participated in this study were simply told that we were investigating weight control and eating habits in college students. They were not told that the effects of oral contraceptives were the main focus of interest; consequently the power of suggestion was reduced. Weight gain was estimated as the difference between their measured weight prior to OCA use or a matched period of time for the non-users. We found that

measured body weight, body mass index, and estimated weight gain were similar in OCA users and non-users, but those measurements were positively correlated with estrogen potency in the combined oral contraceptives.

Excess estrogen has been associated with cyclic weight gain (1,5,7,8,21,30,31) characterized by fluid retention, bloating, and edema. Tyrer (23) has reported that the estrogen-induced weight gain is associated with an increase in subcutaneous fat in the breasts and hips, but to our knowledge this has not been reported elsewhere. In our study we did not find a correlation between OCA use and circumference or skinfold measurements in those areas, nor did the women report related changes. But we did observe a positive correlation between estrogen potency and circumference measurements of the upper arm and thigh; and estrogen potency was negatively correlated with the circumference fat distribution score (CFD), which means that the higher the estrogen potency in the combined OCA, the more the fat was peripherally located.

Distribution of fat has several clinical implications. Lanska et al (70) and Vauge (71) found that the android distribution of fat where fat is centrally located is associated with a greater incidence of diabetes, gout, urinary calculi, and

atherosclerosis than the gynoid distribution where the fat is more peripherally located.

A notable finding in this study was that even though the OCA users and non-users weigh the same, the OCA users wanted to weigh less than the non-users. Although the reason for this difference is not clear, a larger percent of the OCA users were sexually active, and they may have been more concerned about their physical appearance, but this reasoning is speculative. The difference in desired weights may have some importance in that concern over weight gain by OCA users may be due in part to the fact that they want to weigh less.

Even though the OCA users wanted to weigh less than the non-users, they apparently did not try harder to lose weight. A similar percentage of OCA users and non-users were on reducing programs at the time of the study; and a similar number had tried to lose weight during OCA use or a matched period of time (non-users). Specific weight loss practices tried were also similar except that more of the OCA users had tried over-the-counter diet pills, indicating perhaps a greater willingness to take pills. The greater use of over-the-counter diet pills by the OCA users merits attention in that many preparations contain phenylpropanolamine which is contraindicated for those

with hypertension (94); and elevations in blood pressure are commonly reported side effects of oral contraceptives (1,2,5,7,20,21,24).

Weight gain observed with OCA use has been attributed to an increased appetite as a result of the androgenic (anabolic) properties of progestin (1,14,23,47). Several sources consider oral contraceptives oretic drugs (60,61,62,63). In our study, the women taking OCAs did not report changes in overall appetite or desire for specific food categories that were different than those of the non-users. Estrogen potency was correlated with a reduced desire for dairy products for women using the combined OCAs, but the reason for this finding is not clear.

In summary, although we found few overall differences between OCA users and non-users when comparing physical measurements and weight control practices, estrogen potency of the contraceptive is an important factor to consider when studying weight gain in oral contraceptive users. Furthermore, the finding that OCA users are more likely to use over-the-counter diet pills merits attention because of the possible consequences on hypertension.

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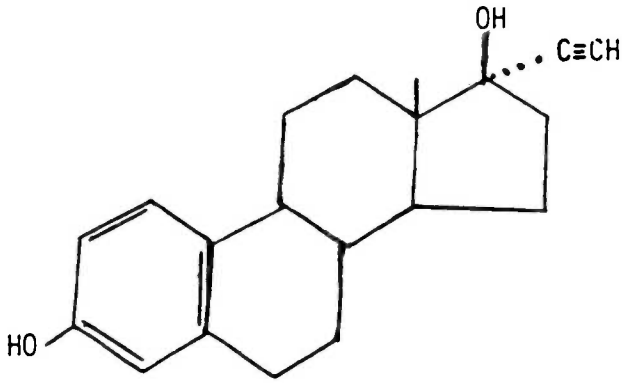
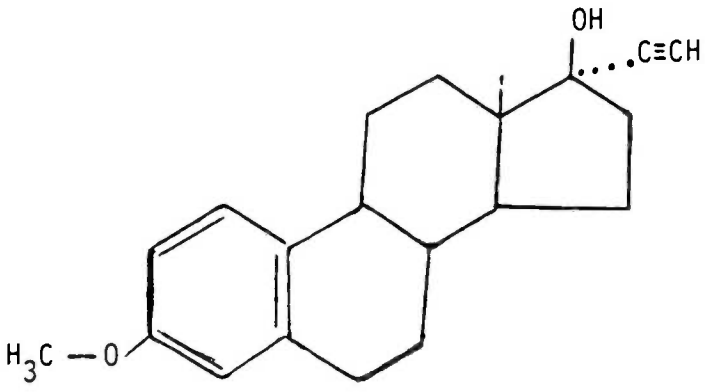
APPENDIX

APPENDIX A

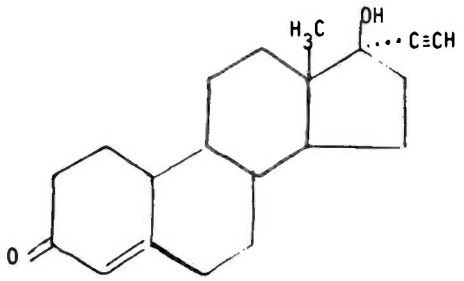


ETHINYL ESTRADIOL

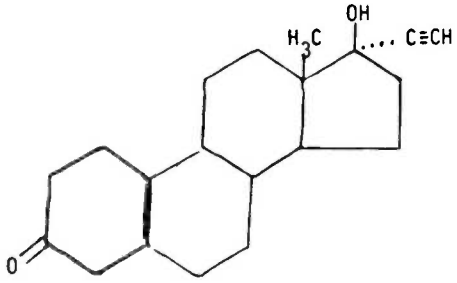


MESTRANOL

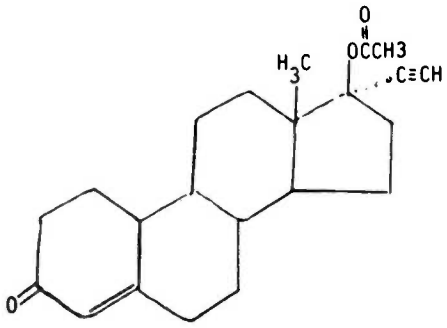
APPENDIX B



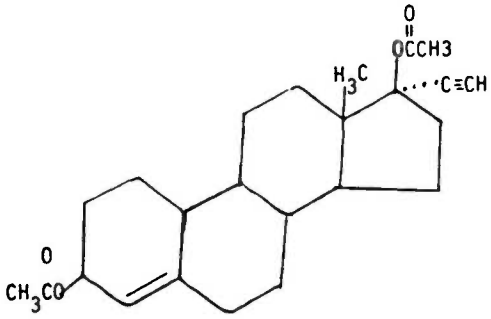
NORETHINDRONE



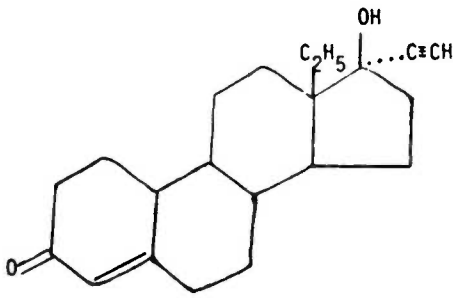
NORETHYNODREL



NORETHINDRONE ACETATE



ETHYNODIOL DIACETATE



NORGESTREL (LEVONORGESTREL)

APPENDIX C

ESTROGEN EXCESS
Nausea/vomiting
Dizziness
Edema
Gas/constipation
Irritability
Bloating
Hepatic adenoma
Cyclic weight gain
> female fat deposition
Clear vaginal discharge
Uterine enlargement
Fibroid growth
Cervical extrophia
Cystic breast changes
> breast size
Visual changes
Chloasma-hyperpigmentation
Telangiectasia
Vascular headache
Hypermenorrhea
Dysmenorrhea
Chloasma
Uterine cramps
Hypertension
Headaches(taking pill)
Lactation suppression
Breast tenderness
Thrombophlebitis
Cerebrovascular accidents
Myocardial infarction

ESTROGEN DEFICIENCY
Irritability
Hot flashes/vasomotor symptoms
Uterine prolapse
Early & midcycle spotting
< amount of menstrual flow
No withdrawal bleeding
< libido
Diminished breast size
Dry vaginal mucosa
Headaches
Depression
Pelvic relaxation
Hirsutism

PROGESTIN EXCESS
Increased appetite
Hypertension
Post-pill amennorrhea
Non-cyclic weight gain
Acne
Tiredness/fatigue
Depression
Decreased libido
Loss of hair
Cholestatic jaundice
< length of menstrual flow
Breast tenderness
Headaches(not taking pill)
Vaginal infection
Increased breast size
Dilated leg veins
< carbohydrate tolerance
Pelvic congestion

