Order Number 8810560

The role of dietary fiber in the etiology of noninsulin-dependent diabetes mellitus

Marshall, Julie Ann, Ph.D.

University of Washington, 1987

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300 N. Zeeb Rd.
Ann Arbor, MI 48106

The Role of Dietary Fiber in the Etiology

of Noninsulin-Dependent Diabetes Mellitus

by

Julie Ann Marshall

A dissertation submitted in partial fulfillment
of the requirements for the degree of

Doctor of Philosophy

University of Washington

1987

Approved by Noel S. Weiss
(Chairperson of Supervisory Committee)

Program Authorized
to Offer Degree Epidemiology

Date December 30, 1987

Doctoral Dissertation

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Abstract

THE ROLE OF DIETARY FIBER IN THE ETIOLOGY OF NONINSULIN-DEPENDENT DIABETES MELLITUS (NIDDM)

by Julie Ann Marshall

Chairperson of the Supervisory Committee:
Professor Noel S. Weiss
Department of Epidemiology

Diet histories of cases of NIDDM and controls were compared to determine whether low dietary fiber intake was positively associated with the occurrence of NIDDM. The study group included prevalent, medically diagnosed diabetics between the ages of 20 and 74 years, residing in Alamosa and Conejos counties in Colorado and controls selected by geographically-based multi-stage sampling. Fiber intake prior to NIDDM diagnosis, or a comparable time in the past for controls, was obtained by food frequency questionnaire (FFQ). Current fiber intake was ascertained by FFQ and 24-hour diet recall. Three analyses were performed: 1) cross-sectional analysis of current fiber intake and plasma insulin concentrations among the controls; 2) comparison of prior fiber intake in previously known diabetics and nondiabetic controls; and 3) comparison of current fiber intake in previously undiagnosed diabetics and normal controls. Analyses were adjusted for age, sex, ethnicity, and body mass index. Other factors studied included disease duration, waist to hip ratio, parental history of diabetes, calories, carbohydrate, and season of interview.

Among controls, dietary fiber was inversely associated with fasting plasma insulin concentration adjusted for calories and carbohydrate. However, in a comparison of previously known diabetics and nondiabetic controls there was a tendency toward higher reported intakes of fiber prior to diagnosis among the diabetics. A decrease in fiber of 10 g/day was associated with a decrease in risk

of NIDDM of 0.80 (95% CI=0.65,0.98). When the diabetic group was limited to cases of less than five years duration, the association was no longer present. When previously undiagnosed diabetics were compared to normal controls, the odds ratio relating a 10 g/day decrease in fiber intake to NIDDM was 1.61 (95% CI=0.94,2.77) after adjusting for calories. The odds ratio relating fiber to NIDDM was reduced to 1.12 (95% CI=0.62,2.00) when carbohydrate was included in the model. This study has limitations including retrospective recall of diet, fiber measurement, and relatively small numbers of previously undiagnosed diabetics. Nonetheless, these findings do not support the hypothesis that increasing dietary fiber intake could reduce the future occurrence of NIDDM.

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ACKNOWLEDGEMENTS

The author thanks and sincerely appreciates Professors Noel Weiss and Richard Hamman for their guidance in this research and assistance in the preparation of this dissertation. Thanks to the Supervisory Committee and, especially, to Professor Barbara McKnight for her consultations. Special thanks to my husband Dick for continued statistical and moral support, to my son Earl who helped me maintain a healthy perspective, and to my parents Hal and Maxine who have always supported me in reaching new goals. Thanks also to all the staff of the San Luis Valley Diabetes Study who helped in collecting the data presented here and to Lorraine Beverly who facilitated the logistics of completing the degree at a distance. This work was supported by NIH grant DK30747.

CHAPTER 1

Introduction

The Occurrence of Noninsulin-Dependent Diabetes (NIDDM)

Diabetes mellitus is a metabolic disorder characterized by sustained high blood glucose levels (1). Four major types of diabetes are currently recognized: insulin dependent diabetes (IDDM), non-insulin dependent diabetes (NIDDM), diabetes secondary to other conditions, and gestational diabetes (2). NIDDM constituted 90 to 95 percent of the estimated 5.8 million prevalent cases of diagnosed diabetes in the United States in 1982 (3,4). In addition, as a consequence of the insidious onset of NIDDM, the number of people with undiagnosed NIDDM is estimated to be about equal to the number with diagnosed NIDDM as determined by glucose tolerance testing in the National Health and Nutrition Examination Survey (NHANES II) (4). For today's diabetic population, the cost of medical care and loss due to disability is estimated at $14 billion annually (5).

Disease prevalence is a function of detection, incidence and duration. Because of long average duration, the proportion of people with diabetes today has been influenced by changes in incidence and mortality patterns over the last 30 to 40 years. Longitudinal data collected on all diabetics (not subclassified by type) in Rochester, Minnesota from 1945 to 1970 determined that a newly diagnosed diabetic's 20 year survival was 69 percent relative to that of the general population. In this population, relative survival increased during this 25 year period (6). The age-adjusted incidence was 1.7 to 2.0 times as great in the 1960-69 period compared with 1945-59, in obese and nonobese persons, respectively.

Most of this apparent increase in incidence resulted from increased screening and detection (7).

NIDDM incidence increases with age and is similar for males and females (8). Incidence estimates per 1,000 person years, age-sex adjusted to the 1980 U.S. population, range from 0.65 in Rochester, Minnesota (1945-69) to 28.1 in the Pima Indians of Arizona (1965-75) (8). To prevent diabetes in future generations, knowledge of specific etiologic factors, epidemiologic features, and the natural history of the disease are needed (9).

Race-specific diabetes rates vary between migrants and their nonmigrating counterparts (10,11) and within population groups studied over time (12). Also, there is a gradient in diabetes occurrence between affluent and developing countries (10,13,14). These findings suggest a role for environmental or host acquired risk factors which may be preventable. The aim of this dissertation is to evaluate dietary fiber as a specific environmental risk factor for NIDDM.

Etiologic Risk Factors in NIDDM

Inquiry into the etiology of NIDDM has been hindered by a number of factors. The onset of NIDDM is not well represented by its date of diagnosis. In case-control studies and in cohort studies of short duration, risk variables measured at diagnosis may be misclassified due to physiologic changes that occur with onset of the disease process prior to diagnosis (e.g. body weight). Actual year of onset often precedes diagnosis by a decade or more which also allows factors related to the frequency of testing to affect the apparent incidence (10). In case-control studies, a family history of diabetes may be selectively biased by increased testing for diabetes among diabetic family members and not among family members of the controls. Another difficulty in evaluating

individual risk factors is that many of the proposed risk factors for NIDDM tend to be highly correlated (e.g. obesity, caloric intake, and physical activity) making it difficult to distinguish the effects of one potential risk factor from another.

In the last decade, heterogeneity in the diabetes syndrome has been recognized (15). The International Classification of Diseases did not separately classify IDDM and NIDDM until the ninth revision which was implemented in 1979. Advances in knowledge differentiating IDDM from NIDDM led the National Diabetes Data Group (2) to formally establish guidelines for the classification of IDDM and NIDDM which no longer used age of onset as a criterion. The age of onset of the two types overlaps and other characteristics provide better discrimination. IDDM is usually characterized by clinically abrupt onset of symptoms, insulinopenia, dependence on injected insulin to sustain life, and proneness to ketosis (2). NIDDM patients are not dependent on insulin to prevent ketonuria but may require insulin for correction of fasting hyperglycemia. They are not prone to ketosis and their insulin levels may be low, normal, or elevated. While insulin deficiency is generally accepted as the primary defect in IDDM, the pathophysiology of NIDDM may involve insulin deficiency, hepatic insulin resistance, peripheral insulin resistance, receptor defects or post-receptor defects (16).

In spite of guidelines for subclassification, difficulties remain in assigning a diagnosis (2). It may be impractical to take the thin NIDDM patient, who is on insulin, off insulin to determine insulin dependence. Similar features between IDDM and NIDDM at different stages in the natural history of each disease also makes classification of the disease difficult. Additional tests required to determine whether a patient has diabetes secondary to another condition may not have

been done. A non-diabetic may have transient elevated fasting plasma glucose levels due to stress, illness, trauma, pregnancy, or drug induced hyperglycemia. Other factors affecting blood glucose levels are physical inactivity, carbohydrate intake less than 150 grams per day over several days preceding the oral glucose tolerance test (OGTT), administration of the test in the afternoon, and lack of fasting or prolonged fasting. In the literature cited below, diabetes was only rarely subclassified by type (i.e. NIDDM). When age at diagnosis was available, only the data on older onset diabetics has been presented. In population surveys where type is not specified, 90 to 95 percent of diabetics are expected to have NIDDM (4).

Family History. In general, people with NIDDM have been found to have an increased family history of diabetes when compared to non-diabetics, though some authors have reported contradictory findings (10). Cross-sectional data of persons 35-74 years old examined in NHANES II reported parental diabetes in 17 percent of persons with normal glucose tolerance, in 28 percent of persons with abnormal glucose tolerance and no medical history of diabetes (predominantly NIDDM since diagnosis was after age 35), and in 35 percent of persons with a medical history of diabetes (not subclassified by type of diabetes) (8).

In a study of 1,031 incident cases of NIDDM in Rochester, Minnesota, the proportion with positive family histories for diabetes in parents or siblings was 39% (7). No controls were studied. However, these data are consistent with the NHANES II data cited above for people with a medical history of diabetes.

A cross-sectional screening survey for cardiorespiratory disease and diabetes conducted in 18,882 male Civil Servants in London, age 40 years and older (the Whitehall Study), included an oral glucose tolerance test (OGTT) and a "systematic" enquiry for family history (family not specified) of diagnosed and treated

diabetes (17). A family history of diabetes was 2 to 3.6 times more common in persons with a medical history of diabetes (type not specified). The family history prevalence ratios (PR) increased with age (40-49: PR=2.0, $p < 0.01$; 50-59: PR=3.1, $p < 0.01$; and 60+: PR=3.6, $p < 0.01$). When the prevalence of a family history of diabetes in previously undiagnosed diabetics was compared to normals, the data suggested an increased family history only in older onset cases (40-49: PR=1.0, NS; 50-59: PR=2.3, $p < 0.05$; 60+: PR=1.5, NS) (18).

Measuring the contribution of a positive family history to a person's risk for diabetes in a cross-sectional or case-control design is often complicated by 1) differential recall, since diabetics may be more aware of a family history of diabetes, and 2) testing frequency, relatives of diabetics may be more likely to be tested for the disease than relatives of non-diabetics. Prospective studies that involve periodic testing of all subjects can eliminate this selective misclassification of family history.

Keen et al (19), in a ten-year follow-up of 241 adults in Bedford, England, identified with impaired glucose tolerance, found an inconsistent relationship between family history and subsequent glucose tolerance results. Of 55 persons with persistent impaired glucose tolerance after 10 years, only 2% had a first degree relative with diabetes. In 128 reverting to normal, 12.5% had a positive family history, and of the 36 worsening to diabetes, 19% had a positive family history. Family history was not included in multivariate analyses relating worsening to diabetes and baseline variables.

Dunn et al (20) reported diabetes incidence by family history in white collar workers offered periodic health examinations in the eastern U.S. Thirteen thousand, one hundred and forty eight men age 30 to 59 years, who were examined two or more times between 1950 and 1964 in eight clinics, were included in this

analysis. The average length of followup was 3.8 years. In univariate analyses of family history (family not defined), the relative risk (RR) of diabetes increased with age (30-39: RR=1.9; 40-49: RR=2.1; 50-59: RR=2.8, no statistical tests were presented). The summary relative risk adjusted for baseline blood sugar, age, serum cholesterol, relative weight and systolic blood pressure was 1.8 ($p < 0.005$).

Paffenbarger and Wing (21) reviewed the reported family history of diabetes obtained from college entrance medical records among Harvard and University of Pennsylvania alumni. A positive family history was reported three times more frequently ($p < 0.02$) in students who subsequently developed diabetes after age 25 than in students who did not develop the disease. Even though data obtained may have underestimated family history of diabetes, there is no reason to expect that subsequent diabetics would have reported more accurately than non-diabetics.

In the Pima Indians (22), the risk of developing NIDDM in the offspring of two diabetic parents was 3.9 ($p = 0.0003$), and 2.3 ($p = 0.039$) when one parent was diabetic. These risk estimates are relative to offspring of non-diabetic parents and were adjusted for age and obesity.

Of the studies reviewed, only one with data on family history reported a positive association between diabetes and family history that was limited to middle aged, overweight men. This was a study of 2,927 subjects over age 20 seen between 1959 and 1960 in Tecumseh, Michigan, and located for followup of health status in 1977 to 1979 (23). A statistically significant excess risk (odds ratio = 1.63, $p < 0.02$) was observed in a crude analysis of family history (diabetes in a parent or sibling) and subsequent development of diabetes. Further analysis revealed that the highest excess risk associated with family history was in males 40-54 years of age with a subscapular skinfold thickness greater than 14 mm (odds ratio = 3.8, $p < 0.01$). The relative risks relating family history to

diabetes and adjusting for subscapular skinfold, in the other age-sex categories, ranged from 0.67 to 1.98 with p-values from 1.00 to 0.23, respectively. These last results were compatible with no true association.

Estimates of NIDDM risk associated with a positive family history of diabetes have been difficult to compare across studies due to varying definitions of "family". In the study by Paffenbarger and Wing (21), Harvard had collected data on family, in general, not otherwise defined. In contrast, the data from the University of Pennsylvania referred specifically to parents of the student. In both cases, the excess risk for a positive history was approximately three-fold, but "family" history (8.4%) was approximately three times as common as "parental" history (3.4%) in combined diabetic and non-diabetic groups. A more accurate method of assessing family history would be to standardize which individuals are to be included and then test those individuals for diabetes. The fact that some individuals may have many more first degree relatives, and consequently a higher probability of a positive family history, may be accounted for in the design by balancing family size between control and disease groups or in the analysis by adjustment. Even with these improvements in measuring family history, the relative contributions of environment (i.e. familiality) and genetics (i.e. heredity) remain undetermined when a positive association of diabetes and family history is observed.

Twin Studies. The twin method is predicated on the grounds that phenotypic differences in genetically identical monozygous (MZ) twins must be caused by environmental influences. High concordance, however, is not sufficient evidence to attribute genetic factors as the cause of a given condition since many aspects of the twin environment will also have been the same. Dizygous (DZ) twins, with only half of their genes in common by descent, act as controls - having been

born at the same time and exposed to similar environmental conditions. Similar levels of concordance in both MZ and DZ twins would suggest that genetic factors are not particularly important (24).

Harvald and Hauge (25) reported followup on 7,000 pairs of twins from the Danish Twin Registry born between 1870 and 1910. Three hundred and four diabetic probands were identified from detailed medical histories available on all 14,000 individuals. In 211 pairs, the co-twin was alive when the proband was diagnosed with diabetes. In 193 (85%) pairs, the proband was also an adult (not defined) at diagnosis. Concordance was 55 percent in 47 MZ adult pairs and 6.8 percent in 146 DZ adult pairs.

Gottlieb and Root (26) identified 242 diabetic twins from all Joslin Clinic records between August 1949 and May 1966. Both twins were alive in 105 of these pairs (a triplet set was analyzed as two twin sets - the diabetic proband was counted twice). Forty seven of these pairs were examined, 43 responded to a questionnaire, and 15 (17%) did not participate. In 38 pairs where the proband was diagnosed at 40 years or older, 70 percent were concordant among MZ twins and 3.5 percent were concordant among DZ twins.

Higher concordance of NIDDM in MZ twins has been reported by Barnett and coworkers (27,28). Of 53 identical twin pairs where the proband had NIDDM as determined by treatment history, 61 percent of the co-twins were known diabetics. Glucose tolerance testing on all twins increased the concordance to 90.5 percent. Metabolic studies on the five twins without diabetes and five age-sex-weight matched controls showed significantly higher fasting blood glucose in the twins (99.1 versus 68.5 mg/dl, $p < 0.05$) and higher mean 3-hour blood glucose following an OGTT (117.1 versus 88.3 mg/dl, $p < 0.05$). The serum insulin response to an OGTT was significantly lower in the twins (peak serum

insulin: 32 versus 97 mU/l, $p < 0.05$). The authors concluded that the non-diabetic members of the five discordant twin pairs showed metabolic abnormalities that would deteriorate further to frank diabetes and that concordance of NIDDM in MZ twins approaches 100 percent.

The discrepancy in degree of concordance for diabetes in MZ twins (55 to 70 percent (25,26) versus 90 to 100 percent (27,28) may be due in part to incomplete testing for diabetes in the studies where a lower concordance was observed. However, the degree of concordance in Barnett's study group was probably overestimated due to referral bias. Mann (29) estimated that the 53 twin pairs studied by Barnett and coworkers were about 3 percent of the total identical twin pairs with at least one twin having NIDDM in the United Kingdom. Of the 48 concordant pairs described by Barnett, 31 were referred after both twins had been diagnosed with NIDDM. Since patients were included because they were diabetic, concordant twins had a higher probability of being referred. The strength of the studies by Harvald (25) and Gottlieb (26) are that DZ twins were included with equal probability as the MZ twins and provided a comparison group where similarities of the twin environment could be controlled. These studies suggest that genetic factors play a role in the familial aggregation of NIDDM.

Studies in Migrants and Other Populations in Transition. Migrant studies often allow a unique opportunity to study the influence of differing environments on disease occurrence in the presence of genetic homogeneity. The observation of dramatically changing rates of NIDDM in populations migrating from eastern to western societies, from rural to urban areas, and in stationary populations undergoing relatively rapid westernization, has provided the opportunity for such contrasts.

The prevalence of diabetes (not subclassified by type) in Japanese migrants to Hawaii was compared to diabetes prevalence in the Hiroshima prefecture of Japan by survey in 1975 and 1976 (30). The ancestors of the Japanese-Hawaiians migrated primarily from Hiroshima and today environmental factors like diet and physical activity vary greatly between these two populations. The crude prevalence of diabetes was 12.3 percent in Hawaii and 6.9 percent in Hiroshima. The age-sex adjusted prevalence ratio (PR) was 1.79 ($p < 0.01$). Ecologic data on the same two groups indicated that the Hiroshima subjects were engaged in heavier work for both past and present activity and that their diet contained about half as much animal fat and simple carbohydrates, twice the quantity of complex carbohydrate and similar total energy consumption as Japanese in Hawaii. The excess prevalence remained after adjusting for obesity ($PR = 1.74$, $p < 0.05$).

Zimmet et al (31) contrasted rural and urban villages in Western Samoa. Prior studies in urbanized groups of Melanesia, Micronesia, and Polynesia provided evidence of NIDDM prevalence rates 3 to 10 times those of "typical" Caucasian populations, while diabetes remained almost unknown where a traditional lifestyle had been maintained. The prevalence of known and newly diagnosed NIDDM in the urban population of Western Samoa was found to be approximately three times (10.1% versus 3.6%, $p < 0.001$) that in the rural areas. Females had higher rates than males in the rural areas whereas males had higher rates in the urban areas. No significant differences in body mass index (BMI=weight/height^2) were observed between diabetics and non-diabetics in rural areas but urban diabetics had a significantly higher mean BMI than non-diabetics in both males (BMI: 30.5 versus 27.8 kg/m^2, $p < 0.001$) and females (BMI: 33.6 versus 30.6 kg/m^2, $p < 0.001$). However, obesity and age did not completely explain differences in prevalence.

West (32), in his own studies of 22 American Indian tribes of Oklahoma and in a compilation of prevalence data available on other American Indians, Eskimos, Pacific Islanders, and some Asian populations, concluded that diabetes was probably uncommon in all these native groups prior to 1940. Since 1940, marked differences in diabetes prevalence among native groups have been documented. In North America and the Pacific islands, West reviewed data on 40 aboriginal populations with high rates of diabetes and 25 with low rates relative to white populations of the United States and Canada. Of full blooded Oklahoma Indians receiving outpatient and inpatient care from the Indian Health Service, roughly 12 percent over the age of 34 years had diabetes in the early 1970's. The diabetes syndrome in American Indians is characterized by onset after age 20 years, absence of ketosis, marked hyperglycemia, obesity, and generous amounts of endogenous insulin, as in NIDDM. West (32) acknowledged the methodological problems in comparing prevalence data across groups where frequency of screening, diagnostic procedures, diagnostic criteria, and population characteristics like age may differ. However, he concluded that it would be difficult to account for the magnitude of the changes in diabetes prevalence before and after 1940 by these factors alone.

Studies in the Pima Indians present the most extensive longitudinal follow-up data in any of the high prevalence populations. In this population, the prevalence of diabetes (predominantly NIDDM) increased 42 percent from 1967 to 1977 (24.0 versus 34.1 per 100 persons, $p < 0.001$). Methods of examination and diagnostic criteria were the same in the two exams and prevalence was adjusted for changes in the age and sex distribution of the population (33). Useful data on obesity, family history, plasma glucose and insulin levels have also been obtained (34)

and are discussed elsewhere in this chapter. However, very little has been done to study the contribution of diet, physical activity and stress.

It is Neel's hypothesis (35) that NIDDM confers a significant biological handicap and that selective disadvantage would be expected to prevent this apparently genetic disease from attaining such a high frequency. The high prevalence in American Indians and similar populations leads to the proposal that the suddenness of their transition from a hunting-gathering early agricultural lifestyle, from overall restricted and irregular availability of food to essentially unrestricted food throughout the year, and from hard physical labor to a decreased need for physical activity, might be "telescoping" genetic adjustments which Caucasian ancestors spread over many generations. In times of feast and famine this diabetic genotype gave selective advantage (referred to as a "thrifty" genotype) and has only expressed itself in the form of disease in the presence of changing environmental factors.

Proposed contributing variables have changed simultaneously in contrasts of migrating groups, urban versus rural, or stationary groups over time. The change is often referred to as westernization or acculturation. Zimmet et al (36) suggest that the main contenders for a role in the high diabetes prevalence groups are obesity, reduced physical activity, dietary factors and stress. Further studies at the individual level are needed to determine the relative contribution of each of these factors.

Obesity. The opportunity to study the relationship between obesity and NIDDM has been facilitated by the relative ease of measuring height, weight and skinfolds, and by the routine collection of the former two variables upon medical examination. There is no doubt that obesity is related to diabetes but

the nature of the association - the magnitude, whether it is causal, and genetic dependence or independence - is less clear.

Cross-sectional or prevalence studies often report no association or even an inverse association between obesity and diabetes (37). These findings can probably be explained by changes in weight occurring due to the disease or to diagnosis. Melton and coworkers (7) in a review of NIDDM incidence in Rochester, Minnesota, over the period 1945-1969, found that weight loss prior to diagnosis was a frequent occurrence and often of sufficient magnitude to change a patients' apparent weight class. In a ten-year follow-up of "borderline" diabetics in Bedford, England, serial observations of the BMI indicated that those who worsened to diabetes showed a coincidental fall in BMI with the development of metabolic deterioration. Crude risk was doubled over the ten year followup (19.5 versus 9.3 percent) in men with a BMI greater than 25 and in women with a BMI greater than 27 at baseline. The excess was concentrated in the second five years of followup (9.8 versus 0.9 percent). Baseline BMI adjusted for baseline blood glucose and age was a significant predictor of worsening to diabetes (19).

Knowler et al (22) did a comparative analysis of incidence and prevalence data on obesity and diabetes in the Pima Indians. They found that there was little relationship between diabetes prevalence and concurrent obesity, but that obesity was strongly related to diabetes incidence with a positive dose-response over the entire range of BMI. The age-sex adjusted prevalence ratio for BMI greater than 39 compared to BMI less than 20 was 4.3. The age-sex adjusted incidence ratio was 90.3. Subjects were examined at intervals of approximately two years so BMI reflected obesity at some point within two years prior to diagnosis. The pattern of weight change for the period four years before and two years after diagnosis varied between young and old age groups. Mean

weights for subjects aged 25 to 44 years increased whereas the mean weight in subjects aged 45 years and over did not increase during the period prior to diagnosis. This suggests that weight change may play a different role depending on an individual's age.

Others have confirmed the relationship between obesity and diabetes prospectively. Paffenbarger and Wing (21) obtained a 71% response to a mailed questionnaire sent to 26,954 male alumni of Harvard and the University of Pennsylvania from classes of 1916 to 1950. Responses revealed 395 men who had developed diabetes after age 25. A historical review of college entrance medical records showed ponderal index (PI=height/weight$^{1/3}$) to be significantly related to future occurrence of diabetes (PI < 12.9/PI >= 12.9: RR=1.5, $p < 0.01$). Dunn et al (20) studied 13,148 white collar workers seen at least twice (average length of followup 3.8 years) for periodic health exams between 1950 and 1964. An excess relative weight of 25 percent was associated with subsequent development of diabetes. The summary relative risk adjusted for baseline blood sugar, family history of diabetes, age, serum cholesterol, and systolic blood pressure was 1.3 ($p < 0.075$).