PROGESTIN DEFICIENCY
Late breakthrough bleeding & spotting
Decreased breast size
Heavy menstrual flow & clots
Delayed onset of menses
Dysmennorrhea
Weight loss

ANDROGEN EXCESS
Cholestatic jaundice
Pruritis
Increased appetite
Non-cyclic weight gain
Hirsutism
Acne
Oily skin
Increased libido

APPENDIX D

Contraindications to combined pill use:

- heart attack/stroke
- blood clots in legs or lungs
- angina pectoris
- cancer of breast or sex organs or suspected cancer of these areas
- unusual vaginal bleeding which has not been diagnosed
- liver disease or severity impaired liver function
- confirmed or suspected pregnancy

Relative Contraindications to combined pill use:

- family history of breast cancer
- breast nodules, fibrocystic breast disease, abnormal mammogram
- diabetes
- high blood pressure
- high cholesterol or triglyceride levels
- cigarette smoking
- migraine headaches
- heart, kidney, or liver disease
- epilepsy
- fibroid tumors of uterus
- gallbladder disease
- suggestive anovulation and infertility problems

APPENDIX E

KSU
KANSAS STATE UNIVERSITY

Department of Foods and Nutrition

Justin Hall
Manhattan, Kansas 66506
913-532-5508

January 20, 1986

TO: Robert D. Reeves, Ph.D., Human Subjects Committee

FROM: Kathy Grunewald, Ph.D., R.D. KKG

RE: Application for approval to use human subjects for project entitled "Oral contraceptive use in young college women: A study on weight gain and body composition"

Your approval is requested to conduct a research study involving the effects of oral contraceptive agents (OCAs) on weight gain in young college women. The project will serve as the M.S. thesis research for Ruth Litchfield, who is my advisee. The study will involve approximately 270 students: 170 from Basic Nutrition (FN 132) and Concepts of Personal Health (FCD 352); and has the approval of both course instructors. Based on information from previous classes, approximately 30% of the class women use OCAs at a given time, making it a good population to study.

Attached is the following to help you evaluate the project:

1. Application for approval to use human subjects
2. Questionnaire (informed consent forms are orange)
3. Mini-proposal explaining the rationale and procedures (written by Ruth Litchfield)

The questionnaire will query the student's health habits (green), eating habits (yellow food frequency chart), and weight control practices (blue). Some of the questions on page 2 of the green sheets are somewhat personal, i.e., OCA use, pregnancy, and sexual activity, but students will be guaranteed confidentiality. Subjects will not be told that the main purpose of the study is to study the effects of OCAs.

Subjects will receive extra credit points applied toward the course grade in both classes. They will also receive a complete computer print-out of their diet analysis, and a measurement of their per cent body fat. All subjects will be given an opportunity to participate, but only data from the women ages 18-26 will be used in the study. Students choosing not to participate will not be penalized.

I hope this meets with your approval. If you have any questions let me know.

College of Home Economics

APPLICATION FOR APPROVAL TO USE HUMAN SUBJECTS

1. ACTIVITY OR PROJECT TITLE: Oral contraceptive use in young college women: A study on weight gain and body composition

2. PROPOSED SPONSOR (IF ANY): (none)

3. Kathy Grunewald, Ph.D., R.D. | Foods and Nutrition | 532-5508
NAME (applicant must be faculty member) | DEPARTMENT | PHONE

4. RISK

A. Are there risks to human subjects? _____yes __X__no

If yes, briefly describe. (See definition of risk, page 2 of the Handbook.)

B. Describe the benefits of the research

a) to the subjects: Subjects will get a computer print-out of their diet analysis, and a measure of their per cent body fat. Students will also receive extra credit points applied toward their course grade, but if they choose not to participate, it will not penalize them in any way.

b) to the discipline/profession: Many women in the U.S. are overweight and in this study the effects of oral contraceptives are explored as one contributing factor. Because we have a rather homogeneous population with an estimated oral contraceptive use of 30%, it is a good population to study.

5. INFORMED CONSENT: General informed consent requirements are described on pages 3 and 4 of the Handbook. The written informed consent document must include the following: (1) a fair explanation of procedures to be followed, (2) description of discomforts and risks, (3) description of benefits, (4) disclosure of appropriate alternatives available, (5) an offer to answer inquiries, and (6) instructions that the subject is free to withdraw consent and participation at any time. Special informed consent policies relative to questionnaire/survey studies are described in the "Handbook Supplement" dated July, 1977.

On what page(s) of the proposal are your informed consent procedure and/or forms described? (If not a part of your proposal, the procedures and informed consent document must accompany this application.)

The consent are the orange sheets on the top of the questionnaire forms

(OVER)

6. EMERGENCIES

A. Are any possible emergencies anticipated? _____yes ___x___no

If yes, describe briefly or give the page of the proposal where these are described.

B. Describe procedures for dealing with emergencies, or give the page of the proposal on which these descriptions may be found.

7. PRIVACY: On what page of the proposal do you discuss procedures for keeping research data private? orange cover sheet This should include procedures for maintaining anonymity of subjects. Supplemental information concerning privacy of data may be discussed below. (See page 3 of the Handbook on "Safeguarding Information.")

8. STATEMENT OF AGREEMENT: The below named individual certifies that he/she has read and is willing to conduct these activities in accordance with the Handbook for Research, Development, Demonstration, or Other Activities Involving Humar Subjects. Further, the below named individual certifies that any changes in procedures from those outlined above or in the attached proposal will be cleared through Committee 8290, The Committee on Research Involving Humar Subjects via the College of Home Economics Subcommittee.

Signed Kathleen D Gemmell Date January 20, 1986
(Applicant)

Send applications to:

APPENDIX F

KSU
KANSAS STATE UNIVERSITY

Department of Foods and Nutrition

Justin Hall
Manhattan, Kansas 66506
913-532-5508

January 22, 1986

TITLE: Oral Contraceptive Use In Young College Women: A Study On Weight Gain and Body Composition

PRINCIPAL INVESTIGATOR: Katharine Grunewald, Ph.D.
Foods and Nutrition

Research activities involving no more than minimal risk and in which the only involvement of human subjects is within selected categories may be reviewed by the expedited review procedure authorized in 45CFR46:110 #9. The proposal is recommended for approval for a period of 12 months. If this proposal extends beyond 12 months from its date of approval, the proposal must again be reviewed by the subcommittee. Request for an extension of approval is the responsibility of the principal investigator. Any substantial revision in this study relative to human subjects should be reviewed again by the college subcommittee.

Robert D. Reeves, Ph.D.
Chairman
Subcommittee on Research Involving Human Subjects

APPENDIX G

Extra credit
(20 points)
NOTE THE DATES DUE

Student's copy

WEIGHT CONTROL AND EATING HABITS IN YOUNG COLLEGE STUDENTS

INSTRUCTIONS: For 20 extra credit points you can participate in a large research study involving weight control and eating habits in young college students. The project is comprised of a two-part questionnaire and a physical measurement session. You need to do all of them for the 20 extra credit points. The time schedule and description of each section follows:

1. Part I of the questionnaire is the yellow and green section attached to these orange cover sheets. It includes your background, brief medical history, medications you are taking, and a FOOD FREQUENCY CHART. Take it home and fill it out as accurately as you can. From the food frequency chart we can give you a computerized diet analysis. It will probably take you 30 minutes to complete. Sign the bottom of both of these orange pages. Tear off page a and keep it for yourself. Turn in the rest (orange page b, yellow and green sheets) Wednesday, Feb. 5 in class.

2. Physical Measurement Session. This is a 10-minute session you sign up for to be held during the week of Mon. Feb. 10 through Fri. Feb. 14. It will be held in Dickens Hall, rm 10 which is a short, old building just to the west of Justin Hall, or between Justin Hall and the Farrell Library. It will have signs on the outside doors saying "BASIC NUTRITION". I will measure for each of you your height, weight, seven skinfolds (back, abdomen, side of waist, thigh, chest, back of arm, and lateral side of chest), and five circumference measurements (arm, chest, waist, hip and thigh). From these measurements we can tell you your percent body fat, as well as your distribution of body fat. None of these measurements should cause you any discomfort and they will be performed in private by myself and a graduate student, Ruth Litchfield.

   Please wear clothing which would enable us to obtain accurate measurements. You need to wear LIGHT clothing such as GYM SHORTS, T-SHIRTS, LEOTARDS, SWIMMING SUITS, OR PANTY HOSE. You can come wearing these under heavier clothes since it is cold outside. There is a bathroom upstairs in Dickens Hall if you need to change. You can also take them off in the lab where we will be doing the measurements. Also, if it doesn't matter to you, I can take them in your underwear (women only please). But we cannot take them if you are in jeans, sweatpants, sweatshirts, skirts, etc.