In Tecumseh, Michigan, Butler et al (23) found a significant association between a relative weight index (RWI=weight/height compared to standard values of the Metropolitan Life Insurance Company tables) measured at screening of non-diabetics during 1959 to 1965 and subsequent occurrence of diabetes as reported by subjects reinterviewed during 1977 to 1979 (69% response). The mean age-adjusted RWI was significantly higher among diabetics compared to non-diabetics (males: 130.2 versus 115.9 RWI, $p < 0.01$; females: 144.2 versus 122.9, $p < 0.01$). Similarly, in a 14-year followup of 5082 participants age 33 to 67 years in the Framingham study, the unadjusted mean RWI was significantly

higher among those who subsequently developed diabetes (males: 126.6 versus 119.7 RWI, $p < 0.001$; females: 139.7 versus 119.5, $p < 0.001$) (38).

The Israeli Ischemic Heart Disease Study followed 9,462 male government employees age 40 years and over for five years (1963-1968). In this group, BMI was positively associated with incidence of diabetes (BMI 24.17-27.03/BMI < 24.17: RR=1.65; BMI > 27.03/BMI < 24.17: RR=2.52). In multiple logistic regression analysis, BMI was significantly associated with diabetes incidence after adjusting for age, peripheral vascular disease, cholesterol, systolic blood pressure, uric acid, hemoglobin, birthplace and education (39). The Israel Study of Glucose Intolerance, Obesity and Hypertension started in 1969 and sampled individuals from the Israel Central Population Registry who were born between 1912 and 1941. Data from 2,140 participants (94% response) who underwent a full OGTT in 1977 to 1982, and whose height and weight were recorded twice, at a mean interval of ten years, were included in analyses of BMI. In analyses of past and concurrent BMI and weight changes during the 10-year interval, past BMI was the main determinant of NIDDM incidence after adjusting for age, sex, and ethnic group (BMI >= 27/BMI < 27: RR=2.35, $p < 0.001$) (40).

In all of the studies cited above, obesity has been regarded as excess adipose tissue. The distribution of body fat has also been suggested as a predictor of NIDDM (41,42,43,44,45,46). A cross-sectional survey was conducted among members of TOPS, a weight reduction organization (42). Twenty-one thousand and sixty five women, between the ages of 40 and 59 in 1969, participated. In this group, the ratio of waist to hip circumference (WHR) was associated, independent of the RWI, to self reported diabetes (not otherwise defined). The summary odds ratio comparing WHR > 0.80 to WHR < 0.73, adjusted for age and RWI, was 3.1 ($p < 0.001$). These findings are consistent with evidence that the

size of abdominal, but not gluteal or femoral, fat cells is associated with metabolic variables like plasma insulin (46).

The evidence reviewed suggests that obesity is a risk factor in the subsequent development of NIDDM. Studies of the metabolic and cellular pathogenesis of NIDDM provide support for the biological plausibility of a causal relationship between obesity and NIDDM. Obesity is known to be associated with elevated fasting plasma insulin levels, increased plasma insulin response to insulinogenic stimuli and insulin resistance (16). Further evidence of the causal relationship between obesity and diabetes and implications for prevention are provided by the fact that hyperglycemia and many other metabolic abnormalities are at least partially reversible with caloric restriction and weight reduction (22).

Physical Inactivity. Physical inactivity has been proposed as a risk factor for NIDDM, first, because of its role in energy balance and obesity. And second, because glucose tolerance has been observed to improve after physical training.

Krotkiewski et al (47) studied 28 obese subjects with NIDDM before and after 3 months of physical training. C-peptide values during an OGTT were higher after training, insulin values remained the same, and glucose tolerance was improved (sum of 4 glucose values during 2-hour OGTT: 1,131 versus 1,070 mg/dl, $p < 0.05$). Non-diabetic obese controls (n=13) had decreased C-peptide values, similar insulin levels and a nonsignificant decrease in glucose tolerance after training (sum of 4 glucose values during 2-hour OGTT: 578 versus 568 mg/dl, NS). Further studies provided evidence that insulin resistance, typical in NIDDM, is lowered by physical training and, consequently, insulin secretion decreases (47).

However, epidemiologic data supporting physical inactivity as a risk factor for NIDDM are limited (10). Physical inactivity and obesity were assessed in

cross-sectional diabetes surveys in the Micronesian population of Nauru, in Fiji Melanesians, and in Asian Indians in Fiji (48). Activity was assessed by interviewers who were "instructed to take both occupational and leisure activity into account." In sex-race-specific analyses, the relative risk estimates for physical inactivity, adjusted for age, BMI, and urbanization, ranged from 1.2 (NS) to 2.9 (95% CL: 1.1-7.7). The authors note that if diabetes were to reduce habitual physical activity, then a spurious association between physical inactivity and diabetes could occur in a study of prevalent cases.

Two prospective studies reported no association between diabetes and physical inactivity. Paffenbarger and Wing (21) in their study of college alumni did not find an association between participation in varsity athletics or "weekly recreation" and subsequent development of diabetes. Medallie et al (39,49) reported that physical activity status in 1965 was not associated with development of diabetes in the following five years in Israeli male civil servants.

Stress. The evidence implicating stressful occurrences as the precipitating factor in the onset of diabetes have for the most part relied on clinical case studies or associations between diabetes and psychiatric illness (50). Lack of a generally recognized definition of the term stress has made evaluations of the significance of psychologic or mental stress difficult (10). The stress inherent in the adjustment to the diagnosis of diabetes suggests that the temporal occurrence of stress with respect to disease onset must be carefully considered in study design. Of the prospective studies reviewed, only the Israeli Ischaemic Heart Disease study (51) published findings related to psychosocial factors. The answers to three questions (Do you consider yourself a tense person? Do you generally suffer from anxiety? Do you generally suffer from sleep problems?) were combined into an anxiety index. It was found that those reporting the

fewest problems had the highest incidence of diabetes. This same index was positively associated with myocardial infarction and angina pectoris. After controlling for age, weight/height, peripheral vascular disease, cholesterol, education, and birthplace, anxiety was no longer associated with an increased risk of diabetes. Later analyses, after 5 years of followup, revealed negative associations between diabetes incidence and psychosocial variables including hurt by a coworker; present family trouble; hurt by family; and efforts, disappointments and worry about standard of living (39). A combined 5-point psychosocial score based on these same questions did not show significant differences in incidence between the highest and lowest scores (49).

The physiologic response to biologic stress (e.g. trauma, sepsis, burns, hypoxia, cardiovascular disease, hypothermia and cold stress) has been systematically studied. A review by Porte and Woods (52), discussed in detail the neuroendocrine response, particularly, neural regulation of the pancreatic islet in the regulation of plasma glucose, to these types of stressors. There are differences in metabolism depending on the cause of stress hyperglycemia, which highlights the need for refined definition of stress in future population studies.

Diet. Two micronutrients, vitamin D and chromium, have been studied as factors in glucose tolerance. Gedik and Akalin (53) suggested that vitamin D deficiency could alter pancreatic beta cell function and calcium transport and, thereby, insulin release. They studied the insulin response to an OGTT in four vitamin D deficient subjects before and after six months of vitamin D supplementation. The mean serum vitamin D ($1,25\text{-}(OH)_2D$) was increased from 29.8 to 70.3 pg/ml ($p < 0.05$) with treatment. The insulin area increased from 9.09 to 13.6 mU/min ($p < 0.05$) with treatment and the insulin area in 10 normal subjects was 11.9 mU/min. The dietary protocol before and during supplementation

was not stated, however, the BMI of the four subjects was the same before and after supplementation. To the extent that other factors affecting glucose tolerance did not change coincidentally with vitamin D supplementation, these data suggest that insulin release following a glucose challenge is impaired in patients with vitamin D deficiency and that it is restored to normal after vitamin D treatment.

In animals, impaired glucose tolerance is the earliest detectable and most prominent feature of chromium deficiency. Nanogram quantities of chromium have been shown to act as a cofactor for the optimal effect of insulin on insulin-dependent systems of the body (54). In 1977 (55) a patient receiving long-term total parenteral nutrition developed diabetic symptoms including glucose intolerance, impaired nerve conduction and weight loss. Following two weeks of chromium supplementation, all diabetic-like abnormalities returned to normal. A comparison of 80 insulin-dependent and non-insulin-dependent diabetics with 47 healthy volunteers demonstrated low plasma chromium in diabetics (mean plasma chromium: 0.56 versus 0.82, $p < 0.0001$) (56). In a randomized chromium supplementation trial of eight patients with mild NIDDM and 16 normal volunteers, the glucose response to an OGTT was significantly lowered in the chromium-rich yeast supplementation group and not in the chromium-poor yeast supplementation group (delta glucose area: 83 versus 13 mg/dl/hr, $p < 0.01$). A nonsignificantly lower insulin response was observed (delta insulin area: 47 versus 25 uU/dl/hr, NS) (57). These micronutrients have not been studied prospectively to determine if the risk for developing NIDDM is higher in people deficient in either vitamin D or chromium.

The relative roles of carbohydrate and fat and the importance of calories in the etiology and treatment of diabetes have been controversial for centuries (58). In the decade following the discovery of insulin, several

investigators observed that progressive improvement in blood glucose curves could result from periodic doses of glucose. The most extensive studies to determine the dietary factor responsible for improved glucose tolerance were carried out by Himsworth (59,60,61). He observed the glucose response in healthy young men while systematically varying dietary fat, protein, carbohydrate, and calories. Each particular diet was given for at least one week before an OGTT was performed. In the first series of diets protein and calories were held constant. The area under the glucose tolerance curves progressively decreased as the carbohydrate to fat ratio increased (Table 1.1a). It was concluded that protein and calories did not cause the variation in glucose tolerance. Next Himsworth increased the fat calories and carbohydrate calories while keeping the ratio of fat to carbohydrate calories and grams of protein constant. Glucose tolerance improved as carbohydrate and fat calories increased (Table 1.1b). In the third series of diets, carbohydrate and protein were held constant while varying the fat and calories. The glucose tolerance area did not change as dietary fat and calories were increased. Himsworth concluded that glucose tolerance was determined by the carbohydrate content of the diet and that glucose tolerance improved as dietary carbohydrate was increased.

The improvement of glucose tolerance with high carbohydrate formula diets has been demonstrated in normals and mild diabetics. In a metabolic ward study by Brunzell et al (62), nine healthy subjects and 13 subjects with mild diabetes (untreated patients with NIDDM) were fed low and high carbohydrate formula diets for eight to 10 days each and then fasting (N=23) and OGTT (N=9) blood glucose and insulin concentrations were determined. The low carbohydrate diet contained 45 percent carbohydrate, 15 percent protein, and 40 percent fat and the high carbohydrate diet contained 85 percent carbohydrate, 15 percent

Table 1.1. Mean area of glucose tolerance on diets varying in carbohydrate and fat content.

	Number of Subjects	Carbohydrate (grams)	Fat (grams)	Mean Area[1] (mgs/min)
a.	3	50	240	9,358
		200	173	6,013
		350	107	5,202
		425	73	3,598
b.	1	100	87	7,050
		150	132	5,505
		200	173	4,470
		300	260	3,460
	1	313	92	4,430
		437	128	4,170
		555	163	2,595
c.	1	250	100	3,500
		250	200	3,527
		250	300	3,534

[1]Mean area included blood glucose readings until glucose readings returned to baseline. In a. all subject's curves were truncated when the first subject returned to baseline.

protein, and zero percent fat. Fasting plasma glucose and serum insulin concentrations were both significantly decreased on the high carbohydrate diet (fasting glucose: 97.2 versus 89.3 mg/dl, $p < 0.001$; fasting insulin: 19.3 versus 16.2 uU/ml, $p < 0.02$). Similar differences were observed in normals and diabetics. The OGTT glucose area up to 120 minutes was decreased 8.1 percent ($p < 0.05$) on the high carbohydrate diet. A nonsignificant reduction of 22 percent was observed in the OGTT insulin area. These data support the hypothesis that high carbohydrate feeding improves glucose tolerance.

The composition of carbohydrate was not stated in the studies by Himsworth. And Brunzell et al administered formula diets of dextrose and dextrins plus maltose. Simpson et al (63) investigated the effect of digestible carbohydrate

on glucose tolerance, in nonformula diets and independent of the amount of dietary fiber. Ten subjects with NIDDM were administered two isocaloric diets for four weeks each in a randomized crossover design. The low carbohydrate diet contained 35 percent of calories as carbohydrate and 14.3 grams per day of dietary fiber. The high carbohydrate diet contained 60 percent of calories as carbohydrate and 16.8 grams per day of fiber. In 24-hour profiles, basal plasma glucose was the only measure that was significantly lower on the high carbohydrate diet (mean of the 0300, 0500, and 0700 samples: 106.3 versus 95.5 mg/dl, $p < 0.05$). Postprandial blood glucose measures were all significantly higher on the high carbohydrate diet. And glycohemoglobin and mean 24-hour plasma glucose were the same on both diets. These results suggest that digestible carbohydrate lowers basal blood glucose but not glucose levels following a meal or blood glucose averaged over a longer period of time.

Physiologically, dietary intake of carbohydrate is important in the normal insulin response to a meal. Interactions between the gastrointestinal tract and endocrine pancreas are known to be mediated by hormones and neurotransmitters. GIP (Gastric Inhibitory Polypeptide or in the context of its second function described here, Glucose-dependent Insulinotropic Polypeptide) is the humoral secretion of the intestine primarily responsible for potentiation of insulin release (64). Of the macronutrients, carbohydrate is the strongest stimulant of GIP secretion (65).

Traditionally, carbohydrate has been divided into "complex" and "simple" carbohydrates. Simple carbohydrates have been assumed to be readily absorbed from the gastrointestinal tract, causing a more pronounced rise in blood glucose than complex carbohydrates. Recent evidence (16,66,67) suggests that there is significant overlap in the glycemic response to simple and complex carbohydrate

food. Fructose gives a lower response than glucose, sucrose and some starches (67). Potatoes create a response more like glucose, whereas rice and beans are similar to fructose. There is variation by the method of preparation and by the composition of the meal. These findings suggest that the physiologic response to a food may be a more meaningful measure of risk than the amount or type of carbohydrate in that food item. Jenkins et al (66) have developed such a measure, called the glycemic index, which compares the area under the blood glucose response curve for each food as a percentage of the area after taking the same amount of carbohydrate from a reference food like white bread.

Some of the complex issues which can arise in studies of diet and disease are highlighted in the following section which discusses the nature and measurement of dietary fiber. Population studies of diet and diabetes are included in the final section of this chapter where the evidence for and against low dietary fiber as an etiologic risk factor in NIDDM is reviewed.

Nature and Measurement of Dietary Fiber

Dietary fiber is defined as those plant materials which are not digested by endogenous secretions of the human gastrointestinal tract (68,69,70,71). It is a complex and variable mixture of substances (72) which include materials of diverse chemical and morphological structure that are ubiquitous in plant foods (69). Botanical, chemical and physiologic research have all contributed to the evolution of the current concept of dietary fiber. Dietary fiber is generally accepted to be of plant origin and derived specifically from plant cell walls, intracellular storage polysaccharides, and secretions of specialized secretory cells (69,70,71,72).

Plant cell structure plays an important role in understanding the varied physiologic responses to fiber and in appreciating associated methodological problems in its analytic definition. Figure 1.1 summarizes the botanical and biochemical components of dietary fiber. The cell wall is composed of microfibrils of cellulose embedded in a matrix of noncellulosic polysaccharides (NCP), protein, lipid and inorganic constituents like calcium and silica (72). The primary cell wall is rich in pectin. The secondary cell wall forms later than the primary cell wall and is rich in cellulose. Lignin is formed as the matrix matures, imparting extra rigidity to the plant structure. Aging of the plant cell is thus associated with increasing proportions of cellulose and lignin (69). NCP not found in the cell wall (i.e. nonstructural polysaccharides) include gums which are secreted by specialized plant cells in response to trauma (69), mucilages which retain water and so protect seeds from desiccation (73), storage polysaccharides which are found in large amounts in leguminous seeds (74), and food additives including plant derived gums, algal polysaccharides, and synthetic modified cellulose (70,72).

The primary chemical structures in dietary fiber are cellulose, NCP, and nonpolysaccharides. Cellulose is made up of long chains of glucose molecules. Strong hydrogen bonds connect adjacent chains and result in the characteristic microfibrils or "fiber" (70,72). Cellulose makes up 10-25% of the plant cell wall (70). The remaining NCP and nonpolysaccharide components of dietary fiber are not fibrous (72). NCP make up 55-85% of the polysaccharides in dietary fiber (72). The NCP matrix of the cell wall has been subclassified in two ways. First, cell wall NCP has been divided into the pectic fraction (rich in uronic acid) and hemicellulose (poor in uronic acid) based on sequential extraction methods (72). However, a more physiologically useful nomenclature subclassifies

Structural plant cell wall
- polysaccharides
  - **cellulose**
  - noncellulose (NCP)
    - **insoluble**
    - **soluble**
- nonpolysaccharides
  - **lignin**[1]
  - **protein**[2]
  - **lipid**[2]
  - **organic constituents**[2]

Storage and secretory plant components
- polysaccharides
  - noncellulose
    - soluble - **gum, mucilage**

Food additives
- polysaccharides
  - **modified cellulose**
  - noncellulose
    - insoluble - **algal**
    - soluble - **gums, algal**

[1]included in some analytic definitions.
[2]not included in analytic definitions.

Figure 1.1. **Components** of dietary fiber

fiber fractions according to their solubility in water (70). The nonstructural storage and secretion polysaccharides, as well as the pectins, and part of the hemicelluloses of the cell wall, are included in the water soluble component (70,72). The nonpolysaccharides are a minor proportion of the cell wall. Only lignin has been included in analytic definitions of dietary fiber. However, the nonpolysaccharide components may modify the chemical properties of the polysaccharide component (72) and factors associated with fiber, like enzyme inhibitors which are found in high concentrations in many plant seeds, may influence digestibility of unprocessed foods (75).

Cellulose and NCP are beta-linked polymers which resist digestion by the alpha-glucosidase enzymes of the human digestive tract (72). Consequently, cell wall components survive transit through the stomach and small intestine in the form of a lattice work which acts to package and surround nutrients (71). Cellulose microfibrils and the crosslinked crystalline structures of insoluble NCP have important physical characteristics including water holding capacity, cation exchange, adsorption, and rigidity (76). These properties may be modified by particle size, pH, osmolality, and bacterial degradation (76). Soluble NCP have colligative properties including osmotic pressure, viscosity and movement (76). The solubility depends on pH since more NCPs are extracted at a higher pH (72). As fiber moves through the gastrointestinal tract there is alteration in the matrix structure and function as the surrounding pH and osmolality change (69).

Because of these physicochemical properties, dietary fiber appears to influence each stage of gastrointestinal function. Fiber gives food structure and acts as a solidifier and water trapper. In the mouth, it cleans the teeth and stimulates chewing and saliva secretion (70). The integrity of the cell wall structure and the need to chew have been associated with satiety (77). Foods are eaten more slowly and gastric emptying is slower when fiber is included in the meal. This slowing effects the subsequent rate of absorption of nutrients in the small intestine (78). In addition, because of the colligative properties of soluble fiber and the surface properties of insoluble fiber, it has been suggested that the gel fiber matrix moves through the intestine acting like a molecular sieve or ion exchange chromatography system which interferes with micelle formation and separates nutrients from digestive enzymes and absorptive surfaces (78).

The properties of the gel fiber matrix are determined by the fiber composition, the meal taken with it (both content and preparation), digestive

enzymes of the small intestine, and bacterial fermentation in the colon. The matrix behaves differently in the small and large intestine. Dietary fiber slows transit of a meal through the small intestine and speeds transit through the large intestine. In the large intestine it acts as a substrate for bacterial fermentation, and softens and enlarges the stool.

As understanding of the structure and function of dietary fiber has evolved, so has the terminology. It is now recognized that dietary fiber is not an inert filler in the large bowel (i.e. bulk), nor is it abrasive (i.e. roughage), and it is not the total fecal content (i.e. residue) (79). Bacterial fermentation in the colon renders some of the fiber available for absorption and consequently the term "unavailable carbohydrate" is technically incorrect (74). A commonly available food composition measure is crude fiber, which was measured in animal feeds as early as 1820. Crude fiber was simply defined as plant food resistant to hydolysis by acid and subsequently by alkali (74). However, unlike human digestive processes, the analysis of crude fiber eliminated 20 to 50% of cellulose, 80% of hemicellulose and 50 to 90% of lignin (73). Because of the variable mix of these components across foods, crude fiber not only underestimates but may also incorrectly rank foods with respect to dietary fiber content. Kay (69) states that traditional crude fiber analysis bears no consistent or quantitative relationship to dietary fiber.

Development of a standard procedure for analyzing the dietary fiber content of foods has been difficult to establish. Southgate (72) reviewed 14 methods, 6 published since 1980. Ten different combinations of dietary fiber fractions were measured by these 14 methods. Five of the 14 methods provided only a composite measure. Because fiber has a variable composition and because physical and physiologic properties of dietary fiber are related to specific fiber fractions,

data on the composition of dietary fiber is highly desirable. Food composition tables for estimating dietary fiber intakes should specify the analytic method used in order to determine the dietary fiber fractions being measured (72). Southgate and Englyst (72) recommend the method of Englyst which combines gas-liquid chromatography and colorimetric techniques to measure cellulose, soluble NCP, insoluble NCP, and resistant starch individually. However, a limited number of foods have been analyzed using this method. The most uniform source of data on an extended list of foods comes from McCance and Widdowson's The Composition of Foods (68) where dietary fiber is defined as "the sum of the polysaccharides and lignin which are not digested by endogenous secretions of the human gastrointestinal tract; this fraction has a variable composition and is made up of different types of polysaccharides (cellulose, hemicellulose, and pectic substances and the noncarbohydrate lignins)."

Ambiguities remain in the definition of dietary fiber. Dietary fiber is not digestible by the human gastrointestinal enzymes but as little as one-fifth of ingested fiber survives passage through the colon intact. In the colon, it acts as the major substrate for bacterial fermentation and some of the end-products are absorbed (70). On the other hand, there are plant products not included in the definition of dietary fiber which are also not digested. For example, the structure of high amylose starch can be affected during cooking so that it becomes resistant to digestive enzymes (72). The amount of resistant starch in a food is highly variable depending on processing, cooking, and storage time (72). Heating, like toasting bread, can result in a non-enzymic browning, the Maillard reaction, which alters the food surface and leaves it indigestible (69).

A number of factors contribute to the variability of the fiber composition of foods. As mentioned above, fiber varies according to the age of the plant.

However, since lignin gives plants a woody texture, there is selection for young plant foods and lignin usually contributes only a small amount to total dietary fiber. Species, anatomical source (e.g. seed, stem, fruit) (69), season of harvest for annual plants, climatic conditions (80), and biologic variability (72) also contribute to variability in amount and composition of fiber. Methods of preparation which involve physical disruption, heat, hydration, storage over time, or additives will effect the physical and chemical properties of fiber. As noted, the method of fiber analysis will also alter the relative importance of specific foods as dietary fiber sources because of the variable mix of fiber components across foods combined with variations in the fractions measured by different analytic methods.

Determination of dietary fiber intake in human populations combines problems in estimating the fiber content of foods with error in assessing the amount of foods people eat. However, with this in mind, Bingham (81,82) has compiled a summary of the available dietary fiber consumption data for Europe, Africa, Australasia, Japan, and North America. The most extensive analyses come from Great Britain where different methodologies (e.g. population wide food consumption, individual dietary questionnaire, and one week weighed samples for both individuals and families) all suggest dietary fiber consumptions around 20 grams per day (range 19.7-22.7).

The European data presented by Bingham (82) suggest variation in fiber intake within individuals from day to day, by geographic location, by season, and according to vegetarian status. In a study of four Scandinavian cities plus Cambridge, England, the within person (day-to-day) variability was similar to the between person variability. The ratio of between to within person variance ranged from 0.5 in Cambridge to 2.0 in Them, Sweden. Seasonal variation was

dependent on the area studied. Eleven regions of the European Economic Community in Holland, Belgium, Germany, France, and Italy showed greater variation by season than by area. In Great Britain, there was significant seasonal variation in total dietary fiber intake and the source of fiber by season differed. Vegetables, like root vegetables and brussel sprouts, contributed to higher intakes in the winter, whereas less dense summer salad vegetables contributed to lower intakes in the summer.

Vegetarians in England reported fiber intakes of 33.0 to 41.5 grams per day in two studies. This was 1.5 to 1.9 times the amount reported for nonvegetarians similarly studied. Bingham also reported differences by income, with higher income groups having lower fiber intakes. Differences in fiber intake by age and sex are largely due to differences in energy intake. On the whole, dietary fiber intake has remained fairly constant in Great Britain over the last 80 years except for a marked increase during World War II.

Bingham (82) presented preliminary information suggesting a large variation in dietary fiber intake worldwide, both in total quantity and in distribution across cereal, vegetable and fruit sources. Intakes as high as 130 to 150 g/day were estimated in the largely vegetarian Kikuyu of Kenya and the plantain eating villages of central Uganda, which contrasted with intakes around 20 g/day in developed countries. Cereal was estimated to contribute 0% to total dietary fiber in Uganda, 61% among the Kikuyu, and 30% in the United States and Great Britain.

Low Dietary Fiber as an Etiologic Risk Factor in NIDDM

Low dietary fiber has been suggested as a risk factor in the onset of diabetes in animals and in humans. In humans, ecologic and clinical studies

have been the primary method of investigating this hypothesis. A series of studies by Hackel, Schmidt-Nielsen and coworkers (83,84,85) investigated the observation that laboratory colonies of the sand rat developed diabetes mellitus when fed on standard laboratory diets. The authors do not discuss the type of diabetes, but the associated symptoms (e.g. obesity and continued secretion of insulin) suggest the disease in these animals was like NIDDM. Initial studies fed one group of ten rats a fresh vegetable diet of beet roots, beet greens, spinach, and carrots and a second group of 12 rats laboratory chow plus fresh vegetables ad libitum for six months. The rats fed laboratory chow showed significantly higher weight (256 versus 134 grams, $p < 0.001$), plasma glucose concentrations (325 versus 125 mg/dl, no statistical test presented), and urine glucose concentrations (4,560 versus 7.3 mg/dl, no statistical test presented) (83,84). A third study (85) showed no difference in the insulin and glucose response when total calories were limited to 30 calories per day on the these two diets. This suggested that total calories and increased weight were responsible for the previously observed induced abnormal glucose tolerance or that weight loss accounted for the normal glucose tolerance in the latter study. It would be necessary to test these diets under weight maintaining conditions to isolate effects of diet composition on metabolic parameters. The fiber content of the two experimental diets was not reported, but it is presumed that the fresh vegetable diet was higher in dietary fiber. The percent of calories from fat and carbohydrate may also have varied. In conclusion, the data presented on sand rats suggest that diabetes can be induced by diet but it remains unclear whether this can be accounted for by energy imbalance or some qualitative aspect of the diet. More recent studies in the sand rat have not resolved these issues (86).

Ecologic studies of human fiber consumption and diabetes rates or related metabolic parameters have been looked at in cross-sectional contrasts and migrant studies. A number of investigators have reported a gradient in diabetes prevalence over time or across populations in Africa (87,88). Differences in diet, in particular, the amount of carbohydrate and degree of refining, have been observed to be negatively correlated with this difference in prevalence between groups (88,89,90). Prevalence in these reports was usually determined by hospital or clinic patients, so differences in prevalence may relate to the size or makeup of the population being served by these hospitals and differences in medical practice.

Further studies in Africa have documented blood glucose and insulin parameters across groups with differing carbohydrate and fiber intake. Rubenstein et al (91) studied metabolic response to an oral glucose load in seven African and eight white healthy males in South Africa. Fasting and post-load blood glucose values did not differ significantly between the two groups. Fasting insulin values were similar but insulin levels in response to glucose were significantly lower in Africans (mean 30-minute insulin concentration: 115 versus 61 uU/ml, $p < 0.02$; mean area under the insulin curve: 11,100 versus 7,980 uU/min, $p < 0.02$). In contrast to whites in Johannesburg, "Africans have a much lower standard of living, they are physically active, and their diet is low in animal protein and fat and high in unrefined carbohydrates such as maize, bread, and sorghum."

Walker et al (92) reported lower fasting and 1-hour post glucose load values for Bantu (n=80) compared to Caucasian (n=not stated) school children in Johannesburg (mean fasting blood glucose: 88 versus 43 mg/dl, no statistical test reported; mean 1-hour blood glucose: 115 versus 54 mg/dl, no statistical test

reported). The Bantus ate much lightly refined corn meal and the Caucasians ate western diets (90).

Wapnick et al (93) found significantly lower fasting blood glucose (79 versus 68 mg/dl, $p < 0.05$), 60-minute serum glucose (38 versus 9 mg/dl, $p < 0.05$) and 60-minute serum insulin (30 versus 15 uU/ml, $p < 0.02$) following an oral glucose tolerance test (OGTT) when 50 African hospital cleaners eating a high proportion of maize meal were compared to 12 Europeans. No difference was seen in fasting serum insulin. A group of 35 African students on a "western" diet, high in sucrose and white bread starch, had intermediate values on all parameters.

In a study of the impact of urbanization on the insulin response to a starch tolerance test (STT), O'Dea et al studied full-blood Australian Aborigines (94). Subjects spent three months living in their "traditional" hunter-gatherer life-style after which a STT was conducted. These findings were compared with a STT conducted three months after returning to their urban environment. Basal insulin levels were similar but areas under the insulin curve in the first hour were 4478 uU/ml/min after urban living and 2959 uU/ml/min after traditional living ($p < 0.001$). In the traditional life-style, no refined food or alcohol was consumed and the level of physical activity was generally high. In the urban environment, subjects ate a high level of refined carbohydrate, low protein, and there was generally heavy alcohol consumption and reduced physical activity.

Migrant studies in Japanese indicate 1.74 times the prevalence of diabetes in migrants to Hawaii compared to their counterparts in Hiroshima Prefecture in Japan, after adjusting for age, sex, and obesity (30). Migrants on average reported similar total energy consumption but more than twice as much animal fat and simple carbohydrate and less than half the quantity of complex

carbohydrates compared to Japanese in Hiroshima. Since fiber is of vegetable origin and depleted with processing, inference of decreasing fiber intake was made from shifts from vegetable to animal sources of dietary fat and from complex to simple carbohydrates.

In general, group level or ecologic comparisons may be useful in generating hypotheses but their role in supporting hypothesized relationships must be viewed with caution. Many social, economic, and dietary variables tend to be more highly correlated on a group basis (95), especially when the groups differ by something as pervasive as technology development (i.e. comparisons over a time of technology development within a population or across populations at different stages of development). Non-dietary risk factors for diabetes, like age, obesity, physical inactivity, and stress are likely to be correlated with diabetes rates and, if accounted for, may explain observed associations between fiber and diabetes. In establishing diabetes rates, the screening procedures, glucose load, time of day examined, hours since last feeding and the chemical method of blood analysis may differentially influence the prevalence data collected in these group comparisons (32). Finally, in ecologic studies, it is not known whether the people with diabetes are also the people with low fiber intakes. However, in spite of the limitations of ecologic or group analyses, the data available are at least consistent with the hypothesis that a low fiber diet increases an individual's risk for developing NIDDM.

Clinical investigations have studied the effects of dietary fiber on metabolic responses in normal and diabetic subjects. A large literature has accumulated on this topic since the mid-1970s and a number of reviews have been published (75,96,97,98,99,100,101,102,103,104,105,106,107). Clinical investigations of fiber have employed a wide range of research strategies with sample sizes ranging from 5

to 40. In order to vary the fiber exposure, fiber additives, both separate from the meal and as part of the meal, and high and low fiber foods have been tested. Not only has the quantity of fiber been varied in these experiments, but so has the method of preparation, food source (i.e. fruits and vegetables, cereals, legumes) and fiber fraction (i.e. insoluble and soluble). A large number of glucose, lipid and hormone response variables have been measured under fasting, postprandial, and basal conditions. Single meals, second meal effects, and diet periods of days to months have been observed. Other potentially confounding or modifying variables in these studies include subject disease status (i.e. normal versus diabetic, controlled versus uncontrolled diabetes, fasting plasma glucose levels, current therapy), other subject characteristics (i.e. age, sex, weight, fat distribution, physical activity) and meal characteristics (i.e. calories, amount and source of carbohydrate, fat and protein, and compliance on the longer term diets). The studies cited below are limited to studies of fiber in natural food form and administered under isocaloric conditions controlling for the percentage of carbohydrate, fat, and protein, and where both glucose and insulin were measured as response variables. All studies administered the diets in a randomized crossover design except for the study by Kay et al (108) which administered the high fiber diet followed by the low fiber diet in all patients. When serum insulin and glucose concentrations are cited below, values for the low fiber diet are presented first.

Haber et al (77), studied ten healthy volunteers and showed a dose response relationship between equal carbohydrate meals of whole apples, apple puree, and apple juice and postprandial serum glucose and plasma insulin levels. Juice evoked an earlier and greater serum insulin rise than whole apples (mean peak value: 44.7 versus 23.9 mU/l, $p < 0.05$) and apple puree was intermediate. There

was no change in the postprandial glucose rise. However, in the second hour after the juice meal, mean plasma glucose fell 18 mg/dl below fasting levels. Plasma glucose after whole apples did not fall below fasting levels and puree was intermediate. The hypoglycemic response may be explained by the plasma insulin findings. The authors suggest that, given the similar postprandial glucose rise, a lower serum insulin response to whole apples results from reduced stimulation of the enteroinsular axis following a high fiber meal.

Similarly, Golay et al (109) and O'Dea et al (110) studied the effects of processing white beans and rice, respectively, on metabolic parameters. In the first study, 50 gram carbohydrate portions of beans were processed into two physical forms, one maintaining the integrity of the bean cells (undamaged) and the other rupturing the bean cells (damaged). Golay et al found that eight non-diabetics fed undamaged white beans had nonsignificantly lower glucose response areas and significantly lower insulin response areas (mean 3-hour incremental insulin response area: 49 versus 26 uU/ml/hr; $p < 0.05$) than when fed damaged white beans. In eight persons with NIDDM, both the glucose and insulin areas were significantly lower (mean 3-hour incremental plasma glucose area: 150 versus 73 mg/dl/hr, $p < 0.001$; mean 3-hour incremental plasma insulin area: 67 versus 46 uU/ml/hr, $p < 0.05$). O'Dea et al compared ground and unground white and brown rice in six healthy males. The areas under both the glucose and insulin response curves were significantly higher with ground compared to unground white rice (mean 60 minute total glucose response area: 380 versus 280 mM/min, $p < 0.01$; mean 60 minute total insulin response area: 5000 versus 2200 uU/ml/min, $p < 0.005$). Similar differences were observed for ground and unground brown rice. Brown rice elicited a reduced response compared to white rice only in the unground form, but this decrease was not statistically significant.

Studies by Burke et al (111) and Potter et al (112) observed a gradient in the effect of dietary fiber on insulin and glucose parameters depending on the source of dietary fiber. Burke et al found significantly reduced levels of 24-hour urinary C-peptide (suggesting lower total insulin secretion) after two days on a diet with predominantly bean sources of fiber compared to predominantly cereal sources of fiber (1265 versus 1056 umol/mol creatinine, $p < 0.05$) in six healthy volunteers. Fasting blood glucose (FBG) was reduced 18 mg/dl ($p < 0.02$). Similar differences in C-peptide results were reported for eight obese NIDDM patients, however, FBG was not significantly different in this group. Plasma glucose and insulin values obtained by Potter et al were highest for glucose formula (no dietary fiber (DF)), then brown rice (2.8 grams DF), All Bran (18 grams DF), and the lowest values were obtained for pinto beans (16.2 grams DF). The peak glucose response to formula was 27 percent greater than to All Bran and pinto beans and the peak insulin response was nearly 2-fold greater. Similar to the study of apple juice by Haber et al (77) the plasma glucose for formula fell below fasting levels at three hours.

The studies described above have shown a consistent reduction of approximately 50 percent in peak serum insulin concentrations in normal subjects with a high fiber meal (77,109,110,112). Plasma glucose differences were smaller and less consistent across studies. Legume fiber appeared to have a greater effect than cereal fiber in reducing both glucose and insulin parameters (111,112). With the exception of the study by Burke where subjects were given the test diet for two days, the above cited studies were single meal tolerance tests. These studies address the acute effects of fiber ingestion but are limited to observations on changes in postprandial responses.

Longer term studies of fasting and basal levels of glucose and insulin can address whether fiber has an effect on mean levels of these parameters or an effect only on the excursions about the mean (i.e. postprandial rise and fall). Since, by definition, fasting blood glucose is raised in NIDDM, a reduction in fasting or basal levels of blood glucose and insulin with high fiber diets would provide support for the hypothesis that high fiber acts as a protective factor in the etiology of NIDDM.

Table 1.2 summarizes longer term studies of high versus low fiber diets. Of these studies, Albrink et al (113) and Ullrich et al (114) were the only two authors to study non-diabetic healthy volunteers. However, the percent of calories as carbohydrate was extremely high (e.g. 70%) and the meals provided were not usual meals. In Albrink's study, the control diet was a low fiber liquid formula and the high fiber diet was composed primarily of high fiber cereal and beans. In the study by Ullrich, both groups had solid meals, but again the diet was predominantly cereals and beans. Both studies used an oral glucose tolerance test (OGTT) and a meal tolerance test (MTT). No difference in fasting levels of glucose and insulin was observed in either study. Albrink et al observed significantly lower insulin levels during the high fiber MTT. The area under the insulin response curve above baseline levels was 42% lower for the high fiber meal than for the low fiber meal. No differences in glucose levels were observed. No significant differences were observed by Ullrich et al when high and low fiber diets were compared.

Studies by Kay et al (108), Karlstrom et al (115), and Hollenbeck et al (116), were conducted for longer time periods (two to four weeks) in subjects with NIDDM. All three studies administered "conventional" diets where the high fiber diet substituted less refined preparations (whole wheat bread versus white

Table 1.2. Longer term studies of dietary fiber[1]

Author (Ref)	Number of Subjects Normal	NIDDM	IDDM	Time[2]	% of Calories Fat	CHO	Protein	Gms Fiber[3] /2000 Kcal	Change[7] Ins	Gluc
Albrink (113)	7	0	0	1 week	15	70	15	0.8 vs 14[4]	-	NS[8]
Ullrich (114)	8	0	0	4 days	13	72	14	15 vs 39[5]	NS	NS
Kay (108)	0	5	0	2 weeks	37	40	23	10 vs 30[6]	-	-
Karlstrom (115)	0	14	0	3 weeks	34	47	21	22 vs 48	NS	-
Hollenbeck (116)	0	6	0	4 weeks	25	60	15	22 vs 53	NS	NS

[1]Diets used natural foods, not fiber additives. They were approximately isocaloric and controlled for percent of fat, carbohydrate and protein.
[2]Time period each diet was administered.
[3]Total dietary fiber (68), except where noted otherwise.
[4]crude fiber.
[5]neutral detergent fiber.
[6]gms/day: sufficient data to calculate gms/2000 kcal was not presented.
[7]Direction of change in insulin and glucose parameters in high compared to low fiber diets. (e.g. "-" means that that parameter was lower on the high fiber diet)
[8]NS=no statistically significant difference.

bread, apple slices versus apple juice) and high fiber foods (40% bran flakes versus rice krispies, pea soup versus cream of tomato soup, whole corn versus rice). Karlstrom also used fiber enriched crisp-bread to increase fiber intake. In the following presentation of results, data for the low fiber diets are presented first, followed by comparable values on the high fiber diets. Kay et al observed significant changes in one of two glucose parameters (mean basal plasma glucose concentrations: 9.2 versus 9.1 mmol/l, not significant; maximum postprandial plasma glucose increase: 30 versus 14 percent, $p < 0.01$), two insulin parameters (mean basal plasma insulin: 22 versus 17 mU/l, $p < 0.05$; mean 60 minute

postprandial increment above basal: 14.8 versus 7.7 mU/l, $p < 0.05$), and in one of two GIP measures (mean basal plasma GIP: 891 versus 581 ng/l, $p < 0.05$; mean plasma GIP levels 90 to 150 minutes postprandial, not significant). On the high fiber diet, Karlstrom et al found significant decreases in all glucose parameters (mean 24-hour urinary glucose: 102 versus 64 mmol/l, $p < 0.05$; mean fasting blood glucose: 10.1 versus 9.2 mmol/l, $p < 0.01$; mean peak postprandial blood glucose: 16.2 versus 13.9 mmol/l, $p < 0.01$; and mean blood glucose at 0700 hours: 10.1 versus 9.5 mmol/l, $p < 0.05$, 1100 hours: 15.1 versus 13.1 mmol/l, $p < 0.01$, and 1500 hours: 13.2 versus 12.7 mmol/l, not significant), but no change in fasting or postprandial serum insulin concentrations. Hollenbeck et al found no difference in fasting or day long glucose and insulin parameters at four weeks of followup.

The discrepancy in results across these studies may be due to factors that remained uncontrolled. Administration of low and high fiber diets to all subjects forces subject characteristics to be similar across fiber groups, however, subject characteristics which may modify the effect of fiber intake on glucose and insulin parameters have not been controlled. For example, if a high fiber diet reduced serum insulin levels in non-obese patients but not in obese patients, then mixing results of obese and non-obese subjects would obscure the true relationships. In the six patients studied by Hollenbeck et al, the BMI ranged from 20.5 to 29.7 and fasting glucose ranged from 150 to 255 mg/dl. In the study by Karlstrom et al, "all patients were poorly controlled on admission and had high blood glucose values." Kay et al studied five elderly residents of a nursing home with percent ideal weight for age ranging from 101% to 151%.

Inferences about diet in populations from experimental studies have been limited by the short period of time on special diets, by limited power, and by

lack of control for potentially modifying variables like obesity. The metabolic response observed may reflect a process of adaptation to dietary change rather than what would occur under longer term diets.

Few epidemiologic studies measuring both fiber intake and diabetes status in individuals have been reported. Two studies in vegetarians are consistent with the hypothesized relationship between low fiber intake and diabetes. Gear et al (117) reported biochemical and hematological variables including FPG in 91 vegetarians and 264 nonvegetarians. Nonvegetarians had significantly higher FPG concentrations than vegetarians (101 versus 90 mg/dl, $p < 0.001$). A second study, among 25,698 adult White Seventh-day Adventists identified in 1960 and followed 21 years, found that the risk of diabetes as the underlying cause of death was approximately one-half the risk for all U.S. Whites. In this group, non-vegetarians had an increased risk of subsequent diabetes-related mortality of 1.8 (95% confidence limits: 1.3,2.5) in males and 1.4 (95% CL: 1.2,1.8) in females when compared to vegetarians and adjusted for age and percent desirable weight (118). To the extent that meat consumption correlates with a low intake of fiber, these data are consistent with an inverse association between fiber and diabetes incidence. However, inferences about etiology from mortality data are limited because 1) mention of diabetes on the death certificate is known to underrepresent the number of people with diabetes who die by as much as 48 percent (119), and 2) mortality is affected by both etiologic and prognostic risk factors making it more difficult to isolate etiologic factors.

Prospective data on diet and subsequent development of NIDDM has been reported for the Pima Indians (120). One hundred and eighty seven non-diabetic Pima Indian women, aged 25 to 44, received a modified Burke dietary interview in 1968. After an unspecified period of followup, 87 of the subjects, or 47%,

developed NIDDM. These subjects had significantly higher intakes of total carbohydrate and starch than subjects who did not develop NIDDM. To the extent that fiber consumption is positively associated with starch consumption, these results are not consistent with the hypothesized relationship between low fiber intake and diabetes incidence. However, when the authors looked at NIDDM incidence by tertiles of intake, increasing intakes of calories, fat, carbohydrate, and starch were all highly correlated with increasing NIDDM. Subjects developing NIDDM ate 100 calories more per day on average and comparisons were not adjusted for weight gain or obesity which may account for the observed positive association between carbohydrate intake and NIDDM.

The most consistent finding to emerge from this review is that dietary fiber is inversely associated with post-prandial concentrations of blood insulin. In support of this finding, GIP has also been measured in higher concentrations in the blood after a low fiber meal (108,121,122,123,124,125). GIP is the primary humoral secretion of the intestine responsible for potentiation of insulin release (64) and both the transitory and prolonged phases of insulin release are stimulated by GIP (126). Work by Thomas and colleagues (127) indicates that the primary site of endogenous GIP release is the proximal small intestine but smaller quantities are also released by the distal small bowel. Since fiber slows gastric emptying and separates nutrients from digestive enzymes and absorptive surfaces in the small intestine, it follows that nutrients associated with a low fiber meal would be absorbed proximally relative to nutrients associated with a high fiber meal. GIP secretion would therefore be enhanced and postprandial insulin levels would be elevated.

Chronically elevated concentrations of insulin in the blood (i.e. hyperinsulinemia) have been shown to decrease the number of cellular insulin

receptors leading to decreased insulin sensitivity. Such "down regulation" occurs in obesity and in NIDDM (128). With a reduced number of receptors, a greater proportion of the receptors must be occupied to obtain a maximal biologic response and a higher insulin concentration is needed to saturate the reduced number of receptors (129). The ability of insulin to maintain glucose homeostasis is maximized at low insulin concentrations and the effects are buffered at elevated concentrations (130). It is hypothesized here that habitual high insulin levels following low fiber meals contribute to down regulation of insulin receptors, which compromises the body's ability to maintain glucose homeostasis.

If this hypothesis is true, and if this mechanism is operating in the population under investigation, then diabetics on average would be expected to report lower intakes of fiber prior to diagnosis of their disease than controls. The study presented in the following chapters used a case-control design to evaluate the role of dietary fiber as an etiologic risk factor for NIDDM in a free-living population-based setting.

CHAPTER 2

Methods

Summary

This study employed a case-control design. Diet histories of cases of NIDDM and controls were compared to determine whether low dietary fiber intake was positively associated with the occurrence of NIDDM. The study group included prevalent, medically diagnosed diabetics between the ages of 20 and 74 years, residing in Alamosa and Conejos counties in the San Luis Valley of southern Colorado and controls selected from the same area by geographically-based multi-stage sampling. An oral glucose tolerance test (OGTT) was used to confirm the presence or absence of NIDDM in both cases and controls. Fiber intake prior to NIDDM diagnosis, or a comparable time in the past for controls, was obtained by a food frequency questionnaire (FFQ) developed to account for dietary habits unique to this population. Current fiber intake was ascertained by FFQ and 24-hour diet recall. Three analyses were performed: 1) cross-sectional analysis of current fiber intake and plasma insulin concentrations among the controls; 2) comparison of prior fiber intake in previously known diabetics and nondiabetic controls; and 3) comparison of current fiber intake in previously undiagnosed diabetics and normal controls. Analyses were adjusted for age, sex, ethnicity, and body mass index. Other factors studied included disease duration, waist to hip ratio, parental history of diabetes, calorie intake, carbohydrate intake, and season of interview.

Study Site

Alamosa and Conejos counties in the south-central San Luis Valley (SLV) were selected as the geographic boundaries for this study (see map, Figure 2.1).

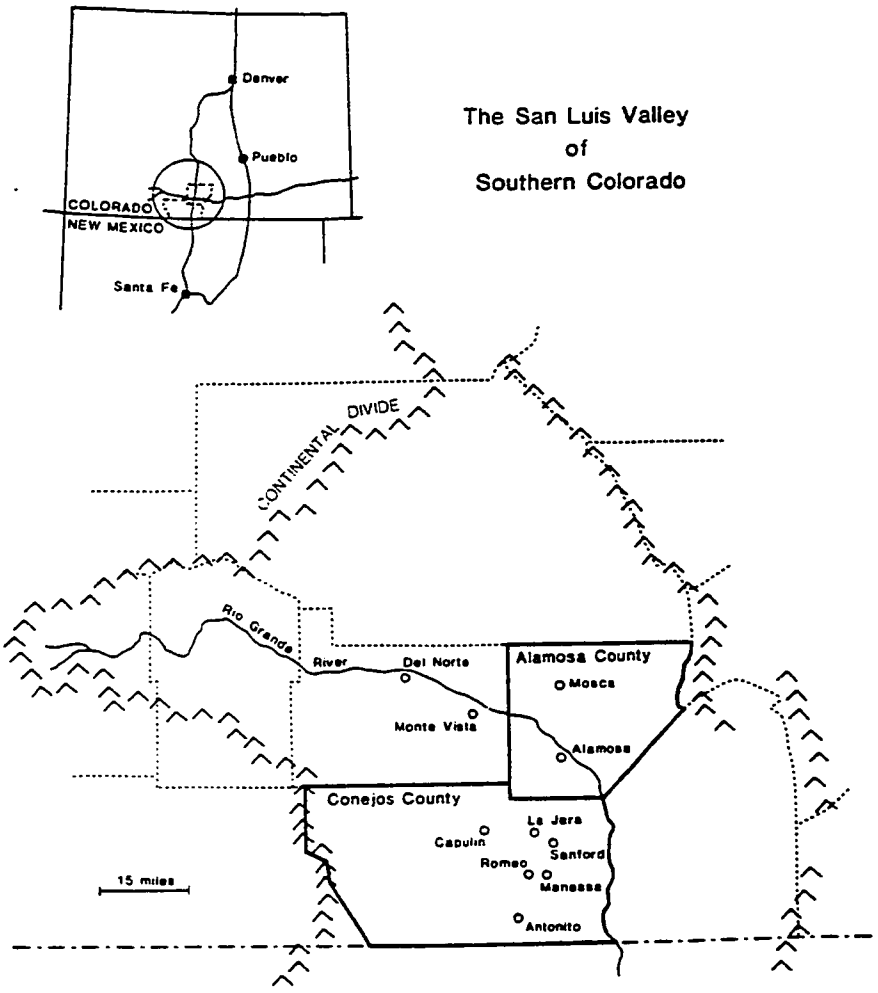

Figure 2.1. Alamosa and Conejos counties in the San Luis Valley, Colorado

In the 1980 U.S. census (131), this area had a population of 19,593 persons including 8,539 persons of Spanish origin and 10,753 non-Spanish whites. Conejos county has the highest proportion of Hispanics of any county in the state and it nearly coincides with the original Spanish land grant area of

settlement where many of the oldest families still reside. Anglo residents of Conejos County include a high proportion of Mormon families that also descended from early settlers. Alamosa County contains the largest city in the SLV. It serves as the center for transportation, commerce and medical care in the valley. Medical care for these two counties is provided by a small number of physicians and two hospitals, one in each county. In order to develop and maintain good relationships with residents and health care providers in the study area and to help assure that the research plan and methods were sensitive to local concerns, an advisory board was formed of 18 members from the community. These persons included prominent church, civic, and medical community leaders who were asked to review and endorse study procedures.