3. Part II of the questionnaire you will receive at the physical assessment session (blue sheets). This section will ask you questions on your weight control habits. You will each be given a particular time frame on which to answer your questions — and this is unique to each of you. That is, you will be asked to report on your weight control practices based on the last ____ months, where we will fill in the blank for each of you. Part II of the questionnaire is due Monday, Feb. 17 in class.

You need to make an appointment for the 10-minute physical assessment session by signing your name on the clipboard on the table at the front of the class. There is one clipboard for each day, so make sure you sign up for the day and time you intended. Fill in your Date and Time below as a reminder for yourself:

Date: _____ Time: _____ Please be on time

When answering the questionnaire, please be as honest as possible. Do not worry about whether your answers are "right" from a nutritional point of view (you will not be "graded" on them). I am far more interested in your actual practices.

You may choose to terminate your participation at any point in the study, however you need to complete both parts of the questionnaire, and the physical measurements to get the full 20 extra credit points. (We need all sections to make a complete assessment). The information obtained from you will be held confidential, and will be stored on computer disk by an arbitrary ID number, and will be destroyed at the end of the study. If you have any questions about the study, you can call me at the office (532-5508) or home (539-7070), or call Ruth Litchfield at the office (532-5506) or home (539-6523).

This project has been approved by the campus committee for research involving human subjects. If you would like to participate, sign the INFORMED CONSENT below.

***

INFORMED SUBJECT CONSENT

I have read and understand the instructions given above. As indicated by my signature below, I voluntarily consent to serve as a subject in the project explained above, "Weight Control and Eating Habits in Young College Students", dated February, 1986, and agree to provide information that is accurate to the best of my knowledge.

NAME (printed) Last _____ First _____ Age _____ Signature _____ Date _____

TEAR OFF THIS SECTION AND KEEP FOR YOURSELF. TURN IN THE REST WEDNESDAY, FEBRUARY 5 IN CLASS.

Extra credit
(20 points)
NOTE THE DATES DUE

WEIGHT CONTROL AND EATING HABITS IN YOUNG COLLEGE STUDENTS

INSTRUCTIONS: For 20 extra credit points you can participate in a large research study involving weight control and eating habits in young college students. The project is comprised of a two-part questionnaire and a physical measurement session. You need to do all of them for the 20 extra credit points. The time schedule and description of each section follows:

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   Please wear clothing which would enable us to obtain accurate measurements. You need to wear LIGHT clothing such as GYM SHORTS, T-SHIRTS, LEOTARDS, SWIMMING SUITS, OR PANTY HOSE. You can come wearing these under heavier clothes since it is cold outside. There is a bathroom upstairs in Dickens Hall if you need to change. You can also take them off in the lab where we will be doing the measurements. Also, if it doesn't matter to you, I can take them in your underwear (women only please). But we cannot take them if you are in jeans, sweatpants, sweatshirts, skirts, etc.

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You may choose to terminate your participation at any point in the study, however you need to complete both parts of the questionnaire, and the physical measurements to get the full 20 extra credit points. (We need all sections to make a complete assessment). The information obtained from you will be held confidential, and will be stored on computer disk by an arbitrary ID number, and will be destroyed at the end of the study. If you have any questions about the study, you can call me at the office (532-5508) or home (539-7070), or call Ruth Litchfield at the office (532-5508) or home (539-6523).

This project has been approved by the campus committee for research involving human subjects. If you would like to participate, sign the INFORMED CONSENT below.

***

INFORMED SUBJECT CONSENT

I have read and understand the instructions given above. As indicated by my signature below, I voluntarily consent to serve as a subject in the project explained above, "Weight Control and Eating Habits in Young College Students", dated February, 1988, and agree to provide information that is accurate to the best of my knowledge.

_____ _____ _____ _____
NAME (printed) Last First Age Signature Date

TURN IN THIS SHEET (SIGNED) AND ALL THE REST (YELLOW AND GREEN) WEDNESDAY, FEBRUARY 5 IN CLASS.

Questionaire: Part I
Due Wednesday, Feb. 5 IN CLASS

Student Number_____

PHYSICAL MEASUREMENTS (Students do not fill in)

	ID	_ _ _
Skinfolds (mm):	Triceps	_ _
	Chest	_ _
	Axilla	_ _
	Subscapular	_ _
	Abdomen	_ _
	Suprailiac	_ _
	Thigh	_ _
Height without shoes (cm)		_ _ _
Weight in light clothing (kg)		_ _._
Circumference (cm):	Arm	_ _
	Chest	_ _ _
	Waist	_ _ _
	Hip	_ _ _
	Thigh	_ _ _

BACKGROUND

(computer scoring only)

Circle each answer or fill in the blank as indicated

1. Gender: 1. male 2. female

2. Height (in stocking feet) _____in

3. Weight (in light clothing) _____lbs

4. Age (at last birthday):_____years (Age) _ _

5. Marital status: 1. single 2. married 3. separated or divorced 4. widowed _

6. How many biological children do you have? _____ _

7. Race/Origin (optional) _
   1. White (non-hispanic) 4. American Indian
   2. Black 5. Hispanic
   3. Asian or Pacific 6. Other (specify) _____

8. Academic classification: 1. Freshman 2. Sophomore 3. Junior 4. Senior 5. Other _

9. Have you ever been diagnosed as having any of the following diseases (check with an "I" if you have)

Hypothyroidism__	Anemia__
Hyperthyroidism__	Hypoglycemia__
Hypogonadism__	Heart disease__
Hypothalmic injury__	Gallbladder disease__
Ovarian cysts ___	Lactose intolerance__
Tumors of the pancreas__	Kidney disease__
Diabetes__	Hyperglycemia__
Consistently high blood pressure__ (higher than 140/90)	Ulcers__

10. Are you presently taking any medication (excluding birth control pills)? 1. yes 1. no _

    If so, what is the medication (name) _____

    And what are you taking it for?_____

11. Do you presently smoke cigarettes? 1. yes 2. no _

WOMEN ONLY (Men do not answer - go to next page)

12. How old were you when your periods started? _____years old — —

13. Are you pregnant? 1. yes 2. no —

14. Have you ever been pregnant? 1. yes 2. no —

15. Are you breastfeeding right now? 1. yes 2. no —

16. Have you been sexually active in the last 3 months? 1. yes 2. no —

17. Have you ever been on oral contraceptives? 1. yes 2. no —

18. Are you presently on oral contraceptives? —
1. yes (go to question 19)
2. no (stop - go to the next page)

19. Which brand of oral contraceptive are you presently on? (circle) —

1. Lo/Ovral
2. Micronor
3. Modicon
4. Norgette
5. Norinyl 1 + 35
6. Norinyl 1 + 50
7. Norlestin 1+ 50
8. Ovculen
9. Ortho-Novum 1 + 35
10. Ortho-Novum 1 + 50
11. Ortho-Novum 7-7-7
12. Ovrette
13. Ovulen
14. Ovral
15. Tripnasil
16. Other (specify) _____

20. How many months have you been taking that brand? _____months. — —

21. Did you take another brand before that? 1. yes 2. no —

If so, what was the name of that brand? _____

and why did you switch brands?_____

22. Did you previously take a different dosage of oral contraceptive (the same brand)? —
1. yes 2. no

If so, what was the dosage that you previously took?_____

23. What is the primary reason that you take oral contraceptives? —
1. birth control
2. regulate menstrual cycle
3. reduce menstrual cramps
4. lessen migraine headaches
5. other (specify)_____

24. What TIME OF THE DAY do you usually take the oral contraceptive? —
1. Morning (5am - 11am)
2. Mid-Day (11am - 4 pm)
3. Late afternoon (4pm-7pm)
4. Evening (7pm-11pm)
5. Night (11pm-5am)

DO NOT FILL IN

Age __ __

Sex __ Weight (kg) __ __.__

Level1	Level2	Level3	Level4	Level5

FOR (ID) __ __ __

DAY __ __/__ __/__ __

ACTIVITY LEVEL

The purpose of this section is to get a rough approximation of how many calories you expend in an average day. This is important because the need for some vitamins is based on your caloric expenditure. Below, you will see that all activities are divided into 5 categories or "levels" with Level 1 being the lightest activity level and Level 5 being the highest activity level. FOR EACH LEVEL FILL IN THE TOTAL NUMBER OF HOURS A DAY. If you spend alot of time doing something that is not listed below (eg dressing or getting ready), find an activity level in the table that you think it is closest to, and count it as part of that activity level. All 5 levels should add up to 24 hours a day and represent your present activity levels.