Identification of Persons with Diabetes

Case ascertainment was accomplished through review of medical records, conducted or supervised by the SLVDS field coordinator, in health care facilities in the SLV. All health care facilities in the study area participated. This included two group practices and a small solo practice in Alamosa, the Alamosa County Hospital, the Evergreen Nursing Home, two clinics in La Jara, two clinics in Antonito, and the Conejos County Hospital. One physician's office in nearby Monte Vista was also included because the physician had previously practiced in Conejos County and still had a number of patients who drove 30-50 miles to see him. The other clinics in Monte Vista and Del Norte, and the Monte Vista Hospital were contacted but the physicians reported that there were extremely few, if any, diabetics seen from the study area, so medical records abstracting was not done in these facilities. Additional diabetics were identified following local advertisement of the SLVDS and in the SLVDS

household enumeration for controls. People who self-reported to SLVDS, either in person, by phone, or through the sampling process, were added to the list of diabetic cases and their medical records were abstracted if they could be located.

The procedures used to identify diabetics in each of the medical care facilities varied due to differing organization and record keeping practices. Computerized billing in the two Alamosa group practices and in the Monte Vista practice allowed a computer generated list of diabetics to be prepared based on diabetes diagnostic code 250. In the other private practices all charts were manually reviewed for mention of diabetes. Where possible, these lists were compared with billing transaction printouts. Hospital records were abstracted where a primary or secondary diagnosis of diabetes was reported on discharge.

The names of persons with diabetes were entered onto a master list where duplicates were identified. A letter was then sent from the physician to the subject requesting permission for the SLVDS to review the medical record. The letter described the study and asked the subject to call the physician's office within one week if they did not want their medical record reviewed. If no response was received within one week, demographic data and clinical data related to diagnosis and treatment were abstracted from the medical record. Subjects who were 20 to 74 years of age and living in the study area were then entered on the diabetic contact list to be contacted for a clinic visit. The medically diagnosed diabetics identified by medical record review and self report are described in detail below.

Control Selection

Nondiabetic controls were selected using a geographically-based multi-stage sampling procedure. First, a random sample of households was selected. Second, an age, sex, and ethnic group stratified sample was drawn from all persons enumerated in the households of stage one. The sampling strategy was implemented by county.

In Alamosa county, rural addressing maps, utility hook-up listings, 1979 state highway maps, and the 1984 phone books were used to identify all structures. Because rural addressing maps and utility hook-up listings were not available in Conejos county, 1979 United States Geological Survey (USGS) maps and aerial photographs, 1979 state highway maps, and the county assessors maps were used in conjunction with the 1984 phone books to locate all structures. The structures on aerial photographs were transferred to the state highway maps. These maps were then crosschecked with the dwellings marked on the county assessor and USGS maps. Ambiguous areas were verified directly by the field staff.

An initial enumeration of 10 percent of all occupied structures in Alamosa county yielded too few eligible controls in the older age groups and an additional seven percent of the households in that county were enumerated. In the seven percent enumeration, census enumeration districts (ED) with a median age of 26 years or greater were selectively sampled. Households were randomly sampled within these ED. Based on the need to resample in Alamosa county, an initial 26 percent sample of occupied structures was selected in Conejos county.

Households that were selected in stage one of the sampling procedure were enumerated by a five to ten minute home interview (Appendix A). An index adult was interviewed to determine the demographic composition of the

household. For all persons living in the household, the interview asked first and last name, relation to head of household, date of birth, sex, Spanish origin, whether they were born in the United States, and whether they had ever been told by a doctor that they had diabetes. Table 2.1 describes the enumeration response and Table 2.2 compares the demographic characteristics of the enumerated sample (column 2) with the 1980 U.S. census for these two counties (column 1). The differences by age and Spanish origin are largely due to the over-sampling that occurred in Conejos county. County-specific age, sex, and Spanish origin distributions suggest that the enumerated sample was representative of the two county population.

Table 2.1. Response to enumeration of households

	Alamosa		Conejos	
	Number	Percent	Number	Percent
Total Occupied Structures[1]	3,921	100.0	2,356	100.0
Occupied Structures Sampled	679	17.3	630	26.7
Structures Attempted	769	100.0	728	100.0
Vacant Structures	90	11.7	98	13.5
Occupied Structures	679	100.0	630	100.0
Interviews Completed	652	96.0	612	97.1
Refusals	27	4.0	18	2.9

[1]Data source (132).

In stage two of the sampling procedure, a random sample of individuals stratified by age, sex, and Spanish origin was selected from the household enumeration list. Persons reported to have diabetes were excluded from the sampling frame. In Conejos county, all persons enumerated over 50 years of age were included to maximize the number of controls in the older age groups. Because the average household size was 2.8 in Alamosa and 3.3 in Conejos (133)

Table 2.2. Comparison of the population characteristics of the household enumeration and frequency matched sample with the 1980 United States census for Alamosa and Conejos counties by age, Spanish origin, sex and county

	1980 Census N=11,379 percent	Enumerated[1] N=2,257 percent	Selected[2] N=997[3] percent	Percent of Population Selected
	(1)	(2)	(3)	(4)
Age Group				
20-29	31.9	23.1	4.9	1.3
30-39	21.2	24.6	7.7	3.2
40-49	15.7	15.6	17.9	10.0
50-59	14.5	16.0	30.3	18.3
60-69	12.1	15.3	29.2	21.2
70-74	4.6	5.5	10.0	19.2
Spanish Origin				
Yes	40.5	47.2	43.7	9.5
No	59.5	52.8	56.3	8.3
Sex				
Male	49.3	49.0	47.9	8.5
Female	50.7	51.0	52.1	9.0
County				
Alamosa	62.1	50.4	48.5	6.8
Conejos	37.9	49.6	51.6	11.9
Total	100.0	100.0	100.0	8.8

[1]Enumeration of 20.9 percent of occupied structures in Alamosa and Conejos counties.
[2]Stratified sample randomly selected within enumerated sampling frame by 10-year age groups, sex, Spanish origin, and county.
[3]Excludes four sampled controls who were age ineligible in Table 2.3.

and analyses were stratified by age and sex, within household correlation was expected to be minimal. The demographic characteristics of persons sampled in stage two are presented in column 3 of Table 2.2. Column 4 of Table 2.2 shows the percent of the total population sampled in each of the sampling strata. Compared to the 1980 census, the frequency matched sample was older and more

often resident in Conejos county. Only minor differences existed by sex and Spanish origin.

Percent Response and Indicators of Potential Response Bias

After a diabetic or control subject was identified and determined to be eligible for a clinic visit, his name was placed on a contact list. A letter (Appendix B) was sent to each patient describing the purpose of the study and telling him that he would be contacted for his participation. A week or more after the letter was mailed, attempts were made to contact the subject by phone to set up an appointment. Once an appointment was made, the subject was sent a packet containing a map to clinic, instructions for the clinic visit (Appendix C) and a brief self-administered medical and family history.

Each attempt to contact a subject was noted on the contact sheet. When the subject could not be reached during the day, additional attempts were made in the evening and on Saturday. If the subject had no phone, a home visit was made to contact the patient and schedule an appointment. In cases where the initial letter was returned as undeliverable or the phone had been disconnected, the subject was traced by obtaining a forwarding address from the post office or from other sources such as neighbors or relatives; returning to the patient's medical record for more recent location data; or sending a self-addressed, stamped postcard requesting a new phone number and address and providing the subject with the option of refusing or obtaining more information about the study.

Some of the subjects contacted did not want to set up an appointment. If they wanted to come at a later date, they were asked when would be a better time to contact them and they were recontacted at that date. If the subject

refused to participate, this was noted and he was contacted six months later to see if he had changed his mind, and if not, he was asked to answer a brief refusal questionnaire over the phone (Appendix D). This questionnaire is described in more detail below.

Cases and controls were initially screened for age and residence eligibility before being contacted for clinic, as described above. Further eligibility criteria were determined while scheduling the clinic appointment and during the clinic visit. To be included as clinic eligible, subjects had to still be resident in the study area, between 20 and 74 years of age, mentally and physically competent as determined by the SLVDS field coordinator, and alive when appointment scheduling was attempted.

Overall percent response and exclusions due to ineligibility are shown in Table 2.3. Table 2.4 shows the percent response by age and ethnicity. Anglo response was 15.8 percent higher among diabetics and 11.0 percent higher among controls compared to Hispanic diabetics and controls, respectively. Within ethnic groups there was no difference in response by sex for diabetics, but female control response rates were higher than males by 4.6 percent in Anglos and by 13.9 percent in Hispanics.

Data were available for comparison of respondents and nonrespondents from the household enumeration interview for controls and from medical records abstracting for diabetics. These data, summarized in Table 2.5 and Table 2.6, indicate that responders were slightly younger and more often Anglo than nonresponders. Alamosa county had a higher percent response than Conejos county.

The questionnaire administered over the phone to subjects who refused to come for a clinic visit included selected questions from the interviews

Table 2.3. Percent response in the San Luis Valley Diabetes Study by case status, 1984-1986

	Diabetics		Sampled Controls	
	Number	Percent	Number	Percent
Total[1]	643	100.0	1001	100.0
Refused record review	4	0.6	not applicable	
Residence ineligible	32	5.0	77	7.7
Age ineligible	145	22.6	4	0.4
Died before clinic visit	29	4.5	8	0.8
Mentally or physically unable to complete clinic	11	1.7	22	2.2
Other	2	0.3	3	0.3
Clinic Eligible[2]	420	100.0	887	100.0
Attended clinic	343	81.7	607	68.4
Responders[3]	299	71.2	568	64.0
Nonresponders	121	100.0	319	100.0
Refusal questions asked	104	86.0	263	82.4
No Refusal Interview	17	14.0	56	17.6

[1]Medically diagnosed diabetics identified by medical record review and self report. Controls from stage two of multistage sampling procedure.
[2]Study area resident, alive, 20-74 years of age, mentally competent, and controls without a self-reported medical diagnosis of diabetes.
[3]Subjects who came to clinic and completed the prior FFQ.

administered to subjects participating in clinic so that there would be comparable information on diabetic and control responders and nonresponders. Demographic, health related behavior, and disease outcome variables were included in order to determine if subjects who refused to participate in the study were different from subjects who participated with respect to factors which might bias the study findings. Refusal interviews were obtained on 86.0 percent of diabetic nonrespondents and 82.4 percent of control nonrespondents (Table 2.3). Characteristics of responders and nonresponders, based on the refusal interview, are summarized in Table 2.7. These data suggest that both

Table 2.4. Percent response by sex and ethnicity

	Diabetics			Controls		
	Number of Responders	Number Eligible	Percent Response	Number of Responders	Number Eligible	Percent Response
Hispanic						
Males	65	101	64.4	101	198	51.0
Females	109	165	66.1	131	202	64.9
Total	174	266	65.4	232	400	58.0
Anglo						
Males	68	83	81.9	147	221	66.5
Females	57	71	80.3	189	266	71.1
Total	125	154	81.2	336	487	69.0
Total	299	420	71.2	568	887	64.0

responders and nonresponders were long term residents of the SLV. Responders had attained a higher level of education on average and were more likely to be currently married. The responder's self assessment of health was more often excellent or good compared to the nonresponders and diabetic responders had not seen a doctor as often in the past year. History of hypertension, alcohol use and family history of diabetes were all more common among responders. Among diabetics, responders had not had their disease as long as nonresponders. Responders were less likely to be taking insulin for their diabetes and more likely to be taking oral hypoglycemic agents, but the percent on diet alone was about the same. The impact of these differences is discussed in Chapter 4.

Interview, Examination and Laboratory Procedures

Study participants were scheduled for a four hour morning clinic visit. They were asked not to eat or drink anything for 12 hours prior to their appointment time. The detailed instructions sent to each subject are included in Appendix C. General informed consent (Appendix E) was requested when the

Table 2.5. Comparison of respondents and nonrespondents among diabetics by age, sex, Spanish surname, residence, and disease duration

	Percent of Responders N=299	Percent of Nonresponders N=121	Percent of Total N=420
Age 20-29	3.3	1.7	2.9
30-39	9.0	5.8	8.1
40-49	11.7	9.1	11.0
50-59	33.8	24.8	31.2
60-69	28.1	35.5	30.2
70-74	14.0	23.1	16.7
Sex male	44.5	42.1	43.8
female	55.5	57.9	56.2
Spanish Surname	58.2	76.0	63.3
County of residence			
Alamosa	47.5	29.8	42.4
Conejos	52.5	70.2	57.6
Disease Duration			
< 2 years	22.4	20.9	22.0
2-4 years	30.0	19.8	27.1
5-9 years	22.0	27.9	23.5
10-19 years	19.5	27.9	21.7
20+ years	6.5	3.5	5.7
(% missing data	17.7	28.9	21.0)

DATA SOURCE: SLVDS medical records abtraction

patient arrived at clinic and after a description of the clinic procedures. Fasting blood samples were drawn and then the subject was given 75 grams of Koladex ororangedex (Custom Labs, Inc., Baltimore, MD) to drink. One and two hour blood samples were drawn following the glucose load. During this time, interviews and a physical examination were conducted. Table 2.8 summarizes the components of the interview, physical examination, and laboratory procedures. Protocols for the variables included in this analysis are described in more detail below.

Table 2.6. Comparison of respondents and nonrespondents among controls by age, sex, Spanish origin, residence and place of birth

	Percent of Responders N=568	Percent of Nonresponders N=319	Percent of Total N=887
Age 20-29	5.1	4.1	4.7
30-39	8.5	5.3	7.3
40-49	17.6	17.6	17.6
50-59	33.6	27.3	31.3
60-60	26.8	35.4	29.9
70-74	8.5	10.3	9.1
Sex male	43.7	53.6	47.2
female	56.3	46.4	52.8
Spanish Origin	40.9	52.7	45.1
County of residence			
Alamosa	49.5	43.3	47.2
Conejos	50.5	56.7	52.8
Born in U.S.	98.1	98.7	98.3

DATA SOURCE: SLVDS household enumeration

Table 2.7. Selected characteristics of responders and nonresponders by case status

	Diabetics			Controls		
	Percent of Responders N=299	Percent of Nonresponders N=104	Percent of Total N=403	Percent of Responders N=568	Percent of Nonresponders N=263	Percent of Total N=831
Length of residence in San Luis Valley in years						
< 5	6.7	3.9	6.0	3.5	2.3	3.1
5-9	3.7	2.9	3.5	5.8	3.4	5.1
10-19	5.7	3.9	5.2	7.6	8.0	7.7
20 +	84.0	89.4	85.4	83.1	86.3	84.1
Years of education completed						
< 8	13.7	37.9	19.9	9.2	17.8	11.9
8-11	35.5	25.2	32.8	19.0	30.6	22.6
12	29.1	24.3	27.9	34.2	30.6	33.1
13 +	21.7	12.6	19.4	37.7	20.9	32.5

Table 2.7. (continued)

	Diabetics			Controls		
	Percent of Responders N=299	Percent of Nonresponders N=104	Percent of Total N=403	Percent of Responders N=568	Percent of Nonresponders N=263	Percent of Total N=831
Marital status						
never	3.4	4.8	3.7	4.2	6.1	4.8
married	76.9	62.5	73.1	79.2	71.0	76.6
divorced	4.4	5.8	4.7	6.5	6.5	6.5
other[1]	15.4	26.9	18.4	10.0	16.4	12.1
Self assessment of health						
excellent	12.1	8.7	11.2	38.9	34.2	37.4
good	41.8	34.6	39.9	40.0	39.2	39.7
fair	30.3	33.7	31.2	16.2	19.0	17.1
poor	15.8	23.1	17.7	4.9	7.6	5.8
Number of physician visits in past year						
0	11.5	6.8	10.3	30.3	31.4	30.7
1-3	34.8	31.1	33.8	47.2	47.1	47.2
4 +	53.7	62.1	55.9	22.5	21.5	22.1
Current reported weight in pounds						
< 100	1.7	2.0	1.8	1.1	1.6	1.2
100-149	37.5	45.0	39.4	45.6	41.6	44.4
150-199	44.8	39.0	43.4	41.9	50.6	44.6
200 +	16.1	14.0	15.5	11.4	6.3	9.8
HISTORY OF:						
Hypertension	60.2	49.0	57.3	29.3	33.6	30.6
Smoking[2]	55.2	52.9	54.6	54.4	53.2	54.0
Alcohol ever	66.2	49.0	61.8	72.9	68.1	71.4
DM in family	61.1	57.8	60.2	31.3	24.5	29.1
Duration of diabetes in years				Not Applicable		
< 5	37.2	26.4	34.8			
5-9	23.1	17.2	21.8			
10-14	19.3	21.8	19.9			
15 +	20.3	34.5	23.6			
Current treatment				Not Applicable		
Insulin	47.9	58.0	50.3			
Orals	26.0	19.3	24.5			
Diet	53.8	58.0	54.8			
Diet only	10.1	9.1	9.8			

DATA SOURCE: SLVDS clinic visit and telephone refusal interview
[1]Includes widowed, separated, and living together
[2]Percent who smoked more than 100 cigarettes in their lifetime

Table 2.8. Summary of components included in the interview, physical examination, and laboratory

INTERVIEW

Demographic
- Name, address, telephone
- Date of birth, sex
- Social security number
- Contacts for follow-up
- Family composition
- Residence history
- Marital status

Socioeconomic Status
- Education
- Occupation
- Income
- Social/occupational mobility

Acculturation
- Ethnic group identity
- Place of birth
- Language use
- Childhood and adult ethnic associations
- Traditional versus nontraditional Hispanic value orientations to children and family

Social-psychological
- Social-psychological well-being
- Satisfaction with life
- Social connectedness/isolation
- Religious affiliation and attendance

Diabetes
- Other known diabetics
- Family history
- Weight history
- Duration, place of diagnosis
- Complications
- Medications

Cardiovascular Disease
- Family history
- High blood pressure history
- Angina (Rose Questionnaire)
- Intermittent claudication
- Congestive heart failure
- Exercise tolerance

General
- Major illness history
- Smoking history
- Pregnancy outcome (women)
- Exercise and activity levels

Diet
- 24-hour recall
- Food frequency questionnaire about fiber and fat intake
- Alcohol intake

EXAMINATION

Weight and height
Anthropometric measures
- calf, arm, waist, iliac, and thigh circumference
- triceps, subscapular, and suprailiac skinfolds

Skin reflectance
Visual Acuity
Retinal photographs
Peripheral neuropathy
Blood pressure
Electrocardiogram
Pulses, bruits
Amputations
Doppler survey - pre and post submaximal exercise, ankle/arm pressures, segmental leg pressures

Table 2.8. (continued)

LABORATORY

Serum:	Glycosylated hemoglobin
Glucose (fasting, 1 hr, 2 hr after 75 gram glucose load)	Genetic markers
Insulin (fasting, 1 hr, 2 hr)	Hematocrit
C-peptides (fasting, 1 hr, 2 hr)	Urine:
Triglycerides	glucose
Total and HDL cholesterol	protein
Creatinine, BUN	ketones
Uric acid	creatinine
Freezer storage	

Classification of Diabetes. Type of therapy and the results of a 75 gram OGTT were used for both cases and controls to classify persons with diabetes. Subjects reporting that they were currently on insulin or oral antidiabetic drugs were considered diabetic without further testing. An OGTT was administered to all subjects who were able to fast at least eight hours prior to their clinic visit. The 1985 World Health Organization (WHO) criteria for diabetes (134) were then applied as outlined in Table 2.9. One control did not have fasting or OGTT glucose values and based on a casual plasma glucose level of 91 mg/dl was classified as normal.

Table 2.9. WHO criteria for diabetes mellitus

	Plasma Glucose Concentration (mg/dl)
DIABETES MELLITUS	
fasting value	≥ 140
OR	
OGTT 2 hour value	≥ 200
IMPAIRED GLUCOSE TOLERANCE	
fasting value	< 140
AND	
OGTT 2 hour value	140-199

After subjects were classified as normal, impaired glucose tolerant (IGT), or diabetic, the diabetic group was subtyped as IDDM or NIDDM. The criteria used for subtyping included insulin use and C-peptide levels. In the absence of C-peptide data, reported age of diagnosis and duration of insulin treatment were used as criteria. Subjects who were currently on insulin with all available C-peptide values (fasting, 1 hour, 2 hour, or casual) less than 0.1 pmol/ml were classified as IDDM. If no C-peptide data were available, subjects who were diagnosed before 18 years of age and who had been on continuous insulin treatment were classified as IDDM. Subjects not meeting the above criteria were classified as NIDDM. Analyses include cases classified as NIDDM. Subjects selected because of a reported diagnosis of diabetes who tested normal, IGT, or IDDM were excluded from all analyses. Controls diagnosed as NIDDM by OGTT in the SLVDS clinic were analyzed as a separate case group. The studywide distribution of cases and controls by diabetic status are presented in Table 2.10.

Table 2.10. Selection status by diabetic status

	Selection Status	
Diabetic Status	Cases	Controls
NIDDM	279	30
IDDM	25	0
IGT	16	89
Normal	23	488
TOTAL	343	607

Ethnicity. The 1980 U. S. Census question on Spanish Origin was used to determine ethnicity. The question asked "Are you of Spanish/Hispanic origin or descent?" The response categories were 1=no; 2=yes, Mexican, Mexican-

American, Chicano; 3=yes, Cuban or Puerto Rican; 4=yes, other Spanish/Hispanic. Categories 2, 3, and 4 were combined in the analysis.

Body Mass Index and Waist to Hip Ratio. Body mass index was calculated as current measured weight in kilograms divided by height in meters squared. The waist to hip ratio was calculated as waist circumference divided by iliac circumference. Waist circumference was measured at the bottom of the tenth rib at mid-respiration and the iliac circumference was measured at the most lateral tip of the iliac crest. All subjects were examined by the same observer.

Parental History of Diabetes. Subjects were asked whether their mother or father had diabetes in conjunction with similar questions for their grandparents, children, and siblings. Subjects were asked not to include a stepmother or stepfather, or any adopted brothers or sisters, or children who were not blood relatives. Parental history was "positive" if either the mother or father was reported to have had diabetes.

Season of Interview. The date of the clinic visit and United States Department of Agriculture (135) definition of seasons was used to determine the season of the interview. Seasons were defined as follows: spring (April-June), summer (July-September), fall (October-December), and winter (January-March).

Disease Duration. The self-reported age at diagnosis, birthdate, and date of the SLVDS clinic visit were used to calculate disease duration. Controls who were newly diagnosed with diabetes by OGTT during the SLVDS clinic visit were assigned a duration of zero and analyzed as a separate case group.