LEVEL		FILL IN No. of Hours a Day
1	SLEEPING OR RECLINING	_____
2	LIGHT ACTIVITY Most desk jobs, typing, standing, driving an auto, sewing, light dusting, and vacuuming. (Most people average their largest number of hours at this level).	_____
3	MODERATE ACTIVITY Walking 2 1/2 to 3 mph), doing carpentry or restaurant work, playing ping-pong or volleyball, washing windows, and scrubbing or waxing floors, (Many people average a few hours at this level).	_____
4	VERY ACTIVE Walking briskly 3 1/2 to 4 mph), weeding and hoeing, dancing, playing strenous tennis, skiing or biking rapidly. (A few people average some time at this level).	_____
5	EXCEPTIONALLY ACTIVE Constant lap swimming, playing a vigorous basketball or football game or jogging. (Very few people average any time at this level).	24 hrs

DIET ANALYSIS

INSTRUCTIONS: For each group of foods, circle the number of times you consume the food and ALSO the time unit for that consumption. For example, if you consumed skim milk or skim milk drinks 3 times a day, you'd circle a "3" for the number of times, and a "D" for the time unit. If you consumed this group only once a week, you'd circle a "1" for the number of times and a "W" for the time unit. This should reflect your present eating habits.

1. MILK OR MILK DRINKS
(including hot chocolate, milk shakes, chocolate milk drinks)

	Never	No. of times									Per Day	Per Week	Per Month	Per Year
Skim milk or skim milk drinks (including reconstituted dry milk)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y
Low-fat or low-fat milk drinks	0	1	2	3	4	5	6	7	8	9	D	W	M	Y
Whole milk or whole milk drinks	0	1	2	3	4	5	6	7	8	9	D	W	M	Y

2. CHEESE OR COTTAGE CHEESE

											Per Day	Per Week	Per Month	Per Year
Cottage cheese or ricotta cheese	0	1	2	3	4	5	6	7	8	9	D	W	M	Y
Other cheeses such as American, Swiss, Cheddar, or Mozzarella	0	1	2	3	4	5	6	7	8	9	D	W	M	Y

	Never	No. of times									Per				
											Day	Week	Month	Year	
3. OTHER DAIRY PRODUCTS															
Yogurt	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Pudding	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Ice Cream	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Sour Cream or Cream Cheese	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Butter or Margarine	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Eggs	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
4. MEAT															
Hamburger	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Hot dogs or Sausage	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	***
Luncheon meats (bologna, salami, or chicken/turkey roll)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Beef or steak	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Pork or ham	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Bacon	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Liver	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Other meats (veal, lamb, or venison)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
5. POULTRY (chicken, turkey, or duck)															
Fried poultry	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Baked or broiled poultry	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
6. FISH (other than shellfish)															
Canned fish (tuna, salmon, or sardines)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Fried fish	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Baked, broiled, or cooked fish	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
7. SHELLFISH (shrimp, crab, or oysters)															
Raw shellfish	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Fried shellfish	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Baked, broiled, or cooked shellfish	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
8. CEREALS, BREADS, PASTA or STARCHES															
Cooked breakfast cereals	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Ready-to-eat breakfast cereals	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Waffles, pancakes, or French toast (sum of these)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Breads, rolls, muffins, and biscuits, white or whole grain (sum of these)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	***
Rice	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Pasta, macaroni, noodles, or tortilla	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Fried potatoes	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Boiled or baked potatoes	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	

9. VEGETABLES (canned, fresh, or frozen, but NOT juice)	Never	No. of times									Per Day	Per Week	Per Month	Per Year	
Yams or sweet potatoes	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Corn	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Brussel sprouts or cabbage	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Squash, zucchini, or eggplant	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Cauliflower	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Broccoli	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Carrots	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Tomatoes	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Olives	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Lettuce	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Spinach or other greens	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Green peas	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Green or yellow beans	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Dry beans, peas, or lentils	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	•••
Soybeans or soybean products such as tofu or textured vegetable protein	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Other vegetables such as mushrooms, peppers, turnips, or beets	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	

10. FRUIT (fresh, frozen or canned but NOT juice)											Per Day	Per Week	Per Month	Per Year	
Citrus fruits	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Apples or pears	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Peaches or plums	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Cherries or berries	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Bananas	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Melons	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Raisins or other dried fruit	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Other fruits such as fruit cocktail, grapes, pineapple, or nectarines.	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	

11. Miscellaneous Foods											Per Day	Per Week	Per Month	Per Year	
Peanut butter	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Jams and jellies	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Pancake syrup	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Sugar or honey added to food (Do not include that added to coffee or tea)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Pizza	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Soups such as broth, consume, or bouillon	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Other soups	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Meat gravies	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	•••
White or cheese sauce	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Tomato sauce or Ketchup	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Mayonnaise	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	

	Never	No. of times									Per: Day	Week	Month	Year	
Low-cal salad dressing	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Regular salad dressing	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Mustard - condiments	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
12. NUTS AND SNACKS															
Nuts	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Crackers	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Potato chips or corn chips	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Other salted snacks such as popcorn or pretzels	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
13. CANDIES OR SWEET DESSERTS											Day	Week	Month	Year	
Candies	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Other sweets such as cookies, cakes, pies, donuts, danish, or pastries	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Cake icing	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Chocolate syrup	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Sherbert	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
14. NON-ALCOHOLIC BEVERAGES											Day	Week	Month	Year	
Fruit or vegetable juices	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Fruit drinks (such as lemonade or Hawaiian Punch)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Low-cal carbonated soft drinks (such as TAB or Diet Pepsi)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	eee
Regular carbonated soft drinks (such as Coke or Pepsi)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Beverage mixes	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Coffee or tea	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Instant coffee or tea with sugar included in the mix	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Coffee or tea with sugar added	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Do you usually add non-dairy creamers?			( ) yes				( ) no								
Do you usually add milk or cream?			( ) yes				( ) no								
15. ALCOHOLIC BEVERAGES											Day	Week	Month	Year	
Beer	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Wine	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
Liquor or liqueur	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	
16. OTHER											Day	Week	Month	Year	
Skim milk or skim milk drinks (including reconstituted dry milk)	0	1	2	3	4	5	6	7	8	9	D	W	M	Y	

Questionnaires: Part II
Due Monday, Feb. 17, in class

Student Number_____

ANSWER THE FOLLOWING QUESTIONS BASED ON THE LAST |___| MONTHS

ID ___

1. During the last |___| months, indicate the amount of food usually consumed at each of the following times:

	Small or none	Moderately Small	Medium	Moderately Large	Large	
Morning (5am-11am)- - -	1	2	3	4	5	_
Mid-Day (11am-4pm)- - -	1	2	3	4	5	_
Late afternoon(4pm-7pm)-	1	2	3	4	5	_
Evening (7pm-11pm)- - -	1	2	3	4	5	_
Night (11pm-5am)- - - -	1	2	3	4	5	_

2. During what time of the day did you usually eat MOST of your food? _
   1. Morning (5am-11am)
   2. Mid-Day (11am-4pm)
   3. Late afternoon (4pm-7pm)
   4. Evening (7pm-11pm)
   5. Night (11pm-5am)

3. During what time of the day did you usually eat the LEAST of your food? _
   1. Morning (5am-11am)
   2. Mid-Day (11am-4pm)
   3. Late afternoon (4pm-7pm)
   4. Evening (7pm-11pm)
   5. Night (11pm-5am)

4. During what time of the day were you most likely to eat SNACKS in between meals? _
   1. Morning (5am-11am)
   2. Mid-Day (11am-4pm)
   3. Late afternoon (4pm-7pm)
   4. Evening (7pm-11pm)
   5. Night (11pm-5am)

5. How often did you eat breakfast? _
   1. rarely—less than once a week
   2. once a week
   3. 2-3 times a week
   4. 4-6 times a week
   5. once a day

6. How often did you eat lunch? _
   1. rarely—less than once a week
   2. once a week
   3. 2-3 times a week
   4. 4-6 times a week
   5. once a day

7. How often did you eat dinner (supper)? _
   1. rarely—less than once a week
   2. once a week
   3. 2-3 times a week
   4. 4-6 times a week
   5. once a day

8. Which of the following best describes the rate at which you usually ate? (compared to other people) _
   1. Much slower than average
   2. Somewhat slower than average
   3. Average
   4. Somewhat faster than average
   5. Much faster than average

9. How often did you get vigorous continuous exercise lasting at least 20 minutes at a time? _
   Examples of vigorous continuous exercise are: running, fast cycling, swimming, jumping rope, dancing, racquetball, or tennis. They must be at least 20 minutes without stopping to count.
   1. rarely—less than once a week
   2. once a week
   3. 2-3 times a week
   4. 4-6 times a week
   5. once a day
   6. more than once a day

10. If you exercised once a week or more, WHY did you exercise? (Check all that apply)
    1. (N/A) I exercise less than once a week
    2. Lose weight___
    3. Enjoy it___
    4. Relaxation ___
    5. Relieve stress___
    6. Be with others (social)___
    7. Keep physically fit___
    8. My boy/girlfriend or spouse wants me to___
    9. Other (specify)_____

11. How often did you take vitamin and/or mineral pills? _
    1. rarely-less than once a week
    2. once a week
    3. 2-3 times a week
    4. 4-6 times a week
    5. once a day
    6. more than once a day

WEIGHT CONTROL

12. What is your weight NOW? _____pounds

13. What was your weight |___| months ago? _____lbs ___

14. What would you LIKE to weigh? (give one weight only, not a range) _____lbs ___

15. Are you presently on a weight loss program? 1. yes 2. no _

16. Have you tried to lose weight during the last |___| months?
1. yes (answer methods used below)
2. no (skip to question 17 below) _

If so, check ("I") any of the following methods you have tried during that time, even if you have tried it only once.