24-Hour Diet Recall. All subjects were administered a 24-hour diet recall. The interviewer asked the participant to remember everything he or she drank over the 24-hour period prior to starting to fast for the clinic visit. The instructions and documentation form for this interview are shown in

Appendix F. The interview took, on average, between 20 and 30 minutes to administer. Two types of aides were used to assist the participant in estimating portion sizes. The primary aide was a two dimensional visual aid called a Food Portion Visual (FPV) developed by Boston Nutrition Associates (136,137). The FPV is poster size. It has an "A" side with shapes representing volumes and a "B" side with shapes and thicknesses for calculating weights. Three dimensional aides were also available to estimate portion size if the participant had trouble relating to the two dimensional FPV. These three dimensional aides included:

1. a 12 inch ruler
2. two plates (10" and 7"), erasable pens, and a sponge for drawing dimensions of items like meat
3. two bowls (1.75 cup low, flat bowl and 2 cup rounded bowl)
4. three glasses (6 oz, 10 oz, 16 oz)
5. one tea cup and one mug
6. measuring spoons (1 TB, 1 TS, 1/2 TS, 1/4 TS)
7. butter pat (1 TS)
8. pinto beans for measuring "handfuls" of nuts, etc.
9. three dimensional thickness measure

Interviewers were trained and certified by the Nutrition Coding Center (NCC) at the University of Minnesota (138). The training emphasized interviewing techniques, objective probing and detailed documentation. Following the interview, the recall was edited by another interviewer for completeness. The recalls were batched and sent to NCC for coding, computer entry, and nutrient analysis. The nutrient analysis was based on version 14 of NCC's nutrient data base which was released in February of 1987. This version contained food composition data for total dietary fiber, insoluble fiber, and soluble fiber. NCC returned data tapes summarizing the nutrient content of each individual's diet during the 24-hour reporting period.

Average Fiber Intake. The FFQ (Appendix G) was designed to measure current and prior intake of fiber, fat, and alcohol. The participant was asked to estimate how many times they had eaten each food listed. This

questionnaire forced the participant to provide an average intake over the past 30 days and to contrast this current intake to their diet at a defined time in the past. For the diabetics, the prior time was one year before the diagnosis date reported in their medical record. If diagnosis date was missing from the medical record, the subject was asked their date of diagnosis at the beginning of the clinic visit. The assigned prior intake time in controls depended on age (20-44 and 45-74). Controls were assigned a prior time from a randomized list of disease durations plus one year determined from the medical records of the diabetic group.

Methods for developing a FFQ were not described in the literature reviewed prior to 1984. Most authors reporting conclusions from data collected on a FFQ had not included a description of the methods used to derive the food list. Only a few articles referred to some aspect of instrument development. For instance, Hinds et al (139) looked at 300 diet records previously collected on their population and determined a list of foods which contributed to 85 percent of that population's vitamin A intake. To develop the SLVDS FFQ, FFQ validation studies were reviewed. Expert nutritionists and epidemiologists were interviewed to determine how FFQs had been designed in other studies. Food frequency questionnaires from other studies were reviewed and a list of these studies is included in Appendix H.

Foods were selected and grouped within the USDA major food groups (bread and cereals, fruits, vegetables, dairy products and meats). Foods with similar dietary fiber content and foods thought to be readily associated were grouped. To isolate and group foods appropriately by dietary fiber content, dietary fiber data from Southgate (140), Paul and Southgate (68), Anderson (141), USDA Handbook 8-8 (142), USDA Handbook 8-9 (143), and

USDA unpublished preliminary data was entered into an IBM-PC using the data base manager, DBASE II. This allowed foods to be sorted according to the following fiber related variables:

a. grams of fiber per 100 gram edible portion
b. grams of fiber per average portion size
c. grams of fiber per 1000 kilocalories
d. percent soluble fiber of total fiber

and by data source. Within groups of foods with similar fiber content, clustering of foods was based on what subgroups were thought to be readily associated and to be eaten in common portion sizes. For example, based on fiber values alone, green peas could have been included with dried legumes but because they were believed to be more often thought of with green vegetables, they were kept separate.

A diet prestudy was conducted in the study area to be sure that local dietary patterns were taken into account. Fifty one valley residents completed a 24-hour recall and a three-day diet record. These records were manually reviewed for identification of foods not already included in the SLVDS FFQ food list. In addition, NCC did a computer analysis of which foods contributed most to the crude fiber content of this population's diet. Crude fiber was the only fiber measure available in the NCC nutrient data base at that time. This analysis was used to assure that major fiber sources had not been omitted from the food list, to group foods, or to omit foods not contributing significantly to the population's fiber intake. In addition, 24-hour recalls collected by the Women, Infant, and Children's program in Alamosa and the Commodities program in Conejos county were reviewed to better understand local food consumption patterns.

Existing FFQs had used a variety of formats and minimal data existed on the effects of these varying formats on study findings. These formatting issues

are presented in Appendix I. The first draft of the SLVDS FFQ was reviewed by Connie Dressor, NHANES nutritionist, Yvonne Sievert and Marilyn Buzzard, NCC nutritionists, and Marsha Jacobs, SLV consulting nutritionist who had worked in the study area for the previous five years. It was tested for ease of administration by SLVDS staff interviewers. A revised version of the FFQ was used during approximately 30 pilot interviews. Final changes were made and tested in 10 pilot interviews.

Data Handling and Quality Control

Data collection was carried out by a team of seven field staff resident in the SLV. The staff included the field coordinator who was trained as a nurse practitioner with experience in public health nursing and community organizing within the study area. She managed the SLVDS field operations, supervised the field staff, maintained relations with health care providers in the area, and performed the physical exam on all subjects. Two laboratory technicians were primarily responsible for the retinal photographs, pre and post submaximal exercise doppler studies for peripheral vascular disease, and for the collection, preparation, and shipping of blood and urine for laboratory analyses. Three bilingual interviewers, certified by the NCC to collect 24-hour diet recall data, and a community worker hired primarily to provide transportation to study participants, were responsible for household enumeration and clinic interviews, data editing, computer assisted data entry, scheduling of clinic appointments, and many other tasks that came up daily in the operation of the research clinic.

The protocol manual describing the administration of the dietary interview is included in Appendix J. Questionnaires were edited by a second interviewer

on the same day that the interview was administered to identify errors, omissions, or discrepancies. Data forms were sent to a local company for keypunching and independent verification. In addition, the SLVDS staff manually verified a ten percent sample of the keypunched data.

Data quality checks were run at each stage in the calculation of the fiber score. In the raw data, out-of-range values and inconsistent values between the "number of times eaten" and the interval (per day, week, or month) were queried. After the frequencies for each food were calculated, queries were done on extreme frequencies where the criteria for extreme depended on the particular food item. And finally, when the overall scores were calculated, extreme values were queried. Some of the queries resulted in data changes to correct data entry errors. In five cases, decisions were made to change the data that appeared to be an interviewer recording error. For example, eating cereal seven times a day with an otherwise varied diet, was changed to seven times a week. If there was no clear suggestion of error on the original intake form, extreme values were left in the data set. In other cases, the queries were informative as to what variations in the diet had created a high score. For example, eating All Bran once a day made a large contribution to the insoluble fiber score.

Dietary Fiber Scores

The following six fiber scores were calculated from the FFQ:

Frequency-based fiber scores
1. Fruits and vegetables
2. Legumes
3. Fruits, vegetables, and cereals

Semi-quantitative fiber scores
4. Total dietary fiber
5. Insoluble fiber
6. Soluble fiber

Three of these scores are frequency-based (How often are foods with fiber eaten?) and three are semi-quantitative estimates of fiber intake (fiber content times frequency). To calculate each fiber score, the frequency for a given food was multiplied by a weight and the products were summed over all food items on the questionnaire. The weights on the frequency-based scores were one or zero. A weight of one included the corresponding food item in the score. A weight of zero excluded that item. The weights used for the semi-quantitative scores were the fiber fraction content in grams for a common portion size of that food.

The fiber content of foods used in the semi-quantitative scores were compiled from several sources. Tables of the fiber values which were used as weights are included in Appendix K. The food items in the tables correspond to the food items on the FFQ. The data sources used are listed at the end of each table. Table K.4 lists the fiber values used for breakfast cereals. Subjects were asked which two cereals they ate most often. The fiber value for the first cereal listed by the respondent was arbitrarily multiplied by 0.6 and the fiber value for the second cereal was multiplied by 0.4. The two values were added together to come up with the fiber weight for cereal for that individual. This was done separately for hot and cold cereals.

Some subjects were ineligible for analysis (Table 2.3) due to missing data on more than 10 percent of the prior intake food items for a given score. Subjects stated that they could not remember what they ate that long ago. On other records a small amount of missing data occurred either because an individual could not remember for a particular item or because partial data was recorded during the interview. In cases where 90 percent or more of the data was present for a given score, missing data were replaced with the median

frequency that that food was eaten by all other participants with data on that food item.

Analysis

To assess the importance of dietary fiber intake as a risk factor for NIDDM, the odds ratio estimates for total dietary fiber intake, controlling for the effect of confounders, were calculated using a logistic regression model. The semi-quantitative total dietary fiber (SQTDF) score was chosen, a priori, as the best measure of fiber for initial analyses. The SQTDF score was used to determine which interaction terms would remain in the model and to determine the appropriate transformation for continuous variables. After a final model was chosen, odds ratios were calculated for the other five fiber scores using the same model.

In order to look for confounding and effect modification in the presence of other potentially important variables, a step down approach was used. Variables with the weakest prior grounds for being included in the model were evaluated for elimination first. The likelihood ratio test was used to test the overall statistical significance of each variable. Categorized data were tested for linear trend with the likelihood ratio test by treating the categories as continuous. Potential effect modifiers whose interaction terms were not statistically significant and which did not change the coefficient of fiber were eliminated from the model even if the variable, by itself, was a statistically significant independent risk factor for NIDDM in the data set. Potential confounders were kept in the model regardless of their statistical significance.

Continuous variables including fiber, body mass index, and waist to hip ratio, were evaluated to determine whether to include them as categorical,

linear, or transformed variables in the logistic model. Akaike's Information Criterion (AIC) (144) was used as a global measure to compare the fit of models that were not nested and where a likelihood ratio test could not be calculated. For example, AIC was used to compare continuous terms with factored categories for the same variable in an otherwise identical model. Standardized residuals were plotted against each variable in the final model as a visual check that the variables were appropriately entered into the model. Because of omission from the model of risk factors for NIDDM which were not related to fiber, global goodness of fit tests were less useful.

The need to ensure adequate numbers of older disease-free controls, and differences in the age composition of the two ethnic groups led to differential sampling of cases and controls by age and ethnic group. Lower than expected response rates in the first county sampled led to increasing the sampling fractions in the second county in order to ensure a sufficient number of subjects for analysis. Because of differential sampling of the cases and controls by age, sex, ethnic group, and county, all prevalent case-control analyses included a known constant in the logistic regression which was different for each subject. The constant was calculated as minus the logarithm of the sampling fraction for controls in the age, sex, ethnic group and county stratum for that subject. This procedure corrects the coefficients in the logistic regression analysis and allows odds ratios for sampling related variables to be calculated without sampling induced bias. Sampling related variables that would not otherwise need to be included in the model do not need to be included as covariates when this procedure is used (145). For example, if dietary differences explained the difference in NIDDM prevalence between ethnic groups, adjusting for ethnicity would incorrectly reduce the apparent odds ratio

relating dietary fiber to NIDDM. Including the known constant for sampling differences by ethnicity and excluding the ethnicity covariate from the logistic regression allowed this potential source of bias to be evaluated.

In the case-control analyses of previously undiagnosed cases, a procedure recommended by Willett and Stampfer (146) was used to adjust for total calorie intake. Using linear regression, kilocalories was regressed on each of the nutrients of interest. The residuals, interpreted as calorie adjusted intakes, were then included as independent variables in logistic regression analyses.

Linear regression was used in cross-sectional analyses of the control group to determine if total dietary fiber was a statistically significant predictor of fasting and two hour insulin levels following an OGTT. Standardized residuals had a skewed distribution with 10 values greater than three, indicating a poor fit when linear insulin was predicted. Consequently, insulin was transformed to its natural logarithm to obtain a better fit.

When the logarithm of insulin was regressed on a set of independent variables, it was of interest to be able to interpret the coefficients. The coefficient (b_i) represents a change in the log of the dependent variable per unit change in the corresponding independent variable, holding the other independent variables constant. The change in the logs is equivalent to the log of the ratio of the two insulin values,

$$\ln(I_2) - \ln(I_1) = \ln(I_2/I_1) = b_{fiber} \times (fiber_2 - fiber_1)$$

and

$$I_2/I_1 = e^{((fiber_2 - fiber_1) \times b_{fiber})},$$

where I_1 and I_2 equal the predicted insulin levels before and after a change in dietary fiber intake. It follows that the percent change in plasma insulin concentration following a 10 gram increase in total dietary fiber intake is

$$((I_2 - I_1)/I_1) \times 100 = (I_2/I_1 - 1) \times 100 = (e^{(10 \times b_{fiber})} - 1) \times 100.$$

The statistical package SAS was used on a VAX/VMS computer for descriptive analyses. The Generalised Linear Interactive Modelling (GLIM) (147) statistical package on an IBM-PC was used to carry out the logistic and linear regression analyses. The coefficients in the logistic regression were corrected for sampling induced bias by the use of "offset" in GLIM. P-values from normal deviates and chi square were calculated using programs by Rothman and Boice (148) on a Hewlett-Packard 41-CV calculator.

CHAPTER 3

Results

Summary

The results are presented in three sections. The first section includes findings from the comparison of reported prior dietary fiber intake among previously known cases of NIDDM (i.e. prevalent cases) and nondiabetic controls. In the second section, current dietary fiber intakes among 30 "controls" who were diagnosed with diabetes by OGTT during the SLVDS clinic (i.e. previously undiagnosed cases) were compared with normal controls. These results, while limited by small numbers, were not subject to diagnosis related bias (e.g. recall was expected to be the same for cases and controls and cases would not have changed their diet due to diagnosis). In addition, other dietary factors which may act as confounders (e.g. carbohydrate and total calories) were measured for current diet but not for prior diet. The third section presents cross-sectional relationships between current total dietary fiber intake and fasting and two hour plasma insulin concentrations following an OGTT among the controls.

Case-control Analysis of Prevalent Cases

Table 3.1 describes the demographic characteristics of diabetics who were confirmed by OGTT to have NIDDM and controls diagnosed with impaired and normal glucose tolerance. Reference to "nondiabetic" controls in the following text includes both IGT and normal glucose tolerance controls. "Normal" controls excludes persons with IGT. Because controls were frequency matched

Table 3.1. Previously known cases and nondiabetic controls by age, sex, ethnic group, body mass index, and prior FFQ total dietary fiber intake

	DIABETICS		CONTROLS					
	Prevalent Cases		Impaired Glucose Tolerance		Normal Glucose Tolerance		Total "Nondiabetic"	
	N	%	N	%	N	%	N	%
Age group								
20-29	3	1.2	0	0.0	29	6.3	29	5.4
30-39	12	5.0	5	6.1	43	9.3	48	8.9
40-49	32	13.2	11	13.4	88	19.1	99	18.3
50-59	81	33.5	28	34.1	154	33.5	182	33.6
60-69	77	31.8	27	32.9	113	24.6	140	25.8
70-74	37	15.3	11	13.4	33	7.2	44	8.1
Sex								
male	110	45.5	31	37.8	206	44.8	237	43.7
female	132	54.5	51	62.2	254	55.2	305	56.3
Ethnic Group								
Hispanic	158	65.3	46	56.1	170	37	216	39.9
Anglo	84	34.7	36	43.9	290	63	326	60.1
Body Mass Index								
< 25	48	19.8	22	26.8	235	51.1	257	47.4
25-29	111	45.9	31	37.8	158	34.3	189	34.9
30-34	59	24.4	19	23.2	52	11.3	71	13.1
35 +	24	9.9	10	12.2	15	3.3	25	4.6
Prior FFQ Total Dietary Fiber (gms/day)								
> 30	17	7.0	7	8.5	17	3.7	24	4.4
20-29	52	21.5	10	12.2	79	17.2	89	16.4
10-19	132	54.5	44	53.7	264	57.4	308	56.8
< 10	41	16.9	21	25.6	100	21.7	121	22.3
Total	242	100.0	82	100.0	460	100.0	542	100.0

to the cases by age, sex, and ethnicity, the distributions in Table 3.1 do not reflect real differences between cases and controls, and are intended only to show the number of subjects by subgroup who were included in the case-control analyses of previously known diabetics.

Odds ratios and confidence limits of known risk factors for NIDDM are presented in Table 3.2. Age, sex, ethnic group, BMI, waist to hip ratio, and

Table 3.2. Case-Control Analysis of Prevalent Cases: Risk factors for NIDDM among selected subgroups[1]

Risk Factor	Unit Change	Odds Ratio[2]	95% Confidence Interval	P-value Overall[3]
All previously known cases (N=218) and nondiabetic controls (N=515)				
Body mass index	10	3.82	(2.63,5.56)	< 0.0001
Waist to hip ratio	.1	1.61	(1.18,2.19)	0.0023
Parental history of diabetes	yes versus no	2.51	(1.71,3.69)	< 0.0001
Cases of less than 5 years duration (N=85) and nondiabetic controls (N=515)				
Body mass index	10	4.14	(2.51,6.83)	< 0.0001
Waist to hip ratio	.1	1.66	(1.10,2.50)	0.0195
Parental history of diabetes	yes versus no	2.18	(1.28,3.72)	0.0047
Cases of less than 5 years duration (N=85) and normal[4] controls (N=438)				
Body mass index	10	6.48	(3.61,11.65)	< 0.0001
Waist to hip ratio	.1	1.77	(1.15,2.72)	0.0105
Parental history of diabetes	yes versus no	2.35	(1.33,4.17)	0.0038

[1]50 subjects missing parental history of diabetes and one subject missing waist to hip ratio were omitted from the results in this table.
[2]Calculated from the logistic model that included six age groups, sex, two ethnic groups, continuous body mass index, continuous waist to hip ratio, parental history of diabetes, and an offset for the sampling fractions by age, sex, ethnic group and county.
[3]Calculated from the likelihood ratio test with and without the variable in the model.
[4]Excludes controls with impaired glucose tolerance.

parental history of diabetes were included in all of these analyses. The magnitude of the associations for BMI, waist to hip ratio, and parental history of diabetes are consistent with previous studies (17,20,21,22,39,42). When the study groups were restricted to diabetics who reported that they had been

diagnosed in the five years prior to clinic attendance and controls who tested normal by OGTT, the association of BMI with NIDDM was strengthened. Prior studies have shown that BMI is a risk factor for IGT (19,149) and, consequently, removing persons with IGT from the control group would be expected to increase the odds ratio for BMI with respect to NIDDM. The relationship between NIDDM and waist to hip ratio and parental history of diabetes remained unchanged when the study groups were restricted. Tables 3.3, 3.4, and 3.5 discussed below include all prevalent diabetics and nondiabetic controls.

Diabetics tended to consume more fiber than controls (Table 3.1). Of the known risk factors presented in Table 3.2, only BMI was hypothesized as a potential confounder in the relationship between dietary fiber and NIDDM. The odds ratio for the SQTDF score when BMI was included in the model was 0.80 per 10 gram decrease in fiber intake compared to 0.84 when BMI was not included in the model, suggesting only minimal confounding. Age, sex, and ethnic group were potential confounders used as variables for frequency matching in the selection of controls and were included as covariables in the logistic regression analyses. Because differences in prevalence by ethnic group could be due to dietary differences in the two groups, the model was run without ethnicity to see whether the inclusion of ethnicity could be obscuring the relationship between dietary fiber and NIDDM. The odds ratio for the SQTDF score changed from 0.80 per 10 gram decrease in fiber intake to 0.78 when ethnicity was removed from the model, suggesting that the inclusion of ethnicity was not obscuring a true fiber effect. The complete models used to calculate the odds ratios in the following tables are described in the table footnotes.

Ethnicity, parental history of diabetes, BMI, and waist to hip ratio were hypothesized as potential effect modifiers in the relationship between fiber and NIDDM. Table 3.3, column 1, presents the estimated excess risk of NIDDM associated with each of these variables by levels of fiber intake. For the same variables, column 2 presents the excess risk of NIDDM associated with low fiber intake. The tendency for higher fiber consumption among cases was present in Hispanics and Anglos, whether or not there was a parental history of diabetes, and at all levels of BMI. Waist to hip ratio showed small, apparently random, fluctuations of the odds ratios around unity.

Table 3.4 presents odds ratios and confidence limits for the SQTDF score by a priori categories, quartiles, and as a continuous variable which assumes an exponential increase in the odds of disease. To the extent that the SQTDF score accurately reflects the gram intake of total dietary fiber per day, the a priori categories were chosen to separate high and low levels of intake relative to estimated total dietary fiber intake for the general population (81,82,150). In all three models of SQTDF, the magnitude of the odds ratios suggested that high fiber intake was a risk factor for NIDDM. This association was in the opposite direction as what was hypothesized. The likelihood ratio test for trend was statistically significant and AIC indicated that fiber as a continuous variable gave a better fit than fiber as a categorical variable. These results suggest a dose response relationship where increasing dietary fiber intake is associated with an increased risk of having NIDDM.

The odds ratios and confidence limits for the three frequency-based dietary fiber scores and the three semi-quantitative scores are presented in Table 3.5. The odds ratios for each of the six scores suggest, as before, that high fiber intake is associated with NIDDM; however, the results of the overall

Table 3.3. Case-Control Analysis of Prevalent Cases (N=733)[1]: Variations in Prior FFQ total dietary fiber associations with NIDDM over different levels of ethnic group, parental history of diabetes, body mass index, and waist to hip ratio

	Odds Ratios[2]							
	(1)				(2)			
Ethnic Group	Fiber (gms/day)				Fiber (gms/day)			
	> 30	20-29	10-19	< 10	> 30	20-29	10-19	< 10
ANGLO	1.00	1.00	1.00	1.00	1.00	0.61	0.63	0.78
HISPANIC	2.30	2.29	1.17	0.51	1.00	1.39	0.74	0.41
Difference in scaled deviance = 6.1 with 3 degrees of freedom, p = 0.107[3]								
Parental History of Diabetes	Fiber (gms/day)				Fiber (gms/day)			
	> 30	20-29	10-19	< 10	> 30	20-29	10-19	< 10
NO	1.00	1.00	1.00	1.00	1.00	0.47	0.84	0.50
YES	2.35	4.05	2.06	2.55	1.00	0.80	0.73	0.54
Difference in scaled deviance = 1.8 with 3 degrees of freedom, p = 0.615								
Body Mass Index	Fiber (gms/day)				Fiber (gms/day)			
	> 30	20-29	10-19	< 10	> 30	20-29	10-19	< 10
< 25	1.00	1.00	1.00	1.00	1.00	1.20	0.60	0.28
25-34	3.36	1.82	3.25	6.48	1.00	0.65	0.58	0.53
35 +	ND[4]	4.42	8.12	11.89	ND	1.00	0.92	0.62
Difference in scaled deviance = 9.8 with 6 degrees of freedom, p = 0.133								
Waist to Hip Ratio	Fiber (gms/day)				Fiber (gms/day)			
	> 30	20-29	10-19	< 10	> 30	20-29	10-19	< 10
LOW	1.00	1.00	1.00	1.00	1.00	1.62	1.26	1.17
2	5.75	1.55	1.63	1.76	1.00	0.44	0.36	0.36
3	2.19	2.58	1.95	1.59	1.00	1.90	1.12	0.84
HIGH	5.99	2.78	2.80	1.91	1.00	0.75	0.59	0.37
Difference in scaled deviance = 2.6 with 9 degrees of freedom, p = 0.978								

[1]Excluding 50 persons missing parental history of diabetes and 1 person missing waist to hip ratio.
[2]Calculated from the logistic model that included six age groups, sex, ethnic group, body mass index, waist to hip ratio, parental history of diabetes, the fiber score, the interaction term as indicated and an offset for the sampling fractions by age, sex, ethnic group, and county
[3]Significance of interaction term.
[4]ND = insufficient number of observations (2 controls and 0 cases)

Table 3.4. Case-Control Analysis of Prevalent Cases: Semiquantitative total dietary fiber score by a priori categories, quartiles, and as a continuous variable

	Number of Cases	Number of Controls	Odds Ratios[1]	95% Confidence Interval	P-values Overall[2]	P-values Trend[3]
A Priori Categories (gms/day): AIC[4] = 848.69						
30 +	17	24	1.00			
20-29	52	89	0.99	(0.46,2.16)		
10-19	132	308	0.68	(0.33,1.39)		
< 10	41	121	0.54	(0.25,1.19)	0.12	0.02
Quartiles[5]: AIC = 848.18						
1 high fiber	79	135	1.00			
2	67	136	0.78	(0.50,1.22)		
3	45	135	0.55	(0.34,0.90)		
4 low fiber	51	136	0.68	(0.42,1.09)	0.10	0.04
Continuous: AIC = 845.77						
per 10 gram decrease in fiber intake	242	542	0.80	(0.65,0.98)	0.03[6]	

[1]Calculated from the logistic model that included the fiber score, six age groups, sex, two ethnic groups, continuous body mass index, and an offset for the sampling fractions by age, sex, ethnic group and county.
[2]Calculated from the likelihood ratio test for the logistic regression model with and without the factored variable.
[3]Calculated from the likelihood ratio test where categorized variables were treated as continuous.
[4]Akaike's Information Criterion
[5]Quartiles based on frequency of consumption in the controls.
[6]Calculated from the likelihood ratio test with and without the continuous variable.

likelihood ratio tests were compatible with there being no true association. Because soluble fiber has been more highly associated with blood glucose and insulin in previous short term studies, it was hypothesized here that the frequency-based fruits and vegetables score and the semi-quantitative soluble fiber score would be more strongly related to NIDDM than the fruits, vegetables, and cereals score and the insoluble fiber score, respectively.