Moderate calorie restriction__
Exercise__
Diet pills (prescribed by doctor) __
Diet pills (not prescribed by doctor) __
Fasting or starvation__

Skip meals__
Weight Watchers__
Nutri/Systems__
The Diet Center__
Vegetarianism__

Herbalife __
Liquid diet__
Ayds__
Low carbohydrate diet__
Figure salons__

Self-induced vomiting__
Laxatives__
Other (specify) _____

17. During the last |___| months, about what % of the time have you spent trying to lose weight? _
1. 0 (none) 2. 1-10% 3. 11-25% 4. 26-50% 5. 51-75% 6. more than 75%

18. The following questions pertain to your distribution of body fat and how weight gain/loss affects it. Circle the number corresponding to the appropriate column.

	Above the Waist	Waist/Stomach	Below the waist	
What region of your body has the most fat? - - - - - - -	1	2	3	_
What region of your body has the least fat? - - - - - -	1	2	3	_
When you gain weight, where do you first notice it? - - -	1	2	3	_
When you lose weight, where do you first notice it? - - -	1	2	3	_
What region of your body is the most difficult to reduce?	1	2	3	_

19. During the last |___| months has your body shape changed? (Note that "3" means no change)

	definitely smaller	somewhat smaller	no change	somewhat larger	definitely larger	
Chest/bust - - - - - - -	1	2	3	4	5	_
Upper arms - - - - - - - -	1	2	3	4	5	_
Waist/stomach - - - - - -	1	2	3	4	5	_
Hips/buttocks - - - - - -	1	2	3	4	5	_
Thighs - - - - - - - - - -	1	2	3	4	5	_

20. During the last |___| months have any of the following changed? (Note that "3" means no change)

	definitely less	somewhat less	no change	somewhat more	definitely more	
Overall appetite for food	1	2	3	4	5	_
Time trying to lose weight	1	2	3	4	5	_
Time on weight loss diets	1	2	3	4	5	_
Time spent exercising	1	2	3	4	5	_

21. During the last |___| months, has your desire for particular foods changed? (Note that "3" means no change).

	definitely less	somewhat less	no change	somewhat more	definitely more	
Milk or dairy products	1	2	3	4	5	_
Meat, poultry, or fish	1	2	3	4	5	_
Cereal, breads, pasta or starchy foods	1	2	3	4	5	_
Vegetables	1	2	3	4	5	_
Fruits	1	2	3	4	5	_
Snacks such as chips and nuts	1	2	3	4	5	_
Candies or sweet desserts	1	2	3	4	5	_
Alcoholic beverages	1	2	3	4	5	_
Non-alcoholic beverages such as coffee, tea, soft drinks, or fruit or vegetable drinks	1	2	3	4	5	_

22. During the last |___| months have you experienced more or less of the following physical conditions? (Note that a "3" means no change)

	definitely less	somewhat less	no change	somewhat more	definitely more	
Nausea	1	2	3	4	5	_
Vomiting	1	2	3	4	5	_
Headache	1	2	3	4	5	_
Breast tenderness	1	2	3	4	5	_
Chloasma (brown spots on the skin)	1	2	3	4	5	_
Constipation	1	2	3	4	5	_
Diarrhea	1	2	3	4	5	_
Gas	1	2	3	4	5	_
Fatigue	1	2	3	4	5	_
Depression	1	2	3	4	5	_
Acne	1	2	3	4	5	_
Menstrual bleeding	1	2	3	4	5	_
Spotting between periods	1	2	3	4	5	_
Amenorrhea	1	2	3	4	5	_
Menstrual cramps	1	2	3	4	5	_
Bloating	1	2	3	4	5	_
Tension/stress	1	2	3	4	5	_
High blood pressure	1	2	3	4	5	_
Overall appetite for food	1	2	3	4	5	_

THIS IS AN IMPORTANT QUESTIONNAIRE

PLEASE ANSWER ALL QUESTIONS AS HONESTLY AS YOU CAN

DO NOT FILL IN
Computer scoring only

1. How many pounds over your desired weight were you at your maximum weight? (1/3) __

_____pounds

2. How often are you dieting? __
   1. rarely 2. sometimes 3. usually 4. always

3. Which best describes your behavior after you have eaten a "not allowed" food while on your diet? __
   0. return to diet
   1. stop eating for an extended period of time in order to compensate
   2. continue on a splurge, eating other "not allowed" foods

4. What is the maximum amount of weight that you have ever lost within 1 MONTH? (1/3) __

_____pounds

5. What is your maximum weight gain within a WEEK? (1/3) __

_____pounds

6. In a typical week, how much does your weight fluctuate (maximum-minimum)? (1/3) __

_____pounds

7. Would a weight fluctuation of 5 pounds affect the way you live your life? __
   0. not at all 1. slightly 2. moderately 3. very much

8. Do you eat sensibly before others and make up for it alone? __
   0. never 1. rarely 2. often 3. always

9. Do you give too much time and thought to food? __
   0. never 1. rarely 2. often 3. always

10. Do you have feelings of guilt after overeating? __
    0. never 1. rarely 2. often 3. always

11. How conscious are you of what you're eating? __
    0. not at all 1. slightly 2. moderately 3. extremely

12. How would you describe your weight during childhood (ages 5-11)? __
    1. definitely underweight 4. somewhat overweight
    2. somewhat underweight 3. about right 5. definitely overweight

13. How would you describe your weight during adolescence (ages 12-18)? __
    1. definitely underweight 4. somewhat overweight
    2. somewhat underweight 3. about right 5. definitely overweight

14. How would you describe your weight NOW? __
    1. definitely underweight 4. somewhat overweight
    2. somewhat underweight 3. about right 5. definitely overweight

15. How would you describe your biological mother's weight (right now)? __
    1. definitely underweight 3. about right 5. definitely overweight
    2. somewhat underweight 4. somewhat overweight 6. N/A (biological mother not living)

16. How would you describe your biological father's weight (right now)? __
    1. definitely underweight 3. about right 5. definitely overweight
    2. somewhat underweight 4. somewhat overweight 6. N/A (biological father not living)

PHYSICAL MEASUREMENTS
Spring 1986

Student Number_____

ID _ _ _

Trial _ _

SKINFOLDS:	Triceps	(_ _ _)	_ _
	Chest	(_ _ _)	_ _
	Axilla	(_ _ _)	_ _
	Subscap	(_ _ _)	_ _
	Abdomen	(_ _ _)	_ _
	Suprail	(_ _ _)	_ _
	Thigh	(_ _ _)	_ _

HEIGHT (w/o shoes) (cm)		_ _ _
WEIGHT (in lt clothing) (kg)		_ _._
CIRCUMFERENCE (cm):	Arm	_ _._
	Hi Chest	_ _._
	Mid-Chest	_ _._
	Lo-Chest	_ _._
	Waist	_ _._
	Hip	_ _._
	Thigh	_ _._
ELBOW BREADTH (mm)		_ _

Appendix H

```
//LATCHFLD JOB (484726453,GF31015Y,,9),'RUTH',TIME=(3,59)
/*PASSWORD      [illegible]
/*REGION        500K
// EXEC SAS
//DD1 DD DSN=OS15Y.OCAFF,DISP=SHR
//SYSIN DD *
DATA ONE;
INPUT (ID TRIAL NUMBER AGE MARITAL CHILDREN RACE CLASS MEDS SMOKE
    PERIODS PREGNDR PREGPREV LACTATE SEXACTIV OCAPREV OCANOW
    OCBRAND MONTHS BRANPREV DOSPREV OCREASON OCTIME TRICEP CHEST
    AXILLA SUBSCAP ABDOMEN SUPRAILI THIGH HEIGHT WEIGHT ARMCIRC
    HICHEST MIDCHEST LOWCHEST WAISTCIR HIPCIRC THIGHCIR ELBOWBRE
    AMTMORN AMTNOON AMTAFTER AMTEVEN AMTNIGHT MOSTFOOD LEASFOOD
    SNACKTIM BREAK LUNCH SUPPER EATRATE EXERCISE EXREASON VITAMIN)
    (3. 2. 1. 2. 5*1. 2. 6*1. 2*2. 4*1. 7*2. 3. 4.1 3.1 5*4.1
    3.1 2. 15*1.);
SKINFOLD=(TRICEP+CHEST+AXILLA+SUBSCAP+ABDOMEN+SUPRAILI+THIGH);
DENSITY=1.0970-(0.00046971*SKINFOLD)+(0.00000056*SKINFOLD*SKINFOLD)
    -(0.00012828*AGE);
FATPER=((4.95/DENSITY)-4.5)*100;
IF FATPER>30 THEN OBESE=1; IF FATPER=30 OR FATPER<30 THEN OBESE=2;
BMI=(WEIGHT)/((HEIGHT/100)*(HEIGHT/100));
CFD=(29*(LOG10(WAISTCIR)))-(36*(LOG10(THIGHCIR)))+10.5;
WHR=WAISTCIR/HIPCIRC;
WHR=WAISTCIR/MIDCHEST;
CHR=MIDCHEST/HIPCIRC;
AMD=((ARMCIRC/.31429)-TRICEP);
HT=HEIGHT/2.54; ELB=ELBOWBRE/25.4; WT=WEIGHT*2.2;
IF HT<63 AND ELB<2.25 THEN FRAME=1; IF HT<63 AND ELB>2.25 AND ELB<2.5
    THEN FRAME=2; IF HT<63 AND ELB>2.5 THEN FRAME=3;
IF HT>63 AND HT<71 AND ELB<2.375 THEN FRAME=1; IF HT>63 AND HT<71 AND
    ELB>2.375 AND ELB<2.625 THEN FRAME=2; IF HT>63 AND HT<71 AND
    ELB>2.625 THEN FRAME=3;
IF HT>71 AND ELB<2.5 THEN FRAME=1; IF HT>71 AND ELB>2.5 AND ELB<2.75
    THEN FRAME=2; IF HT>71 AND ELB>2.75 THEN FRAME=3;
IF OCBRAND=1 OR OCBRAND=4 THEN ESTROGEN=25; IF OCBRAND=2 THEN ESTROGEN=13;
    IF OCBRAND=3 THEN ESTROGEN=[illegible]; IF OCBRAND=5 OR OCBRAND=9 THEN
    ESTROGEN=35; IF OCBRAND=6 THEN ESTROGEN=[illegible]; IF OCBRAND=7 THEN
    ESTROGEN=14; IF OCBRAND=8 THEN ESTROGEN=17;
IF OCBRAND=1 OR OCBRAND=4 THEN PROGEST=.50; IF OCBRAND=2 THEN PROGEST=.64;
    IF OCBRAND=3 THEN PROGEST=.19; IF OCBRAND=5 OR OCBRAND=6 OR OCBRAND=9
    THEN PROGEST=[illegible]; IF OCBRAND=7 THEN PROGEST=.05; IF OCBRAND=8 THEN
    PROGEST=.53;
IF OCBRAND=1 THEN ANDROGEN=.43; IF OCBRAND=2 THEN ANDROGEN=.52; IF
    OCBRAND=3 THEN ANDROGEN=.17; IF OCBRAND=4 THEN ANDROGEN=.72; IF
    OCBRAND=5 OR OCBRAND=6 OR OCBRAND=9 THEN ANDROGEN=.04; IF OCBRAND=7
    THEN ANDROGEN=.79; IF OCBRAND=8 THEN ANDROGEN=.[illegible];
CARDS;
$ADD OCADATA1
PROC SORT; BY ID;
DATA TWO;
INPUT (ID PREVWEIG LIKEWEIG PRESLOSE TRYLOSE MODERATE EXER PILLSDOC
    PILLSBUY FASTING SKIPMEAL [illegible] NUTRISYS DIETCENT VEGETAR HERBALIF
    [illegible] AYDS [illegible] SARF [illegible] STADIET TIMESPER MOSTFAT
    LEASTFAT GAINFST LOSEFST LOSEDIFF CHANHOUT CHANARMS CHANWAIS

    CHANHIPS CHANTHIG APPETIT TIMELOSE TIMEDIET TIMEEXER DAIRY2
    MEATS2 STARCHE2 VEGGIES2 FRUITS2 SNACKS2 CANDY2 ALCOHOL2 DRINKS2
    NAUSEA VOMIT HEADACHE BREAST CHLOASMA CONSTIP DIARRHEA GAS
    FATIGUE DEPRESS ACNE MENSBLED SPOTTING AMENORR MENSCRAM
    BLOATING TENSION [illegible] APPETIT MAX[illegible] DIETTIME BEHAVIOR MONLOSS
    WEEKLOSS WEEKFLUC FLUCLIFE BINGE [illegible] THOUGHT GUILTY CONCIOUS
    CHILWEIG ADOLWEIG PREGWEIG MOTHER FATHER EXER1 EXER2 EXER3
    EXER4 EXER5 PILL1 PILL2)(3*3. 63*1. 2. 15*1. 3*3.1 2*2.1 2*4.);
TEST=MAX[illegible]+DIETTIME+BEHAVIOR+MONLOSS+WEEKLOSS+WEEKFLUC+FLUCLIFE+
    BINGE[illegible]+THOUGHT+GUILTY+CONCIOUS;
IF TEST=17 OR TEST>17 THEN RESTRAIN=1; IF TEST<17 THEN RESTRAIN=2;
METHODS=MODERATE+EXER+PILLSDOC+PILLSBUY+FASTING+SKIPMEAL+[illegible]+NUTRISYS+
    DIETCENT+VEGETAR+HERBALIF+[illegible]+AYDS+[illegible]+[illegible]+SARF+[illegible]+
    OTHDIET;
CARDS;
$ADD OCADATA2
PROC SORT; BY ID;
DATA THREE;
INFILE DD1;
ARRAY FOODREC (J) FDR1-FDR75;
ARRAY AMOUNT (J) AMNT1-AMNT75;
INPUT @3 ID 3-7;
INPUT @5 DATE $7.;
INPUT COMP $12. 3;
DO J=1 TO 10;
INPUT +2 FOODREC 4. AMOUNT 5.2 @;
END;
JSTRT=11;
DO I=1 TO 3;
JSTP=JSTRT+10;
INPUT;
INPUT @2 CHECK $5. @1 @;
IF CHECK='FOODS' THEN GO TO S2;
DO J=JSTRT TO JSTP;
INPUT +2 FOODREC 4. AMOUNT 6.2 @;
IF FOODREC=. THEN GO TO S1;
END;
JSTRT=JSTP+1;
END;
S1:
INPUT;
S2:INPUT;NUMFDS=J-1;
INPUT;
INPUT @11 ENERGY 7.2 @19 PROT 5.2 @25 VITA 8.2 @33 VITC 5.2
@41 ASCRB 7.2 @49 THIAM 7.2 @57 [illegible] 7.2 @65 NIACIN 7.2 @73 VITB6 7.2
@78 FOLAC 7.2 @85 VITB12 7.2 @93 CA 5.2 @99 PHOS 6.2 @105 MG 7.2
@113 FE 7.2 @120 ZINC 5.2;
```

```
INPUT;
INPUT @13 ENERGYR PACTR VITAR VIT_P ASCORR THIAMR RIBOR NIACINR VIT_6R
      FOLIC_R VIT_B12R CALCIUMR PHOSR MGR IRONR ZINCR;
INPUT;
INPUT;
INPUT @11 PORTION 7.2 @13 H2O 3.1 @2. FAT 7.2 @71 CARBO 7.2 FIBER
      ASH 44-49 1 SODIUM 50-56 2 SATFAT 57-63 2 SATUR MUFA @30 PUFA 3.1
      @63 CHOL 7.2 @70 REFUS 3.1 ENERGYD OFIBER CU MN PANTA;
INPUT @33 CHOP 4.1 @50 FATP 4.1 @71 PROTP 4.1;
INPUT;
INPUT;
INPUT @60 OLEIC LINOLEIC;
INPUT;
INPUT;
INPUT;
INPUT;
INPUT;
INPUT @10 ENERGING PROING VITAING VITCING ASCORING THIAMING RIBOING
      NIACNING VIT6ING FOLICING B12ING CALCMING PHOSING MGING IRONING
      ZINCING;
INPUT;
INPUT;
DO J=1 TO NUMFDS;
FOODID=FOODREC;
FOODAMT=AMOUNT;
OUTPUT;
END;
DROP FDR1-FDR95 AMT1-AMT95 STRT STP NUMFDS COMP I J CHICK;
PROC SORT; BY ID;
DATA FOOD;
SET;
BY ID;
DROP FOODID FOODAMT;
ARRAY FOOD SKIMMLK LOFATMLK WHOLEMLK CCHEESE RCHEESE YOGHURT PUDDING
      ICECREAM SOURCRM BUTTER EGGS HAMBURG HOTDOGS LUNCHMTS BEEF PORK
      BACON LIVER OTHERMTS FRYPOULT BAKPOULT CANFISH FRYFISH COOKFISH
      RAWSHELL FRYSHELL CKSHELL CKCEREAL RTCEREAL WAFFLES BREADS
      RICE PASTA FRYPOTA OTHPOTA YAMS CORN BRUSSEL SQUASH CAULIF
      BROCCOLI CARROTS TOMATOES OLIVES LETTUCE SPINACH GRPEAS GRBEANS
      DRYBEANS SOYBEANS OTHERVEG CITRUS APPLES PEACHES CHERRIES
      BANANAS MELONS RAISINS OTHERFRT PEANUTS JAMS SYRUP SUGAR PIZZA