Table 3.5. Case-Control Analysis of Prevalent Cases: Distribution of cases and controls according to dietary fiber scores

	Percent Cases (N=242)	Percent Controls (N=542)	Odds Ratios[1]	95% Confidence Interval	P-values Overall[2]	Trend[3]
FREQUENCY-BASED DIETARY FIBER SCORES						
Fruits and vegetables[4]						
1 high	33.5	25.1	1.00			
2	26.5	25.5	0.70	(0.45,1.10)		
3	19.8	24.9	0.62	(0.39,1.01)		
4 low	20.2	24.5	0.63	(0.39,1.01)	0.14	0.04
Legumes[4]						
1 high	33.1	27.3	1.00			
2	24.0	23.2	0.87	(0.56,1.37)		
3	21.9	23.4	0.88	(0.55,1.40)		
4 low	20.2	26.0	0.82	(0.51,1.31)	0.85	0.43
Fruits, vegetables, and cereals[4]						
1 high	34.7	25.1	1.00			
2	25.6	24.9	0.73	(0.47,1.14)		
3	21.9	25.1	0.62	(0.39,0.99)		
4 low	17.8	24.9	0.62	(0.38,1.01)	0.14	0.03
SEMI-QUANTITATIVE DIETARY FIBER SCORES						
Total dietary fiber (gms/day)						
30 +	7.0	4.4	1.00			
20-29	21.5	16.4	0.99	(0.46,2.16)		
10-19	54.6	56.8	0.68	(0.33,1.39)		
< 10	16.9	22.3	0.54	(0.25,1.19)	0.12	0.02
Insoluble fiber[4]						
1 high	29.8	24.9	1.00			
2	29.8	25.3	0.88	(0.56,1.37)		
3	19.0	24.9	0.65	(0.40,1.05)		
4 low	21.5	24.9	0.73	(0.45,1.17)	0.29	0.10
Soluble fiber[4]						
1 high	32.2	24.9	1.00			
2	24.8	25.3	0.68	(0.43,1.07)		
3	21.9	24.9	0.71	(0.45,1.14)		
4 low	21.1	24.9	0.69	(0.43,1.10)	0.27	0.14

[1]Calculated from the logistic model that included the fiber score, six age groups, sex, two ethnic groups, continuous body mass index, and an offset for the sampling fractions by age, sex, ethnic group and county.
[2]Calculated from the likelihood ratio test for the logistic regression model with and without the factored variable.
[3]Calculated from the likelihood ratio test where categorized variables were treated as continuous.
[4]Quartiles based on frequency of consumption in the controls.

However, the case-control differences in intake of both soluble and insoluble fiber were similar.

Subsets of cases and controls, where the relationship between dietary fiber and NIDDM might be expected to differ, are presented in Table 3.6. It was hypothesized that dietary differences in this population would be more extreme in the winter months when access to fruits and vegetables is limited. If this were true, dietary interviews taken in the winter would be expected to separate more clearly people with low and high average fiber intakes and the true relationship between fiber and NIDDM would be more apparent. No important differences were seen by season in these data.

Short duration cases were expected to have more accurate recall of their diet prior to diagnosis than cases of long duration. When the case group was restricted to persons with a disease duration less than five years (Table 3.6), the odds ratio for fiber approached 1.0, suggesting no relationship between dietary fiber and NIDDM.

The last two analyses in Table 3.6 excluded persons with IGT from the control group. If dietary fiber were a risk factor for IGT then the inclusion of the controls with IGT in the control group would be expected to bias the odds ratio for fiber and NIDDM toward 1.0. Exclusion of subjects with IGT did not affect the odds ratio relating fiber intake and NIDDM.

Case-Control Analysis of Previously Undiagnosed Cases

Thirty control subjects were first diagnosed with NIDDM from the results of the OGTT performed during the SLVDS clinic. The demographic characteristics of these subjects are shown in Table 3.7. The previously undiagnosed diabetics were older and heavier on average, more often female,

Table 3.6. Case-Control Analysis of Prevalent Cases: Odds ratios for prior FFQ total dietary fiber[1] among selected subgroups

	Number of Cases	Number of Controls	Odds Ratios[1]	95% Confidence Interval	P-value Overall[2]
Season of Interview[4]					
Winter	55	167	0.81	(0.50,1.32)	0.39
Spring	91	115	0.77	(0.53,1.12)	0.17
Summer	56	155	0.82	(0.55,1.24)	0.35
Fall	40	105	0.94	(0.56,1.59)	0.82
All prevalent cases and nondiabetic controls					
	242	542	0.80	(0.65,0.98)	0.03
Cases of duration < 5 years and nondiabetic controls[5]					
	83	282	0.98	(0.66,1.46)	0.92
All prevalent cases and normal[6] controls					
	242	460	0.76	(0.60,0.92)	0.02
Cases of duration < 5 years and normal[6] controls[5]					
	83	234	1.03	(0.69,1.56)	0.86

[1]Estimated odds ratio for a decrease of 10 grams per day in total dietary fiber intake.
[2]Calculated from the logistic model that included the fiber score, six age groups, sex, two ethnic groups, continuous body mass index, and an offset for the sampling fractions by age, sex, ethnic group, and county.
[3]Calculated from the likelihood ratio test for the logistic regression model with and without the continuous variable.
[4]Winter=January-March, Spring=April-June, Summer=July-September, Fall=October-December.
[5]Excludes controls queried on diet more than 5 years prior to interview.
[6]Excludes controls diagnosed with IGT.

and more often Hispanic, when compared to the normal controls.

Table 3.8 presents odds ratios and confidence limits for known risk factors. The excess of NIDDM at higher BMIs is consistent with the results of the case-control analysis of prevalent cases. Waist to hip ratio and parental

Table 3.7. Control subjects by glucose tolerance, age, sex, ethnic group, body mass index, and current FFQ total dietary fiber

	Previously Undiagnosed Diabetics		Impaired Glucose Tolerance		Normal Glucose Tolerance		Total Controls	
	N	%	N	%	N	%	N	%
Age group								
20-29	0	0.0	0	0.0	30	6.1	30	4.9
30-39	0	0.0	5	5.6	45	9.2	50	8.2
40-49	1	3.3	11	12.4	94	19.3	106	17.5
50-59	10	33.3	29	32.6	159	32.6	198	32.6
60-69	12	40.0	32	36.0	122	25.0	166	27.3
70-74	7	23.3	12	13.5	38	7.8	57	9.4
Sex								
male	9	30.0	32	36.0	219	44.9	260	42.8
female	21	70.0	57	64.0	269	55.1	347	57.2
Ethnic Group								
Hispanic	17	56.7	48	53.9	189	38.7	254	41.8
Anglo	13	43.3	41	46.1	299	61.3	353	58.2
Body Mass Index								
< 25	8	26.7	23	25.8	248	50.8	279	46.0
25-29	12	40.0	34	38.2	170	34.8	216	35.6
30-34	9	30.0	22	24.7	54	11.1	85	14.0
35 +	1	3.3	10	11.2	16	3.3	27	4.4
Current FFQ Total Dietary Fiber (gms/day)								
> 30	2	6.7	5	5.6	15	3.1	22	3.6
20-29	7	23.3	13	14.6	81	16.6	101	16.6
10-19	10	33.3	44	49.4	281	57.6	335	55.2
< 10	11	36.7	27	30.3	111	22.7	149	24.5
Total	30	100.0	89	100.0	488	100.0	607	100.0

history show no association with NIDDM in this group. In the Whitehall study (18) of 18,882 male civil servants in London, family history of diabetes was less common in previously undiagnosed diabetics than in known diabetics (prevalence ratio = 1.6 versus 2.9, respectively, when compared to normal glucose tolerance subjects). It is possible that undiagnosed diabetics have a different risk for NIDDM associated with waist to hip ratio and family history of diabetes than

Table 3.8. Case-Control Analysis of Previously Undiagnosed Cases: Risk factors for NIDDM among selected subgroups[1]

Risk Factor	Unit Change	Odds Ratio[2]	95% Confidence Interval	P-value Overall[3]
All incident cases (N=28) and nondiabetic controls (N=547)				
Body mass index	10	3.01	(1.42,6.41)	0.0049
Waist to hip ratio	.1	0.98	(0.49,1.97)	0.92
Parental history of diabetes	yes versus no	0.86	(0.32,2.29)	0.75
All incident cases (N=28) and normal[4] controls (N=464)				
Body mass index	10	4.46	(1.94,10.28)	0.0004
Waist to hip ratio	.1	1.07	(0.51,2.24)	0.86
Parental history of diabetes	yes versus no	0.97	(0.35,2.71)	0.92

[1]30 subjects missing parental history of diabetes and two subjects missing waist to hip ratio were omitted from the results in this table.
[2]Calculated from the logistic model that included six age groups, sex, two ethnic groups, continuous body mass index, continuous waist to hip ratio, parental history of diabetes.
[3]Calculated from the likelihood ratio test with and without the variable in the model.
[4]Excludes controls with impaired glucose tolerance.

medically diagnosed diabetics in the same community. Undiagnosed diabetics may have a milder form of disease or more slowly progressive deterioration, associated with different risk factors. In addition, people with a family history of diabetes may be tested more frequently for diabetes and, consequently, people without a family history of diabetes would be more likely to remain undiagnosed. The lack of association with these variables could also be due to small numbers.

The second half of Table 3.8 compares incident cases with normal controls, excluding controls with IGT. As in the case-control analysis of prevalent cases,

the excess risk associated with BMI was higher when persons with IGT were excluded from the control group. Including the additional 89 controls diagnosed with IGT with the 488 normal controls was expected to give very little increase in the statistical power to detect differences when comparisons were made with the 30 cases. Consequently, the analyses in Tables 3.9 through 3.11 exclude persons with IGT.

Table 3.9 presents odds ratios and confidence limits for the FFQ current semi-quantitative total dietary fiber score and for total dietary fiber from the 24-hour diet recall by a priori categories, quartiles and as continuous variables. The direction and magnitude of the odds ratios varies depending on dietary assessment method and on how fiber was entered into the model.

The odds ratios for the six FFQ fiber scores shown in Table 3.10 were generally at or below 1.0. This is consistent with the findings across scores from the case-control analysis of prevalent cases. Table 3.11 presents similar data from the 24-hour diet recall. Here, the odds ratios for all three scores do suggest an excess risk in the lowest fiber category.

The mean intakes from the 24-hour diet recall for total dietary fiber, carbohydrate, and fat are shown in Table 3.12 for previously undiagnosed diabetics, IGTs, and normal glucose tolerance controls. Dietary fiber and carbohydrate intake was higher on average in the normal controls compared to IGTs and previously undiagnosed diabetics. The percent of calories derived from carbohydrate, as opposed to fat, was also higher in the normal controls.

It was of interest to determine if the relationship between high fiber intake and NIDDM, observed in the case-control analysis of prevalent cases, could be explained by total calories and to look at the association between fiber and NIDDM while holding carbohydrate intake constant. Table 3.13 presents

Table 3.9. Case-Control Analysis of Previously Undiagnosed Cases: FFQ and 24-hour diet recall total dietary fiber by a priori categories, quartiles, and as continuous variables

Fiber Score	Percent Cases (N=30)	Percent Controls (N=488)	Odds Ratios[1]	95% Confidence Interval	P-values Overall[2]	P-values Trend[3]
FFQ SEMI-QUANTITATIVE TOTAL DIETARY FIBER SCORE						
A Priori Categories (gms/day): AIC[4] = 205.24						
30 +	6.7	3.1	1.00			
20-29	23.3	16.6	0.63	(0.10,3.76)		
10-19	33.3	57.6	0.26	(0.05,1.45)		
< 10	36.7	22.7	0.80	(0.14,4.48)	0.08	0.92
Quartiles[5]: AIC = 206.16						
1 high	33.3	25.0	1.00			
2	13.3	25.2	0.44	(0.13,1.37)		
3	13.3	25.0	0.39	(0.11,1.37)		
4 low	40.0	24.8	1.28	(0.49,3.34)	0.11	0.64
Continuous per 10 gram decrease in fiber intake: AIC = 208.09						
	100.0	100.0	1.04	(0.59,1.84)		0.89[6]
24-HOUR DIET RECALL TOTAL DIETARY FIBER						
A Priori Categories (gms/day): AIC = 208.48						
30 +	6.7	10.9	1.00			
20-29	13.3	18.6	0.96	(0.16,5.76)		
10-19	26.7	38.3	0.76	(0.15,3.96)		
< 10	53.3	32.2	1.80	(0.37,8.81)	0.30	0.20
Quartiles[5]: AIC = 206.94						
1 high	13.3	25.0	1.00			
2	23.3	25.0	1.49	(0.41,5.45)		
3	16.7	25.0	0.77	(0.19,3.13)		
4 low	46.7	25.0	2.45	(0.73,8.25)	0.16	0.17
Continuous per 10 gram decrease in fiber intake: AIC = 206.47						
	100.0	100.0	1.33	(0.84,2.11)		0.20[6]

[1]Calculated from the logistic model that included the fiber score, six age groups, sex, two ethnic groups, and continuous body mass index.
[2]Calculated from the likelihood ratio test for the logistic regression model with and without the factored variable.
[3]Calculated from the likelihood ratio test where categorized variables were treated as continuous.
[4]Akaike's Information Criterion
[5]Quartiles based on frequency of consumption in the controls.
[6]Calculated from the likelihood ratio test with and without the continuous variable.

Table 3.10. Case-Control Analysis of Previously Undiagnosed Cases: Distribution of cases and controls according to FFQ dietary fiber scores

Fiber Score	Percent Cases (N=30)	Percent Controls (N=488)	Odds Ratios[1]	95% Confidence Interval	P-values Overall[2]	P-values Trend[3]
FREQUENCY-BASED DIETARY FIBER SCORES						
Fruits and vegetables[4]						
1 high	23.3	25.2	1.00			
2	16.7	24.8	0.44	(0.13,1.51)		
3	16.7	25.0	0.39	(0.11,1.37)		
4 low	43.3	25.0	1.28	(0.49,3.34)	0.38	0.19
Legumes[4]						
1 high	43.3	35.9	1.00			
2	20.0	15.0	1.45	(0.49,4.28)		
3	16.7	33.6	0.41	(0.14,1.23)		
4 low	20.0	15.6	0.72	(0.23,2.29)	0.20	0.21
Fruits, vegetables, and cereals[4]						
1 high	23.3	25.0	1.00			
2	23.3	25.0	0.90	(0.29,2.84)		
3	20.0	25.0	0.86	(0.26,2.81)		
4 low	33.3	25.0	1.70	(0.58,4.97)	0.58	0.34
SEMI-QUANTITATIVE DIETARY FIBER SCORES						
Total dietary fiber (gms/day)						
30 +	6.7	3.1	1.00			
20-29	23.3	16.6	0.63	(0.10,3.76)		
10-19	33.3	57.6	0.26	(0.05,1.45)		
< 10	36.7	22.7	0.80	(0.14,4.48)	0.08	0.92
Insoluble fiber[4]						
1 high	33.3	25.2	1.00			
2	13.3	24.8	0.41	(0.12,1.40)		
3	13.3	25.2	0.44	(0.13,1.52)		
4 low	40.0	24.8	1.29	(0.49,3.35)	0.12	0.56
Soluble fiber[4]						
1 high	36.7	25.0	1.00			
2	10.0	25.0	0.31	(0.08,1.20)		
3	16.7	25.0	0.47	(0.15,1.48)		
4 low	36.7	25.0	1.05	(0.41,2.73)	0.14	0.86

[1]Calculated from the logistic model that included the fiber score, six age groups, sex, two ethnic groups, and continuous body mass index.
[2]Calculated from the likelihood ratio test for the logistic regression model with and without the factored variable.
[3]Calculated from the likelihood ratio test where categorized variables were treated as continuous.
[4]Quartiles based on frequency of consumption in the controls.

Table 3.11. Case-Control Analysis of Previously Undiagnosed Cases: Distribution of cases and controls according to 24-hour diet recall dietary fiber (gms/day)

Fiber Score	Percent Cases (N=30)	Percent Controls (N=488)	Odds Ratios[1]	95% Confidence Interval	P-values Overall[2]	Trend[3]
24 HOUR DIET RECALL						
Total dietary fiber (gms/day)						
30 +	6.7	10.9	1.00			
20-29	13.3	18.6	0.96	(0.16,5.76)		
10-19	26.7	38.3	0.76	(0.15,3.96)		
< 10	53.3	32.2	1.80	(0.37,8.81)	0.30	0.20
Insoluble fiber[4]						
1 high	6.7	25.2	1.00			
2	30.0	24.8	3.31	(0.68,16.1)		
3	13.3	25.2	1.31	(0.22,7.66)		
4 low	50.0	24.8	5.02	(1.06,23.9)	0.03	0.06
Soluble fiber[4]						
1 high	20.0	25.2	1.00			
2	13.3	24.8	0.57	(0.15,2.20)		
3	20.0	25.0	0.76	(0.23,2.52)		
4 low	46.7	25.0	1.94	(0.67,5.61)	0.14	0.08

[1]Calculated from the logistic model that included the fiber score, six age groups, sex, two ethnic groups, and continuous body mass index.
[2]Calculated from the likelihood ratio test for the logistic regression model with and without the factored variable.
[3]Calculated from the likelihood ratio test where categorized variables were treated as continuous.
[4]Quartiles based on frequency of consumption in the controls.

results from the 24-hour diet recall data where other potential dietary risk factors for NIDDM have been included in the analysis. Adjusted odds ratios are presented for total dietary fiber, carbohydrate, and fat intake. It should be noted that the magnitude of the odds ratios depend on the unit change used for comparison. This is defined in the footnotes to Table 3.13. The odds ratios adjusted only for nondietary factors (age, sex, ethnic group, and BMI) are presented first. These are followed by odds ratios for the calorie adjusted nutrients (146). Since body size, physical activity, and metabolic efficiency

Table 3.12. Case-Control Analysis of Previously Undiagnosed Cases: Mean dietary intakes[1] of dietary fiber, carbohydrate, and fat by disease status

	DIABETIC (N=30)		IGT (N=89)[2]		NORMAL (N=488)	
	Mean	95% Confidence Interval	Mean	95% Confidence Interval	Mean	95% Confidence Interval
Total Dietary Fiber						
grams per day	12.1	(8.8,15.5)	15.3	(12.6,18.1)	16.4	(15.4,17.5)
Carbohydrate						
grams per day	162.5	(133.4,191.6)	184.1	(163.7,204.5)	217.7	(207.3,228.1)
% of calories	42.3	(37.8,46.7)	44.8	(42.3,47.3)	46.8	(45.8,47.8)
Fat						
grams per day	81.9	(61.3,102.4)	74.8	(65.8,83.8)	82.1	(77.7,86.4)
% of calories	42.6	(39.0,46.3)	39.8	(37.8,41.7)	38.2	(37.3,39.0)

[1]Estimates of dietary intake from the 24-hour diet recall.
[2]IGTs were excluded from the case-control analysis of previously undiagnosed cases, but included in the cross-sectional analyses which follow.

contribute to the variation in total caloric intake between persons, the calorie adjusted intakes better reflect the contribution of dietary factors independent of body size, physical activity, and metabolic efficiency. Odds ratio are also presented after adjustment for each of the other dietary factors.

Calorie adjusted fiber intake (OR = 1.61) was more highly associated with NIDDM than fiber intake unadjusted for calories (OR = 1.33). The odds ratio was reduced to 1.12 when carbohydrate was included in the model. None of the odds ratios for fiber was statistically significant. Low carbohydrate intake and high fat intake were both associated with NIDDM after adjusting for calories and fiber, but to a lesser extent when carbohydrate and fat were included in the model simultaneously.

Table 3.13. Case-Control Analysis of Previously Undiagnosed Cases (N=518): Odds ratios for 24-hour diet recall total dietary fiber, carbohydrate, and fat adjusted for other dietary factors

Subgroup	Odds Ratio[1]	95% Confidence Interval	P-values Overall[2]
Total dietary fiber[3]			
Fiber	1.33	(0.84,2.11)	0.20
$Fiber_{kcal}$	1.61	(0.94,2.77)	0.07
$Fiber_{kcal}$ ($Carb_{kcal}$)	1.12	(0.62,2.00)	0.71
$Fiber_{kcal}$ (Fat_{kcal})	1.26	(0.70,2.26)	0.42
$Fiber_{kcal}$ ($Carb_{kcal}$,Fat_{kcal})	1.17	(0.64,2.15)	0.87
Total carbohydrate[4]			
Carb	1.37	(0.84,2.23)	0.19
$Carb_{kcal}$	3.71	(1.59,8.63)	0.0015
$Carb_{kcal}$ ($Fiber_{kcal}$)	3.42	(1.33,8.79)	0.0086
$Carb_{kcal}$ (Fat_{kcal})	2.32	(0.52,10.4)	0.29
$Carb_{kcal}$ ($Fiber_{kcal}$,Fat_{kcal})	2.03	(0.43,9.66)	0.40
Total fat[5]			
Fat	1.07	(0.99,1.17)	0.09
Fat_{kcal}	1.43	(1.13,1.80)	0.0017
Fat_{kcal} ($Fiber_{kcal}$)	1.39	(1.08,1.39)	0.0075
Fat_{kcal} ($Carb_{kcal}$)	1.18	(0.79,1.77)	0.37
Fat_{kcal} ($Fiber_{kcal}$,$Carb_{kcal}$)	1.20	(0.80,1.78)	0.33

[1]Calculated from the logistic model that included six age groups, sex, two ethnic groups, continuous body mass index, and the nutrients as listed.
[2]Calculated from the likelihood ratio test for the logistic regression model with and without the continuous variable.
[3]estimated odds ratio for a decrease of 10 grams per day in total dietary fiber intake.
[4]estimated odds ratio for a decrease of 100 grams per day in total carbohydrate intake.
[5]estimated odds ratio for an increase of 10 grams per day in total fat intake.

Cross-sectional Analysis of Controls

The most consistent finding to emerge from the literature cited in Chapter 1 was an association between low fiber intake and high post-prandial concentrations of blood insulin. Chronically elevated concentrations of insulin in the blood have been shown to decrease the number of cellular insulin

receptors leading to decreased insulin sensitivity (128). It was hypothesized here that habitual high insulin levels following low fiber meals contribute to down regulation of insulin receptors, which compromises the body's ability to maintain glucose homeostasis. The total control group described in Table 3.7, excluding 21 subjects who were missing data on plasma insulin levels, were included in analyses to determine if total dietary fiber was an important predictor of fasting and two hour OGTT plasma insulin concentrations. The unadjusted mean plasma insulin levels are presented in Table 3.14 by levels of total dietary fiber intake from the 24-hour diet recall and by level of glucose tolerance. Mean plasma insulin levels are higher in the low fiber categories compared to the high fiber categories. Table 3.15 presents the predicted percent change in plasma insulin that would occur with an increase of 10 grams per day in fiber intake. These results are adjusted for age, sex, ethnicity, body mass index, total carbohydrate and calorie intake.

The p-values in Table 3.15 suggest that fiber is inversely associated with plasma insulin concentration while fasting and two hours after a glucose tolerance test. In the third series of models, fasting insulin was included as a predictor of two hour insulin in order to determine if the fiber effect was important in the insulin response to an OGTT or only as a predictor of fasting insulin. Fiber was not statistically significant when fasting insulin was included in the model. This suggests that fiber is an important predictor of fasting insulin and that its effect on the two hour insulin levels is a result of its effect on fasting levels.

The relationship between total dietary fiber and fasting plasma insulin was independent of calorie and carbohydrate intake as indicated by the results where these variables were included as covariables in the model. Including an

Table 3.14. Mean fasting and 2-hour OGTT plasma insulin concentration by level of total dietary fiber[1] intake and glucose tolerance

		Fasting Insulin (IU/liter)		2-Hour OGTT Insulin (IU/liter)	
	Number of Subjects	Mean	95% Confidence Interval	Mean	95% Confidence Interval
Previously Undiagnosed Diabetics (N=29)					
Total dietary fiber (gms/day)					
> 30	2	16.5	(-118.3,151.3)	77.5	(-650.5,805.5)
20-29	3	14.3	(-19.2,47.8)	32.0	(-48.5,112.5)
10-19	8	21.1	(15.4,26.8)	96.4	(45.4,147.3)
< 10	16	21.1	(13.8,28.3)	99.4	(62.3,136.6)
Impaired Glucose Tolerance (N=89)					
Total dietary fiber (gms/day)					
> 30	5	13.4	(7.1,19.7)	89.8	(73.8,105.8)
20-29	15	15.5	(8.6,22.5)	140.1	(43.9,236.2)
10-19	34	15.4	(12.6,18.3)	120.8	(92.6,149.0)
< 10	35	18.6	(14.8,22.4)	130.7	(107.1,154.3)
Normal Glucose Tolerance (N=468)					
Total dietary fiber (gms/day)					
> 30	52	11.5	(9.6,13.5)	45.9	(36.9,54.8)
20-29	90	10.6	(9.2,12.0)	52.3	(45.4,59.2)
10-19	181	11.8	(10.4,13.2)	55.8	(49.4,62.2)
< 10	145	13.7	(11.7,15.7)	62.3	(55.1,69.5)
All Controls (N=586)					
Total dietary fiber (gms/day)					
> 30	59	11.9	(10.0,13.7)	50.6	(41.8,59.5)
20-29	108	11.4	(9.9,12.9)	63.9	(49.7,78.2)
10-19	223	12.7	(11.4,14.0)	67.2	(59.7,74.6)
< 10	196	15.2	(13.4,16.9)	77.5	(69.5,85.6)

[1]Total dietary fiber from the 24-hour diet recall.

interaction term for BMI and fiber in the model suggested that the percent change in fasting insulin levels was greater in non-obese subjects than in obese subjects. It remains a question whether fiber intake during the previous 24-hours is acting as a longer term indicator of fiber intake in groups or if the prior day's diet is the important factor in predicting fasting plasma insulin concentration.

Table 3.15. Cross-sectional Analysis of Controls (N=586)[1]: Percent decrease in fasting and two hour OGTT plasma insulin concentrations associated with an increase of 10 grams per day in dietary fiber[2] intake

	Percent Change in Insulin (IU/l)	95% Confidence Interval	P-value[3]
Factors[4] included in the model to predict **FASTING PLASMA INSULIN**			
Fiber	-3.7	(-7.6,0.02)	0.06
Fiber + kcal[5]	-4.6	(-9.0,0.00)	0.05
Fiber + kcal[5] + carbohydrate(CHO)[5]	-5.3	(-10.1,-0.3)	0.04
Fiber + kcal[5] + CHO[5] + BMI*fiber[6]			
when BMI = 20:	-10.2	(-16.7,-3.2)	
BMI = 25:	-6.2	(-10.9,-1.1)	
BMI = 30:	-1.9	(-7.9,4.4)	0.02[7]
Factors[4] included in the model to predict **TWO HOUR PLASMA INSULIN**			
Fiber	-5.0	(-9.5,-0.4)	0.03
Fiber + kcal[5]	-5.9	(-11.0,-0.6)	0.03
Fiber + kcal[5] + CHO[5]	-4.8	(-10.4,1.2)	0.11
Fiber + kcal[5] + CHO[5] + BMI*fiber[5]			
when BMI = 20:	-8.6	(-16.4,-0.2)	
BMI = 25:	-5.5	(-11.1,0.4)	
BMI = 30:	-2.2	(-9.1,5.2)	0.13[7]
Factors[4] included in the model to predict **TWO HOUR PLASMA INSULIN** in the presence of fasting plasma insulin (FINS)			
Fiber + FINS	-2.9	(-6.9,1.2)	0.17
Fiber + FINS + kcal[5]	-3.3	(-7.9,1.5)	0.17
Fiber + FINS + kcal[5] + CHO[5]	-1.7	(-6.8,3.6)	0.52
Fiber + FINS + kcal[5] + CHO[5] + BMI*fiber[5]			
when BMI = 20:	-2.7	(-10.0,5.3)	
BMI = 25:	-1.9	(-7.1,3.6)	
BMI = 30:	-1.1	(-7.4,5.5)	0.77[7]

[1]Twenty one subjects missing insulin data were excluded from this analysis.
[2]Total dietary fiber from the 24-hour diet recall.
[3]P-value for the fiber coefficient in the linear regression model.
[4]Linear regression models included six age groups, sex, two ethnic groups, continuous body mass index, and the other factors as listed.
[5]$p > 0.15$; [6]$p = 0.056$
[7]P-value for fiber + bmi*fiber.

CHAPTER 4

Discussion

Limitations of the Present Study

Prevalent case-control design. A major limitation in the interpretation of the results of this study stems from the original design which compared all previously known (i.e. prevalent) cases of NIDDM to a population-based control group. Prevalent cases, in general, underrepresent more severe cases and overrepresent less severe cases. This induces a bias in the odds ratio if severity is also associated with the risk factor of interest. In this case, there are some data to suggest that low dietary fiber intake may be related to survival in people with NIDDM (28). If low dietary fiber intake is a prognostic risk factor in the population studied here, prevalent cases would be expected to have higher fiber intakes on average than incident cases and the odds ratio would be biased toward zero. This might partially explain the unexpected results that low fiber intake lowers rather than increases the risk of NIDDM.

Another problem with the inclusion of prevalent cases, considered more important in this study than survival, is that of obtaining accurate estimates of dietary fiber intake at the relevant time in the pathogenesis of NIDDM. Dietary changes unrelated to diabetes may have occurred in both diabetics and controls over time which would introduce nonselective misclassification into the fiber measure if current dietary intake was used as a surrogate measure for prior intake. Nonselective misclassification would bring the observed odds ratio relating fiber to NIDDM towards one, but not past that value. Also, dietary

changes resulting from diagnosis (i.e. knowledge of existing disease or dietary counseling resulting from diagnosis) are likely to have occurred in prevalent diabetics and not in controls. The recommended diabetic diet has changed over the last ten years. Higher carbohydrate diets and, more recently, increased dietary fiber intake have been recommended (151,152). Because of the long duration of some of the cases, the dietary beliefs in this group are expected to be mixed. Consequently, the effect that dietary changes resulting from diagnosis would have on the odds ratios is difficult to predict.

Several strategies were used in the design and analysis of this study to address these issues. First, subjects were asked about their diet at a defined time in the past. For the diabetics, the prior time was one year before their diagnosis of diabetes. Controls were assigned a prior time from a randomized list of disease durations plus one year determined from the medical records of the diabetic group. This was intended to measure diet at a time more relevant to the pathogenesis of disease in diabetics and prior to dietary changes resulting from diagnosis. However, this approach would not remove error due to poor recall in both groups and bias associated with selective recall in the diabetics compared to the controls. A number of studies have recently been published which evaluate the reliability of retrospective dietary recall (150,153,154,155,156, 157,158,159), and they agree that it is strongly influenced by current dietary habits. All of these studies, except the study by Jensen et al (157), concluded that retrospective assessment is a more reliable estimate of past diet than current diet. Potential bias associated with misclassification of dietary intake is discussed below.

Two strategies were used in the analysis to look for bias in the odds ratio relating fiber to NIDDM resulting from the inclusion of prevalent cases. First,

short duration diabetics were looked at separately (Table 3.5) because it was believed that their retrospective recall of diet would be more accurate. The odds ratio indicates no association (OR = 0.98, comparing low to high fiber intakes) between fiber and NIDDM when the analysis was limited to this group.

The second analytic strategy was to compare the 30 newly diagnosed diabetics from the control group with the normal controls. These 30 cases did not have a medical diagnosis of diabetes at the time of their clinic visit and dietary interview. Consequently, their recall was expected to be the same as the normal controls. The odds ratios relating fiber to NIDDM in the case-control analysis of previously undiagnosed cases (Table 3.9) were at or below 1.0 when the scores estimating current fiber intake from the FFQ were used. Because the same method was used to estimate fiber intake, these results are directly comparable to the results of the case-control analysis of prevalent cases. The odds ratios (Table 3.9) weakly support the association of high fiber intake and NIDDM seen with the prevalent cases. However, the results of the likelihood ratio tests are compatible with there being no true association.

Response. A strength of this study is the population-based design. Bias associated with the selection of study subjects is a problem that goes along with studies where randomization of subjects into exposure categories is not possible. There are many potential sources of selection bias which are of concern and difficult to measure when cases and controls are chosen from hospital patients or when controls are chosen from relatives or associations, like neighbors, of the cases. As a result, with respect to selection bias, the population-based design used here is theoretically preferred, though it is often logistically difficult. Assuming proper sampling of controls and complete case

ascertainment, the main source of selection bias in population-based studies is that due to the associated characteristics of nonrespondents.

In this study, the percent of subjects responding was 71.2 percent among the previously known diabetic cases and 64.0 percent among the controls. This level of response would lead to biased estimates of the odds ratios if the percent response differed depending on disease status and the exposure of interest (e.g. fiber). Unfortunately, no other source of information about the dietary fiber intake of nonresponders was available to evaluate this directly. Among diabetics, approximately equal proportions of the responders and nonresponders (Table 2.7) reported that they were on a special diabetic diet (.54 versus .58, respectively) and that diet was their only current therapy for diabetes (.10 versus .9, respectively). To the extent that current diet therapy is associated with general dietary habits and fiber intake, these findings suggest that nonresponse among diabetics was not associated with fiber intake. Similar data were not available for controls.

The data presented in Tables 2.5, 2.6, and 2.7 indicate that, in most cases, characteristics associated with nonrespondents are in the same direction and of similar magnitude among diabetics and controls. For example, even though the average level of education was higher in controls than diabetics (65.6% versus 47.3%, respectively, had at least 12 years of education) the nonresponders had lower levels of education on average than the responders in both diabetics and controls. This was true for age, Spanish origin, county, length of residence in the SLV, marital status, self assessment of health, ever smoked more than 100 cigarettes, alcohol use, and family history of diabetes. Exceptions included sex, number of physician visits in the last year, current reported weight, and history of hypertension.

Criqui (160) suggested calculating error terms for the odds ratios to help judge the magnitude of bias caused by differential nonresponse in a given data set. Odds ratio error terms are defined as (percent response cell A) x (percent response cell D) / (percent response cell B) x (percent response cell C), where A, B, C, and D represent subgroups defined in the following 2 x 2 table:

	Diseased	Non-Diseased
Exposed	A	B
Unexposed	C	D

Dividing the estimated crude odds ratio by the odds ratio error term gives the true crude odds ratio in the population being studied. Table 4.1 presents the odds ratio error terms for each of the characteristics in Table 2.7. The error terms are close to one, suggesting that, to the extent that these characteristics

Table 4.1 Odds ratio error terms for selected characteristics

Exposure[1]	Percent Response A	B	C	D	Odds ratio error term
Years of residence in SLV (20 +/< 5)	.730	.674	.833	.769	1.00
Years of education completed (> 12/< 8)	.833	.799	.513	.531	1.08
Marital status (divorced/married)	.684	.685	.779	.705	0.90
Self assessment of health (poor/excellent)	.662	.583	.800	.708	1.00
Number of physician visits in past year (4/0)	.713	.692	.829	.675	0.84
Current reported weight (200+ pounds/100-149 pounds)	.774	.803	.713	.710	0.96
History of: (yes/no)					
Hypertension	.779	.654	.692	.695	1.20
Smoking	.750	.687	.732	.676	1.01
Alcohol use	.795	.699	.656	.647	1.12
DM in family	.744	.725	.717	.656	0.94

[1]Comparison categories are in parentheses with the reference category listed last.

can act as indicators of other variables of interest (e.g. fiber), nonresponse is not expected to have altered the findings in this study.

Confounding. A limitation in the interpretation of the case-control analysis of prevalent cases results from the lack of data on total calorie and carbohydrate intake. The association between high dietary fiber intake and NIDDM seen when comparing the prevalent cases and nondiabetic controls may be a consequence of an association between high fiber and high calorie intake. Since total calorie intake is influenced by body weight and physical activity, a calorie adjusted measure of fiber is needed to better reflect fiber's contribution independent of these other factors. Because of the physicochemical properties of fiber described in chapter 1, it is also reasonable that the relative amounts of fiber and total calories would influence fiber's action in the gastrointestinal tract.

Twenty-four hour diet recalls were collected in addition to the FFQ on all subjects and provided a measure of current fiber and calorie intake. Because people's diets are highly variable from day-to-day, nutrient intakes from the 24-hour diet recall for an individual tend not to be an accurate measure of their average intake over a longer period of time. However, equal numbers of persons are expected to have high intakes and low intakes on the recall day relative to their mean intake and the day-to-day variability in this measure averages out when large numbers of subjects are studied. The 24-hour recall has several advantages over the FFQ. First, probing was not limited to selected foods but included all foods eaten in the previous 24 hours; the amount of each food eaten was determined; recall was only required for the most recent 24-hour period; and summaries for all nutrients available in the NCC nutrient

data base could be calculated (e.g. total dietary fiber, insoluble fiber, soluble fiber, total calories, carbohydrate and fat).

The mean fiber intakes from the 24-hour diet recall presented in Table 3.11 show an increasing gradient across diabetic, IGT, and normal controls. Estimates of the percent of IGTs going on to develop diabetes have ranged from 15 percent in 10 years in the Bedford (England) Study (19) to 38.5 percent in a seven year followup of Japanese subjects in Japan (161). In the same two studies, estimates of the percent of IGTs reverting to normal were 53 percent in ten years and 38.5 percent in seven years, respectively. Consequently, IGTs may be viewed as a mix of cases and controls and would be expected to have intermediate levels of intake. The gradient observed here supports the hypothesized relationship between low fiber intake and NIDDM.

Calorie-adjusted fiber intake, based on the 24-hour diet recall, was compared between the 30 previously undiagnosed diabetics and the normal controls to determine if adjusting for calories could account for the positive association between high fiber and NIDDM observed in the case-control analysis of prevalent cases. The results, presented in Table 3.12, show that the fiber effect is small (odds ratio = 1.12 per 10 gram decrease in fiber) when carbohydrate and calories are held constant. The likelihood ratio test is compatible with there being no true association. Carbohydrate and/or fat were more strongly related to NIDDM than dietary fiber in these data.

Misclassification. Misclassification of subjects by disease status was reduced by administering an OGTT to all subjects who were not currently being treated with insulin or oral hypoglycemic agents. As shown in Table 2.10, subjects were removed from both the case and control groups based on WHO criteria for diabetes and SLVDS criteria for NIDDM.

The BMI used in these analyses was based on current measured weight and height. Because weight loss has been documented to occur prior to diagnosis (7,19) and because weight loss is a recommended therapy for NIDDM, the preferred measure would have estimated weight five to ten years before diagnosis. Reported lifetime maximum weight and reported weight at age 20 years were collected on all participants. Reported lifetime maximum weight occurred as long as 31 years prior to diagnosis and 45 years after diagnosis. Because more than 43 percent of the lifetime maximum weights occurred after diagnosis, this measure was not used. BMI at age 20 was entered into a logistic regression analysis and results were compared with current BMI. Table 4.2 presents the odds ratios, confidence limits and likelihoods ratio tests for current BMI and BMI at age 20. Current BMI was linearly related to NIDDM across all BMI groups, whereas, the odds ratios relating BMI at age 20 years to NIDDM decreased in the 40 and higher BMI category. The association between current BMI and NIDDM was stronger at all levels of BMI and AIC indicated that current BMI was a better predictor of NIDDM than BMI at age 20 years. The strong linear trend in current BMI may suggest that weight loss following diagnosis is small relative to between person differences in BMI. The odds ratios relating fiber to NIDDM after adjusting for BMI at age 20 years were consistent with the odds ratios adjusted for current BMI. Current BMI, which was not subject to differential recall in cases and controls, was concluded to be a good surrogate for BMI prior to diagnosis.

A limitation of this study, which is common to case control studies examining dietary factors as exposures of etiologic interest, is the limited ability of existing dietary assessment methods to measure dietary intake in a meaningful way under the logistical constraints of a large epidemiological study.

A three or seven day food record or an extensive dietary history, which are common clinical tools for assessing dietary intake, were not feasible methods in this study.

Table 4.2. Case-Control Analysis of Prevalent Cases:
Excess risk associated with
current body mass index and body mass index at age 20 years

	CURRENT BMI			BMI AT 20 YEARS		
Factor	Odds Ratio[1]	95% Confidence Interval	p-value Overall[2]	Odds Ratio[1]	95% Confidence Interval	p-value Overall[2]
Body Mass Index (BMI) - Categorized						
< 20	1.00			1.00		
20-24	2.03	(0.67,6.14)		0.78	(0.51,1.19)	
25-29	5.19	(1.75,15.38)		1.72	(1.02,2.88)	
30-34	8.08	(2.64,24.75)		5.62	(2.25,14.01)	
35-39	10.61	(3.04,37.10)		12.99	(2.27,74.25)	
40 +	20.84	(3.73,116.38)	<0.0001	5.23	(0.43,64.19)	<0.0001
Total Dietary Fiber[3] (grams/day)						
> 30	1.00			1.00		
20-29	1.01	(0.47,2.19)		1.04	(0.48,2.25)	
10-19	0.68	(0.34,1.41)		0.81	(0.39,1.65)	
< 10	0.55	(0.25,1.22)	0.13	0.62	(0.29,1.36)	0.27
AIC	861.35			877.31		
Body Mass Index - Continuous						
per 10 unit change	3.80	(2.67,5.41)	<0.0001	1.62	(1.16,2.25)	0.003
Total Dietary Fiber[3] (grams/day)						
> 30	1.00			1.00		
20-29	0.99	(0.46,2.16)		1.03	(0.48,2.18)	
10-19	0.68	(0.33,1.39)		0.80	(0.40,1.61)	
< 10	0.54	(0.25,1.19)	0.12	0.66	(0.31,1.41)	0.38
AIC	848.69			900.40		

[1]Calculated from the logistic model that included six age groups, sex, ethnic group, total dietary fiber, BMI as indicated, and an offset for the sampling fractions by age, sex, ethnic group, and county.
[2]Calculated from the likelihood ratio test with and without the variable in the model.
[3]Prior total dietary fiber intake from the FFQ.

The food record or diary, kept by the study participant usually for three to seven days, measures current intake. It is not limited by memory for recall if consistently filled out near the time of a meal or snack. However, subjects may alter their diet as a result of recording their intake. The burden of data collection lies mainly on the subject. It requires literacy and motivation and random samples have been difficult to obtain because of low response rates (162,163). Less interview time may be needed, however, more than one patient contact is often required. The inability to assess diet prior to disease onset, the extra patient visit, and necessary literacy and motivation were major disadvantages of the food record method for the SLVDS.

The diet history developed by Burke and Stuart over some years (164,165) includes three parts. The first part is an extended 24-hour recall including both a measure of the prior 24 hours and a usual day, using the question "What do you usually have for breakfast?" coupled with "What did you have for breakfast this morning?" The second part, the "cross-check", is a list of specific foods and food groups. Given quantities for each food determined in part one, the nutritionist probes as to likes and dislikes, purchasing and use of each food in order to verify or clarify information given in the usual intake. The third part is a three day record kept by the subject and used as an additional means of checking the usual intake.

Many versions of the diet history have been used. The method seeks to measure long term "usual" diet. A total history takes more than an hour with a skilled dietician or nutritionist (166,162,165). It requires that the subject have a defined pattern of diet and knowledge or awareness of his usual eating habits (162). The length of time involved, the relatively unstructured format,

and requirement of administration by a skilled nutritionist were major drawbacks of the "Burke-type" diet history for the SLVDS.

The use of a FFQ of individual food items or of food groups has been increasingly used in field studies of diet and disease and was selected, along with the 24-hour diet recall, for measuring fiber intake for the SLVDS. The FFQ is generally more structured than a diet history, allowing self administration or more standardized administration by a less skilled interviewer. In addition, the FFQ format allowed comparison of current intake with intake at some earlier more appropriate time period with respect to the etiology of NIDDM. Review of published food frequency validations revealed that efforts to validate the FFQ method against other dietary methods has resulted in conflicting opinions on its usefulness. Several factors contribute to the confusion.

First, to determine the accuracy of a FFQ one must be able to measure the "true" intake parameters of interest. There is no absolute measure of dietary assessment against which to measure all others (162). This stems partly from the variability and intimacy of diet, as well as from the different aspects of diet measured by each method. The "Burke-type" history (167,168,169), the seven day estimated record (170,169), hemoglobin values (171), and intake patterns from other diet surveys (171,172) have been used as indicators of "true" intake in validations of FFQs.

Second, quantitative methods of comparing different assessment techniques and criteria for determining validity have varied. Marr (162) suggested that comparison can be made at four levels: (1) absolute agreement for individuals, (2) relative agreement (i.e. consistent differences across individuals), (3) absolute agreement of the mean intake of groups, and (4) relative agreement

across subgroups. In support of measuring relative agreement, Block (173) states that less precise methods which locate individuals on the distribution in broad categories of low, medium, and high intake still permit examination of nutritional hypotheses and the assessment of dose-response relationships. Other analytical departures have included: (1) classification of diet based on frequency of use of food items versus nutrient intake (171); and (2) employment of a variety of statistical methods to show agreement, or lack of agreement, between methods.

Perhaps of most significance in trying to evaluate the validation literature is that the FFQ is not a standardized method. Rather, it may refer to anything on a continuum that includes a questionnaire with 21 food items asking only frequency of consumption (174) to a comprehensive list of foods asking frequency of consumption, portion size, method of preparation, and any unusual recipes (Block's (173) reference to Morgan et al's (175) Burke-type history as a food frequency). The number of food items, whether they're individual or grouped, the information obtained on each food (i.e. frequency, portion size, preparation, seasonality), questionnaire administration, and the populations studied have varied. In many of the published validations reviewed there was insufficient information given on the instrument itself to determine the relevance of their results to the SLVDS (i.e. how many and which food items, what is asked on each item, and how food items were chosen to be included).

It is encouraging that some investigators have been able to observe statistically significant diet-disease associations of biologically plausible hypotheses, supported by other types of investigations, using the FFQ. Mettlin et al (174) used a list of 21 food items high in vitamin A, representing a "substantial sample of food ingestion", to investigate the potential interaction of

vitamin A and smoking in the occurrence of lung cancer. They were able to detect consistent and statistically significant associations using both the total 21 item food list and individual rich sources of vitamin A (i.e. carrots and milk). Using a FFQ of six fiber-rich food groups, Brender (176) observed a statistically significant inverse association between appendicitis and fiber intake in a case-control study of children in Seattle.

As a crude check on the validity of the SLVDS semi-quantitative scores, the current (not prior) FFQ fiber scores for total, insoluble, and soluble fiber intake were compared with corresponding fiber fractions from the 24-hour recall among controls (Table 4.3). As expected, the maximum intake was higher with the 24-hour recall which reflects day-to-day fluctuations in intake. The FFQ asked the subject to give an average intake for the last month which removed some of the within person variability and, consequently, had a narrower distribution. The absence of a sex difference in the FFQ is probably due to the use of common portion sizes for fiber content of foods across all subjects. Systematic differences by sex and ethnicity were accounted for in the analysis by adjustment for these factors.

Systematic differences in reporting between diabetics and controls could partially explain the unexpected finding that high fiber intake is related to NIDDM if diabetics overreported their fiber intake relative to the controls. Byers and colleagues (149) reported findings from a study of 323 noncancer control subjects who were interviewed about their diet between June of 1975 and June of 1979. The same subjects were reinterviewed in 1984-1985 about their current diet and their retrospective recall of their diet during the earlier time period. Ninety one of the subjects had developed heart disease, cancer, peptic ulcer, and/or diabetes subsequent to the first interview. The mean

Table 4.3. Total dietary fiber intake by sex and ethnicity: comparison of FFQ and 24-hour recall assessment methods among controls

	N	Food Frequency Questionnaire (gms/day) MEAN	SE	MAX	MIN	24-Hour Diet Recall (gms/day) MEAN	SE	MAX	MIN
Non-Hispanic									
Male	149	15.9	0.56	4.4	38.6	19.5	1.12	0.0	83.6
Female	204	15.3	0.51	2.3	46.2	14.9	0.64	1.8	67.5
Hispanic									
Male	111	14.3	0.71	2.7	53.9	19.1	1.45	0.4	93.7
Female	143	14.2	0.56	1.7	42.5	11.9	0.72	0.9	50.8
Total	607	15.0	0.29	1.7	53.9	16.1	0.48	0.0	93.7

N=number of subjects; MEAN=mean level of intake; SE=standard error; MIN=minimum reported fiber intake; MAX=maximum reported fiber intake.

reported monthly gram intake of dietary fiber in the 91 subjects with recent chronic disease compared to the 223 subjects without chronic disease was 658 versus 632 on the original interview, 788 versus 715 on the retrospective recall, and 716 versus 644 on the current dietary interview. Pearson's correlations of the original interview and the retrospective recall were 0.60 among those with recent chronic disease and 0.62 among those without recent chronic disease. These findings support the absence of differential recall by diseased persons for dietary fiber.