      BROTH OTHERSOUP GRAVY SAUCE KETCHUP MAYONN COSALAD RESSALAD MUSTARD
      NUTS CRACKERS CHIPS OTHERSNK CANDY CAKE ICING CHOCSYRP SHERBERT
      FRJUICE FRDRINKS COFTDR RSOFTDR BEVERAGE RCOFFEE ICOFFEE SCOFFEE
      NONDAIRY CREAM BEER WINE LIQUOR;
RETAIN SKIMMLK--LIQUOR;
IF FIRST.ID THEN DO OVER FOOD;
      FOOD=0;
      END;
IF FOODID=5571 THEN SKIMMLK=FOODAMT;     IF FOODID=2121 THEN LOFATMLK=FOODAMT;
IF FOODID=5705 THEN WHOLEMLK=FOODAMT;    IF FOODID=[illegible] THEN CCHEESE=FOODAMT;
IF FOODID=[illegible] THEN RCHEESE=FOODAMT;   IF FOODID=57[illegible] THEN YOGHURT=FOODAMT;
IF FOODID=[illegible] THEN PUDDING=FOODAMT;   IF FOODID=[illegible] THEN ICECREAM=FOODAMT;
IF FOODID=[illegible] THEN SOURCRM=FOODAMT;   IF FOODID=1[illegible]3 THEN BUTTER=FOODAMT;
IF FOODID=[illegible]5 THEN EGGS=FOODAMT;     IF FOODID=[illegible]91 THEN HAMBURG=FOODAMT;
IF FOODID=[illegible]12 THEN HOTDOGS=FOODAMT; IF FOODID=[illegible] THEN LUNCHMTS=FOODAMT;
IF FOODID=[illegible] THEN BEEF=FOODAMT;      IF FOODID=[illegible] THEN PORK=FOODAMT;
IF FOODID=[illegible] THEN BACON=FOODAMT;     IF FOODID=3111 THEN LIVER=FOODAMT;
IF FOODID=[illegible] THEN OTHERMTS=FOODAMT;  IF FOODID=[illegible] THEN FRYPOULT=FOODAMT;
IF FOODID=[illegible] THEN BAKPOULT=FOODAMT;  IF FOODID=1[illegible] THEN CANFISH=FOODAMT;
IF FOODID=[illegible] THEN FRYFISH=FOODAMT;   IF FOODID=[illegible]51 THEN COOKFISH=FOODAMT;
IF FOODID=[illegible] THEN RAWSHELL=FOODAMT;  IF FOODID=[illegible]17 THEN FRYSHELL=FOODAMT;
IF FOODID=[illegible] THEN CKSHELL=FOODAMT;   IF FOODID=[illegible] THEN CKCEREAL=FOODAMT;

IF FOODID=5190 THEN RTCEREAL=FOODAMT;    IF FOODID=[illegible]713 THEN WAFFLES=FOODAMT;
IF FOODID=[illegible]1 THEN BREADS=FOODAMT;   IF FOODID=51[illegible] THEN RICE=FOODAMT;
IF FOODID=[illegible]7 THEN PASTA=FOODAMT;    IF FOODID=[illegible]13 THEN FRYPOTA=FOODAMT;
IF FOODID=[illegible] THEN OTHPOTA=FOODAMT;   IF FOODID=[illegible] THEN YAMS=FOODAMT;
IF FOODID=[illegible]71 THEN CORN=FOODAMT;    IF FOODID=10[illegible] THEN BRUSSEL=FOODAMT;
IF FOODID=[illegible] THEN SQUASH=FOODAMT;    IF FOODID=14[illegible]1 THEN CAULIF=FOODAMT;
IF FOODID=[illegible]971 THEN BROCCOLI=FOODAMT;   IF FOODID=14[illegible] THEN CARROTS=FOODAMT;
IF FOODID=[illegible] THEN TOMATOES=FOODAMT;  IF FOODID=3507 THEN OLIVES=FOODAMT;
IF FOODID=[illegible] THEN LETTUCE=FOODAMT;   IF FOODID=[illegible] THEN SPINACH=FOODAMT;
IF FOODID=[illegible] THEN GRPEAS=FOODAMT;    IF FOODID=2977 THEN GRBEANS=FOODAMT;
IF FOODID=[illegible] THEN DRYBEANS=FOODAMT;  IF FOODID=[illegible]1 THEN SOYBEANS=FOODAMT;
IF FOODID=[illegible] THEN OTHERVEG=FOODAMT;  IF FOODID=[illegible] THEN CITRUS=FOODAMT;
IF FOODID=[illegible] THEN APPLES=FOODAMT;    IF FOODID=3919 THEN PEACHES=FOODAMT;
IF FOODID=16[illegible] THEN CHERRIES=FOODAMT;   IF FOODID=[illegible] THEN BANANAS=FOODAMT;
IF FOODID=[illegible]1 THEN MELONS=FOODAMT;   IF FOODID=[illegible]7 THEN RAISINS=FOODAMT;
IF FOODID=[illegible] THEN OTHERFRT=FOODAMT;  IF FOODID=[illegible] THEN PEANUTS=FOODAMT;
IF FOODID=[illegible] THEN JAMS=FOODAMT;      IF FOODID=[illegible]77 THEN SYRUP=FOODAMT;
IF FOODID=[illegible] THEN SUGAR=FOODAMT;     IF FOODID=[illegible] THEN PIZZA=FOODAMT;
IF FOODID=1[illegible] THEN BROTH=FOODAMT;    IF FOODID=[illegible] THEN OTHERSOP=FOODAMT;
IF FOODID=[illegible]79 THEN GRAVY=FOODAMT;   IF FOODID=[illegible] THEN SAUCE=FOODAMT;
IF FOODID=[illegible] THEN KETCHUP=FOODAMT;   IF FOODID=[illegible] THEN MAYONN=FOODAMT;
IF FOODID=3[illegible] THEN COSALAD=FOODAMT;  IF FOODID=51[illegible] THEN RESSALAD=FOODAMT;
IF FOODID=3512 THEN MUSTARD=FOODAMT;     IF FOODID=[illegible] THEN NUTS=FOODAMT;
IF FOODID=24[illegible] THEN CRACKERS=FOODAMT;   IF FOODID=21[illegible] THEN CHIPS=FOODAMT;
IF FOODID=[illegible] THEN OTHERSNK=FOODAMT;  IF FOODID=1[illegible] THEN CANDY=FOODAMT;
IF FOODID=1[illegible] THEN CAKE=FOODAMT;     IF FOODID=11[illegible] THEN ICING=FOODAMT;
IF FOODID=[illegible] THEN CHOCSYRP=FOODAMT;  IF FOODID=[illegible] THEN SHERBERT=FOODAMT;
IF FOODID=[illegible]1 THEN FRJUICE=FOODAMT;  IF FOODID=[illegible] THEN FRDRINKS=FOODAMT;
IF FOODID=[illegible] THEN COFTDR=FOODAMT;    IF FOODID=[illegible] THEN RSOFTDR=FOODAMT;
IF FOODID=[illegible]71 THEN BEVERAGE=FOODAMT;   IF FOODID=[illegible] THEN RCOFFEE=FOODAMT;
IF FOODID=[illegible] THEN ICOFFEE=FOODAMT;   IF FOODID=[illegible] THEN SCOFFEE=FOODAMT;
IF FOODID=5539 THEN NONDAIRY=FOODAMT;    IF FOODID=[illegible] THEN CREAM=FOODAMT;
IF FOODID=[illegible] THEN BEER=FOODAMT;      IF FOODID=[illegible] THEN WINE=FOODAMT;
IF FOODID=[illegible] THEN LIQUOR=FOODAMT;
IF LAST.ID THEN OUTPUT;
```

```
PROC SORT; BY ID;
DATA FIVE; MERGE ONE TWO FOUR; BY ID;
DATA TRIALS; SET FIVE;
DAIRY=SKIMMLK+LOFATMLK+WHOLEMLK+CCHEESE+RCHEESE+YOGHURT+PUDDING+
    ICECREAM+SOURCRM+BUTTER;
MEAT=HAMBURG+HOTDOGS+LUNCHMTS+BEEF+PORK+BACON+LIVER+OTHERMTS+
    FRYPOULT+BAKPOULT+CANFISH+FRYFISH+COOKFISH+RAWSHELL+FRYSHELL+
    CKSHELL;
STARCHES=CKCEREAL+RTCEREAL+WAFFLES+BREADS+RICE+PASTA+FRYPOTA+BOILPOTA;
VEGGIES=YAMS+CORN+[illegible]+CARROTS+TOMATOES+
    OLIVES+LETTUCE+SPINACH+GRPEAS+GRBEANS+DRYBEANS+SOYBEANS+OTHERVEG;
FRUIT=CITRUS+APPLES+PEACHES+CHERRIES+BANANAS+MELONS+RAISINS+OTHERFRT;
MISCELL=PEANUTS+JAMS+SYRUP+SUGAR+PIZZA+BROTH+OTHERSP+GRAVY+SAUCE+
    KETCHUP+MAYONN+[illegible]+MUSTARD;
SNACKS=NUTS+CRACKERS+CHIPS+OTHERCRUNCH+CANDY+CAKE+ICING+CHOCSYRP+SHERBERT;
DRINKS=FRUJUICE+FRDRINKS+LSOFTDR+RSOFTDR+BEVERAGE+RCOFFEE+ICOFFEE;
ALCOHOL=BEER+WINE+LIQUOR;
CHANGE=[illegible];
WTDIFF=WEIGHT-[illegible];
IF TRIAL=0 THEN DELETE;

IF NUMBER=2 THEN DELETE;
PROC SORT; BY OCANOW;
PROC FREQ; BY OCANOW; TABLES MODERATE--OTHDIET;
PROC ANOVA DATA=TRIALS;
CLASSES TRIAL OCANOW;
MODEL MODERATE--OTHDIET=OCANOW TRIAL;
MEANS OCANOW/LSD LINES;
/*
```

APPENDIX I

Self-reported changes in physical symptoms during oral contraceptive (OCA) use or a matched period for non-users

	comparison of OCA users and non-users*		correlation with hormonal potencies in oral contraceptives†	
physical symptom	non-users (no.=60)	OCA users (no.=30)	estrogen (no.=22)	progestational (no.=22)
nausea	3.1 ±0.6	3.2 ±0.6	-0.11	0.11
vomiting	2.9 ±0.6	2.9 ±0.6	-0.23	0.38
headaches	3.2 ±0.8	3.6 ±0.8‡	0.48#	0.08
breast tenderness	3.1 ±0.6	3.3 ±0.6‡	-0.13	0.30
chloasma	2.9 ±0.5	2.9 ±0.5	0.08	0.08
constipation	3.0 ±0.6	3.1 ±0.6	-0.21	-0.15
diarrhea	3.1 ±0.6	3.1 ±0.6	0.13	0.11
gas	3.3 ±0.6	3.2 ±0.6	0.31	-0.05
fatigue	3.6 ±0.8	3.5 ±0.8	-0.07	0.19
depression	3.2 ±0.9	3.2 ±0.9	-0.11	0.45#
acne	3.0 ±0.8	3.0 ±0.9	-0.06	0.17
menstrual bleeding	2.9 ±0.7	2.7 ±0.7	-0.20	0.33
spotting	3.0 ±0.6	3.1 ±0.6	0.31	0.08
amenorrhea	3.0 ±0.4	3.0 ±0.4	0.00	0.00
menstrual cramps	3.1 ±0.9	2.8 ±0.9	-0.29	0.31
bloating	3.2 ±0.7	3.2 ±0.7	-0.11	0.27
tension	3.7 ±1.0	3.8 ±1.0	-0.24	0.73#
high blood pressure	3.0 ±0.5	3.0 ±0.5	-0.14	-0.13

*Each value is mean ± $\sqrt{MSE}$. Values are the degree of change reported using a 5-point scale where 1=definitely less and 5=definitely more.
†Spearman's correlation coefficients for women on combined OCAs.
‡Significantly different from controls, $p<.05$ (F-test).
#Significant correlation, $p<.05$ (Spearman's test).

APPENDIX J

Patterns of daily food consumption in oral contraceptive (OCA) users and non-users

pattern	comparison of OCA users and non-users* non-users (no.=60)	OCA users (no.=30)	correlation with hormonal potencies in oral contraceptives† estrogen (no.=22)	progestational (no.=22)
time oral contraceptive was taken (%)				
morning (5am-11am)	--	33.3	--	--
mid-day (11am-4pm)	--	6.7	--	--
late afternoon (4pm-7pm)	--	13.3	--	--
evening (7pm-11pm)	--	30.0	--	--
night (11pm-5am)	--	16.7	--	--
distribution of food intake‡				
morning (5am-11am)	1.9 ±0.9	1.6 ±0.9	0.44#	-0.11
mid-day (11am-4pm)	2.9 ±0.7	2.9 ±0.7	0.41	-0.62#
late afternoon (4pm-7pm)	3.2 ±0.9	3.0 ±0.9	0.13	-0.07
evening (7pm-11pm)	2.1 ±1.1	2.0 ±1.1	-0.41	0.21
night (11pm-5am)	1.2 ±0.4	1.2 ±0.4	0.25	-0.14
frequency of meal eating¶				
breakfast	3.4 ±1.5	3.1 ±1.5	0.22	0.16
lunch	4.4 ±0.8	4.4 ±0.8	0.40	-0.19
dinner (supper)	4.7 ±0.7	4.6 ±0.7	0.34	-0.27

*Each value is mean ± $\sqrt{MSE}$.
† Spearman's correlation coefficients for women on combined OCAs.
‡ Scale where 1=small or none and 5=large.
#Significant correlation, $p<.05$ (Spearman's test).
¶ Scale where 1=rarely or less than once a week and 5=once a day.

APPENDIX K

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S. JOHN INGRAM, M.D., DIRECTOR
LOUIS HAGLER, M.D., ASSOC. DIR.
MEDICAL SERVICES DEPARTMENT

January 27, 1986

Ms. Ruth Litchfield
Department of Foods and Nutrition
Kansas State University
Justin Hall
Manhattan, KS 66506

Dear Ms. Litchfield:

Your request for information about the effects of oral contraceptives (OCs) on nutritional status (weight gain, body fat, and appetite) has been referred to me.

You have picked a difficult topic for your research project, because few clear-cut answers have emerged from research done to date; most of the studies enclosed were done in the early and later 70's and involved women taking OC's with higher doses of hormone than are currently prescribed. No attempt seems to have been made to group women by the OC each used. Many of the studies involved groups too small to study statistically. In the enclosed packet of published studies, the effects of oral contraceptives on nutrition show variations in results explained by differences in populations studied, in nutritional status of subjects, in hormonal contents of various "pills," or in the duration of contraceptive therapy.

However, let me tell you that in regard to vitamin supplementation, there is some indication that some women using oral contraceptives have lowered blood levels of two of the B vitamins, folic acid and riboflavin, and also of vitamin C. Although this lower vitamin level was observed in studies involving mainly women who were poorly nourished, it is nevertheless possible that the effect may have been real. The mechanism of interaction is poorly understood; it is not certain how constant blood levels of vitamins are in people <u>not</u> using OCs. Since not all OC users show a need for additional vitamin B complex, a decision would probably need to be made based on each patient's vitamin needs and level of nourishment.

I hope I have answered some of your questions and that the enclosed information is helpful. Since numerous references are offered on some of the articles enclosed, you may wish to request a library search from a local medical center, or a local medical library could provide you with a printout of additional references you need.

I wish for you good luck on your project, and please feel free to contact me if I can be of further assistance.

Sincerely yours,

S. John Ingram, M.D., Director
Medical Services Department

SJI/PM/vam/4994Z

Enclosures:

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ORAL CONTRACEPTIVES, WEIGHT CONTROL, AND FAT PATTERNING IN YOUNG COLLEGE WOMEN

by

RUTH EDSON LITCHFIELD

B.A., University of Northern Iowa, 1984

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Foods and Nutrition

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1986

Ninety white single college women (18-26 years of age) participated in a double-blind study designed to compare physical measurements and weight loss behaviors in oral contraceptive (OCA) users and non-users. Oral contraceptive users (n=30) and non-users (n=60) were matched on a 1:2 basis for weight and height. Data were obtained through an anthropometric measurement session and two-part self-report questionnaire. The OCA users had similar percent body fat, circumference measurements, and skinfold measurements when compared to non-users, but they had greater axilla skinfolds. The OCA users reported that they wanted to weigh less than the non-users; but they had similar weight control practices except for the greater use of over-the-counter diet pills by women taking OCAs. Effects of estrogen or progestational potencies were also determined in women using the combined OCAs (n=22). Estrogen potency was positively correlated with measured body weight, body mass index, estimated weight gain, arm and thigh circumference, and peripheral fat distribution. Data indicate that although there were few differences when comparing OCA users and non-users, weight gain was positively associated with estrogen potency in the combined preparations.