In this study, there was potential for error in determining the date of diagnosis and, consequently, in the time period queried for prior dietary intake. The prior time for query of dietary intake was determined from medical records in 82 percent of the cases. The remaining cases were asked their date of diagnosis at the beginning of the clinic visit. In addition, all subjects were asked their age at diagnosis in the medical history interview. Duration of

disease based on the medical history interview was compared with the number of years ago that diet was queried. Thirty-two percent of the prior diet times were subsequent to the reported age of diagnosis. The 68 percent of diabetics with a prior diet time before reported diagnosis were analyzed separately to determine if this discrepancy in reported age at diagnosis and period of diet recall had biased the findings. Table 4.4 presents the odds ratios and confidence limits which relate dietary fiber to NIDDM for all diabetics and for the subgroup where prior reporting time was before reported diagnosis age. The results do not suggest bias due to potential errors in assigning the prior time for diet query. The field staff noted that longer duration diabetics, in

Table 4.4. Case-Control Analysis of Prevalent Cases: Comparison of odds ratios relating total dietary fiber and NIDDM for all diabetics and diabetics with prior diet time before reported diagnosis age

	Number of Cases	Number of Controls	Odds Ratio[1]	95% Confidence Interval	P-values Overall[2]	P-values Trend[3]
All prevalent diabetics (N=242)						
Total Dietary Fiber[4] (grams/day)						
> 30	17	24	1.00			
20-29	52	89	0.99	(0.46,2.16)		
10-19	132	308	0.68	(0.33,1.39)		
< 10	41	121	0.54	(0.25,1.19)	0.12	0.02
Diabetics with prior diet reporting times before reported diagnosis age (N=163)						
Total Dietary Fiber[4] (grams/day)						
> 30	10	24	1.00			
20-29	35	89	1.05	(0.43,2.56)		
10-19	87	308	0.69	(0.30,1.60)		
< 10	31	121	0.62	(0.25,1.52)	0.27	0.08

[1]Calculated from the logistic model that included the fiber score, six age groups, sex, two ethnic groups, continuous body mass index, and an offset for the sampling fractions by age, sex, ethnic group, and county.
[2]Calculated from the likelihood ratio test for the logistic regression model with and without the factored variable.
[3]Calculated from the likelihood ratio test where categorized variables were treated as continuous.
[4]Prior total dietary fiber intake from the FFQ.

general, had difficulty remembering the age at which they were diagnosed. The medical records data are probably a more accurate source of age at diagnosis.

Results in Perspective with the Existing Literature

SLVDS in Perspective. The excess risk associated with BMI (OR = 3.83 per 10 unit change), waist to hip ratio (OR = 1.61 per .1 unit change), and parental history of diabetes (OR = 2.51), when prevalent cases were compared with nondiabetic controls, were all consistent with the existing literature. This suggests, on a global level, that SLVDS study methods were reliable and of similar accuracy when compared to previously reported studies.

Studies of Dietary Fiber and Blood Insulin. The literature relevant to the hypothesis that low dietary fiber is inversely associated with blood insulin concentrations was reviewed in detail in Chapter 1. To summarize, studies in Africa and Australia have observed lower serum glucose and insulin levels in human populations with "traditional" diets, high in unrefined carbohydrates, compared to populations with "western" diets. Conclusions are limited in these group comparisons because other potential risk factors have not been accounted for. A large number of clinical investigations since the mid-1970's have reported the effects of experimental diets high in dietary fiber on carbohydrate metabolism in normal and diabetic subjects. Inferences from these experimental studies have been limited by the short period of time on special diets, by limited power, and by lack of control for potentially modifying variables like obesity. In spite of these limitations, a fairly consistent finding emerged from the literature review presented in Chapter 1 which suggested that dietary fiber is inversely associated with post-prandial concentrations of blood insulin.

The findings from the cross-sectional analyses reported here replicate these ecologic and experimental findings in a population-based setting with nonexperimental methods. Five hundred and eighty six persons without a prior diagnosis of diabetes were administered an OGTT and a 24-hour diet recall. Plasma insulin concentrations were measured while fasting and two hours after the OGTT. The unadjusted mean plasma insulin (Table 3.14) increased as the level of reported total dietary fiber intake decreased. Similar results were seen within subcategories of glucose tolerance (i.e. previously undiagnosed diabetes, impaired glucose tolerance and normal glucose tolerance). To determine if this observation could be explained by other factors related to both fiber intake and insulin levels, fasting and 2-hour OGTT plasma insulin concentrations were regressed on dietary fiber intake while controlling for age, sex, ethnicity, body mass index, calories, and carbohydrate. These adjusted analyses (Table 3.15) suggested that high fiber intake lowered fasting plasma insulin levels, but did not effect the change in insulin concentration following an OGTT.

One way to evaluate whether an observed association in a particular study is a true association is to look for its consistent presence across different study designs. If bias created by a particular study design could explain the observed association then you would not expect to see the association across a variety of study designs which had independent sources of error. As opposed to the experimental studies that have been described, the present study looked at a free living population, not people coming into a clinic to be assigned a new diet. These subjects were reporting what they ate yesterday, not fiber additives or high fiber diets that they would not otherwise eat. In the ecologic studies reported, which collected data on carbohydrate metabolism in individuals and then noted that populations with lower serum glucose and insulin levels also

ate less refined carbohydrate, the group level data on diet did not allow analyses which controlled for other potentially confounding variables. Similar findings are presented here after adjustment for age, sex, ethnicity, body mass index, calories, and carbohydrate.

These cross-sectional findings support the hypothesis that dietary fiber intake is inversely associated with fasting plasma insulin concentration. However, the impact of a 10 percent decrease in plasma insulin concentration on the occurrence of NIDDM remains unclear.

Studies of Dietary Fiber and NIDDM. Relatively few studies have reported findings that relate dietary fiber to the incidence of NIDDM. Diabetes can be induced by diet in sand rats (83,84,85,86), but it remains unclear whether this can be accounted for by energy imbalance or some qualitative aspect of the diet. Migrants from Japan to Hawaii have 1.74 times the prevalence of diabetes compared to their counterparts in Hiroshima. The diets in Hawaii are similar in total energy consumption to those in Hiroshima, but contain more than twice as much animal fat and simple carbohydrate and less than half the quantity of complex carbohydrates (30). While these ecologic data are consistent with the hypothesis that low dietary fiber is a risk factor for NIDDM, many social, economic, physical activity, and dietary variables will also be correlated in these group comparisons and may themselves be the basis for the association. Consequently, the independent role of dietary fiber in the development of NIDDM remains in question.

Few epidemiologic studies have measured both dietary indicators of fiber intake and diabetes in the same individuals. A prospective study of Pima Indian women reported significantly higher intakes of carbohydrate and starch, which would likely be correlated with fiber, in 87 women who subsequently developed

diabetes compared to 100 women who did not develop the disease (120). However, subjects developing NIDDM ate 100 calories more per day on average and comparisons were not adjusted for weight gain, obesity, or calorie intake. Because of this, the role of fiber independent of weight gain and other dietary factors was not clarified.

Two studies compared vegetarians and nonvegetarians. The first found that nonvegetarians had significantly higher FPG concentrations than vegetarians (117) and the second study observed that nonvegetarians had 1.4 and 1.8 times the risk of diabetes-related mortality in females and males, respectively, after adjusting for age and percent desirable weight (118). To the extent that: (1) meat consumption correlates with a low intake of fiber, and (2) FPG and diabetes-related mortality correlate with incidence of NIDDM, these data are consistent with an association between low fiber and NIDDM incidence. However, the dietary or other factor that might protect vegetarians remains in question and the impact on incidence is unknown.

In the SLVDS, prevalent cases had somewhat higher reported intakes of fiber prior to diagnosis than controls. Across fiber categories, there was a systematic trend of decreasing excess risk as fiber intake decreased. This finding was the opposite of that hypothesized at the onset of the study. The findings were similar for all fiber scores, by season of interview, and when IGTs were excluded from the control group. However, when the diabetic group was limited to cases of less than five years duration, the association was no longer present.

The diagnosis of NIDDM in 30 controls without a previous history of diabetes allowed the case-control analysis of prevalent cases to be replicated comparing those 30 previously undiagnosed diabetics to 488 normal controls.

Current dietary fiber intake was measured by two methods, a FFQ and a 24-hour diet recall. Four of the six FFQ scores showed an excess risk of NIDDM in the lowest fiber category. However, the relationship did not hold up at intermediate levels of fiber intake. With the 24-hour recall data, the lowest category of all three of the fiber fractions was associated with an excess risk of NIDDM. After adjusting for calorie and carbohydrate intake, the excess risk associated with low fiber consumption approached 1.0.

The present study is the first relating diet to NIDDM to measure fiber intake directly. In previous studies conclusions about fiber's role in the etiology of NIDDM had to be tempered by caviats like "to the extent that fiber is inversely related to meat consumption". This is also the first population-based study to look at fiber while holding other factors in the diet constant. The prevalent case-control design and consequent retrospective recall of diet appears to be the most serious limitation of the results presented here. Results from the separate analysis of short duration cases and previously undiagnosed cases were consistent with no true association.

Conclusion. Among controls, dietary fiber was inversely associated with fasting plasma insulin concentration adjusted for calories and carbohydrate. However, in a comparison of previously known diabetics and nondiabetic controls there was a tendency toward higher reported intakes of fiber prior to diagnosis among the diabetics. When the diabetic group was limited to cases of less than five years duration, the association was no longer present. When previously undiagnosed diabetics were compared to normal controls, there was no association between fiber intake and NIDDM after adjusting for calories and carbohydrate. These findings do not support the hypothesis that increasing dietary fiber intake could reduce the future occurrence of NIDDM.

Suggestions for Future Research

Because of the very few studies which have studied the relationship between diet and NIDDM incidence, and because the morbidity and mortality from this disease is so high in the United States, continued research should address the role of diet in the primary prevention of this disease. The following recommendations for future research result from the work presented here.

Diet Assessment. Because some nutrients, like vitamin A, are present in a limited number of foods, they do not require information on the complete diet in order to estimate the relative intake among individuals, and the FFQ is an appropriate method for such nutrients. The macronutrients and fiber, however, occur in so many foods that the usefulness of the FFQ method becomes more questionable. Work by Willett et al (178) suggests that these dietary factors can be adequately measured in nurses in Boston, but similar validations of the FFQ method in a variety of populations are needed. The FFQ developed by Willett et al (178) and one developed by Block et al (177) have been proposed for use in other studies. This standardization of the FFQ method should be encouraged. In addition, a short prestudy protocol for assessing the diet in new populations to be studied, which can be used to evaluate the appropriateness of the foods included on the standard FFQ questionnaires, should be developed.

In spite of the criticism which has been directed toward the 24-hour recall method, it is a useful tool in studies of carbohydrate metabolism and in epidemiologic studies investigating factors related to the occurrence of NIDDM. The 24-hour recall is a general method that allows estimates to be made for all nutrients for which food composition has been determined. This includes the

macronutrients and fiber. The same data can be analyzed for other nutrients that become of interest, or become available in the nutrient data base, at a later date. It is a direct measure of the diet 24-hours prior to biochemical measurements taken the same day as the 24-hour recall and may be used to study the impact of short term diet on carbohydrate metabolism. When administered on sufficient numbers of individuals, the 24-hour diet recall provides summary data for describing the nutrient composition of the population's diet and for contrasting subgroups or for comparison with other study populations. To the extent that the average diet is stable over the time of reference, the 24-hour diet recall can be used in cohort studies and in cross-sectional studies of controls with previously undiagnosed diabetes, IGT, and normal glucose tolerance, to look for associations between current diet and disease occurrence. The sample sizes required will depend on the magnitude of the effect expected and the within and between person variability in the population being studied. Findings based on 24-hour recall data can be directly interpreted in terms of the absolute amount of a nutrient which is expected to produce the effect being studied and, consequently, the data is easily translated into public health practice.

Administration of the 24-hour diet recall should only be considered with the support of an existing resource for interviewer training, coding, and nutrient analysis of the recalls as was provided by the Nutrition Coding Center for this study. Resources should not be spent by individual studies to replicate the standardized coding procedures and nutrient data bases already available.

Study Design. The inaccuracy of retrospective recall of diet, and potential bias associated with differential recall between cases and controls, argue that future case-control studies of diet in the etiology of diabetes should not include

long-duration cases. In future case-control studies, the inclusion of previously undiagnosed diabetics and people with impaired glucose tolerance should be considered. The number of people with undiagnosed NIDDM is estimated to be about equal to the number with diagnosed NIDDM in the general population. In our study of 607 controls, 5 percent were diagnosed in our clinic with NIDDM and 15 percent were diagnosed with IGT. These subgroups provide the opportunity to look at risk factors at an earlier stage in the disease progression and before diagnosis related changes in diet occur. Ongoing prospective studies of large cohorts, designed to study heart disease or cancer (e.g. the Nurse's Health Study (178)), have a particularly good opportunity to evaluate the role of dietary fiber in the subsequent development of NIDDM.

Formulating the Hypothesis. Much of the work to date on diet and NIDDM etiology or prognosis, including the study presented here, has attempted to study the independent effect of fiber, carbohydrate or fat. The glycemic index is a recent attempt to focus on the metabolic response to individual foods rather than just the nutrient composition of the diet. The glycemic index compares the area under the blood glucose curve for each food as a percentage of the area after taking the same amount of carbohydrate from a reference food like white bread. These studies have shown that equal amounts of carbohydrate have differing effects on blood glucose depending on the food source and preparation. The glucose response is important in diabetic control, but for etiologic studies it may be of more interest to look at the insulin response to a food or meal. Large population studies which collect diet recalls or food records may be able to look at foods, food groups, meals, or patterns of eating which are associated with the subsequent development of NIDDM rather than just the nutrient content of the diet.

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APPENDIX A: Household Enumeration Interview

SAN LUIS VALLEY DIABETES STUDY
Household Enumeration
Form H1

_____ Address

_____ Household Enumeration Number

_____ Interviewer Number

_____ First Not at Home Date Time

_____ Second Attempt

_____ Third Attempt

Interviewer Actions are CAPITALIZED Interview Remarks are in **"Bold type"**

GREETING: (Spanish version, if necessary)

"Hello, I'm _____ from the San Luis Valley Diabetes Study being sponsored by the University of Colorado School of Medicine. Our study is looking at diabetes and heart disease among residents of the Valley. My purpose today is to do a short health survey on people living here. Here is a letter that explains the study further. You may like to have others living here read it."

HAND LETTER (PROBE: HAND SPANISH VERSION OF LETTER)

"This confidential interview will only take about 5 to 10 minutes. Would you have time now to participate?" (Household Head, Spouse, Adult Child of Household) (PROBE)

IF YES, GO TO BOTTOM OF PAGE FOR BEGINNING OF INTERVIEW

IF NO, ASK: **"Is there another person who might be able to help us?" or "Is there another time that would be more convenient?"**

_____ Appointment Time and Date

IF NO TO THE APPOINTMENT, CHECK HERE / /_____ Reason Given for Refusal

WHEN RESPONDENT IS COMFORTABLE BEGIN IN SPANISH OR IN ENGLISH

"For us to get a good understanding about the people living in this county we need to ask just a few questions about each person living here. I will ask the same questions about everyone. These are questions you can answer for everyone. First, could you please tell me how many people live in this house?"

PUT ANSWER ON NEXT PAGE.

Revision 4, 1/6/84

Form H1, Page 2

Interviewer Number

1 2 Household Enumeration Number
A C
County

1. Number of people in household_____.

2. "Would you please tell me the full name of everyone living in this house starting with your name." (GET RESPONDENT'S NAME FIRST AND THEN ALL OTHERS, WRITE IN SPACE BELOW)

Now for the set of questions on each of these persons, again let's start with you:

	Respondent	Household Members			
For each member of the house ask: What is their:	1	2	3	4	5
Name? First - - - - - - - - - - - - - - - - - Last	- - - - -	- - - - -	- - - - -	- - - - -	- - - - -
Relation to Household Head? 1. HH 2. Spouse 3. Son 4. Daughter 5. Grandparent 6. Other Relative 7. Unrelated Boarder					
Date of Birth? MO/DY/YR					
Sex? 1. Male 2. Female					
Are you/they of Spanish/ Hispanic origin or descent? 1. Yes 9. Don't know 2. No					
Were you/they born in the USA? 1. Yes 9. Don't know 2. No					
Has this person ever been told by a Doctor they have Diabetes? 1. Yes 9. Don't know 2. No					

REPEAT QUESTION SET FOR PERSON 2 AND ALL OTHERS

INTERVIEW CHECK: DOES QUESTION 1 MATCH THE LAST HOUSEHOLD MEMBER'S NAME? YES NO: GO BACK TO CORRECT

"Thank you for your time. Is there a number here that my supervisor could call to verify my work?" _____ Telephone Number

No Phone

1 ☐ Yes 2 ☐ No
Audited

"We certainly appreciate your help with this portion of the study. It is possible that someone in this household will be picked to participate in our bigger study. We would, of course, call them and ask if they would be willing to do so."

Time and Date Completed

Edit: _____ Date _____ Initials

Entry: _____ Date _____ Initials

NOTE: CHECK OTHER SIDE OF FORM!

Revision 4, 1/6/84

APPENDIX B: Letter of Introduction to Participants

University of Colorado Health Sciences Center

Department of Preventive Medicine and Biometrics School of Medicine

Campus Box C-245
4200 East Ninth Avenue
Denver, Colorado 80262
(303) 394-[illegible]

Dear

We are writing to ask you to participate in the San Luis Valley Diabetes Study Clinic. Some time in the past six months, you have been contacted by either your Doctor or one of our staff to ask for your help in an earlier part of the Study. For the next two years, we will be seeing people from Alamosa and Conejos Counties in our Clinic. The aim of the Study is to better understand diabetes and heart disease. Both of these diseases are very common in the United States and the San Luis Valley. With your participation, we hope to better understand both the causes and possible methods of preventing these diseases.

You have been selected to participate as one person in a scientifically determined sample or because you have had diabetes mellitus diagnosed some time in the past. It is important for our understanding that we compare how people are different, so each and every person's participation is essential. Someone will be calling or writing to you to set up a convenient time for you to come to the clinic. A clinic visit will take the better part of a morning to complete. For your participation you will receive about $400.00 worth of FREE medical tests and a $10.00 payment.

Again, welcome to the San Luis Valley Diabetes Study. We will be in touch to set up a clinic appointment. If you have questions, please don't hesitate to call us collect at the Clinic.

Yours sincerely,

SLV Diabetes Study
Field Coordinator

APPENDIX C: Instructions for the Clinic Visit

SAN LUIS VALLEY DIABETES STUDY

1016 West Avenue #4, Alamosa, Colorado 81101
303-589-5801

INSTRUCTIONS FOR YOUR CLINIC VISIT

Thank you for agreeing to participate in the San Luis Valley Diabetes Study. These instructions will tell you about the study and what you need to do before you come to the clinic for your appointment.

BACKGROUND TO THE STUDY

The San Luis Valley Diabetes Study is a three-year project funded by the National Institutes of Health and conducted by the University of Colorado School of Medicine. The aim of the study is to better understand diabetes and heart disease. Both of these diseases are very common in the United States and the San Luis Valley. With your participation, we hope to better understand both the causes and possible methods of preventing these diseases.

We are asking all the diabetics in Alamosa and Conejos counties to come to the clinic. We are also asking an equal number of scientifically selected non-diabetics (controls) to come for a clinic visit. For us to understand these diseases, we need to examine the ways diabetics and non-diabetics may be different from each other. Everyone's participation is essential and will enable us to learn a great deal.

WHAT TO EXPECT AT THE CLINIC

The San Luis Valley Diabetes Study has a staff of 7 extremely well-qualified and friendly people who will be working with you at the clinic. While at the clinic, we will ask you questions about where you live, your schooling, jobs, family history, diet, and your exercise. We will draw blood to test for diabetes, cholesterol level, pancreas, and kidney function; do an examination for high blood pressure, for blood vessel blockage in the legs, and an ECG (heart test); and take photographs of the inside of your eyes. This will require drops in your eyes that will dilate (open) your pupils. These tests are all routine medical tests. Naturally, they will take some time to complete. You should expect your clinic appointment to last about 3 1/2 to 4 hours.

Form A3, 6/12/85
Revision 3

FOR YOUR PARTICIPATION YOU WILL RECEIVE:

1) Approximately $400 worth of standard medical tests for free. Some of these results will be given to you when you finish your clinic visit. The remainder of the results will be sent to you and the doctor of your choice to aid in your medical care about 3-4 months after your clinic visit.

2) Ten ($10) dollars will be given to you for your time and participation at the end of all tests. This is a small way that we can show you our appreciation.

Your clinic appointment is scheduled for:

_____	_____	_____
Day	Date	Time

We are located at 1016 West Avenue, Alamosa (see enclosed map).

1) Please do NOT eat or drink anything for 12 hours prior to your appointment time. This is called a 12-hour fast. It is essential that you follow this requirement, as many tests depend upon this rule. Only with the fast can we accurately test for diabetes in non-diabetics and determine how well the pancreas is working in diabetics. Water is the ONLY thing you may have. This means NO COFFEE; NO FOOD; NO DRINKS; and NO SNACKS of any kind.

   BEGIN YOUR FAST AT: 8:00 PM.

2) Please fill out the family and medical history forms that are included in this packet before coming in for your clinic appointment. PLEASE BRING THEM WITH YOU WHEN YOU COME FOR YOUR APPOINTMENT.

3) Please bring all your prescribed medications and vitamins in their bottles with labels with you to the clinic. You can put them in a paper bag or purse, whatever is easiest for you. If you wear contact lenses, you should be prepared to take them out or wear glasses for your visit.

   Please bring sunglasses and shorts if you own them. These will make the dilation of your pupils and other portions of the physical exam more comfortable.

AGAIN, THANK YOU FOR YOUR PARTICIPATION. WE APPRECIATE ALL YOUR HELP WITH THE STUDY. WE LOOK FORWARD TO SEEING YOU IN THE CLINIC.

SAN LUIS VALLEY DIABETES STUDY

INSTRUCTIONS FOR DIABETICS

We will do a diabetes test that will help tell us how well the pancreas is working. This test will give you some sugar, so you do not have to worry about low blood sugar. After the test, you may have a snack, if you want it.

If you:	then do this:
a) Do Not Take diabetes pills or insulin shots	- Do not eat or drink anything except water for 12 hours
b) Take Only diabetes pills	- Do not eat or drink anything except water for 12 hours - Take your pill before clinic
c) Take Only insulin shots	- Do not eat or drink anything except water for 12 hours - Do not take your shot - Bring your insulin with you - After a blood sample, we will help you take your insulin
d) Take Both pills and insulin shots	- Do not eat or drink anything except water for 12 hours - Take your pill before clinic - Do not take your shot - Bring your insulin with you - After a blood sample, we will help you take your insulin

These instructions have been reviewed by doctors here and at the medical school, and are safe for you to follow. If you do not understand them, or have questions about them, call Louise Kahn, R.N., at the clinic (589-5801). She can contact your doctor for further instructions if necessary.

APPENDIX D: Refusal Questionnaire

SAN LUIS VALLEY DIABETES STUDY
Diabetic Refusal Interview
Form A6D

	ED/BNA	BG	HH	Person	Record 1 Col:
Household Number	____ /	__ /	____ /	__	(1-12)

First Name _____
Middle Initial _____
Last Name _____

Interviewer Code __ __ (13,14)

HELLO, MY NAME IS _____, AND I'M CALLING TO SEE IF YOU WOULD RECONSIDER YOUR DECISION NOT TO COME TO THE SAN LUIS VALLEY DIABETES STUDY CLINIC.

1 = NO (COMPLETE N1)
2 = YES (SCHEDULE CLINIC VISIT; STOP) — (15)

WOULD YOU BE ABLE TO ANSWER ABOUT 5 MINUTES OF QUESTIONS NOW, INSTEAD OF COMING TO OUR CLINIC, TO HELP US WITH OUR STUDY? ALL YOUR ANSWERS WILL BE KEPT CONFIDENTIAL.

1 = Refusal _____ — (16)
Reason

DATE/TIME

2 = No contact (code only after 3 attempts) 1. _____
2. _____
3 = Continue interview 3. _____
STOP

Question Number

S1-1. How long, altogether, have you lived in the San Luis Valley? (IF YOU LIVED IN THE VALLEY FOR 5 YEARS, MOVED AWAY AND HAVE BEEN BACK FOR 4 YEARS, YOUR ANSWER WOULD BE 9 YEARS)

TOTAL: __ __ / __ __ (17-20)
Years Months

S5-1. How many brothers do you have? (INCLUDE DEAD AND ALIVE) __ __ (21,22)

-2. How many sisters do you have? (INCLUDE DEAD AND ALIVE) __ __ (23,24)

-3. How many children do you have? (INCLUDE DEAD AND ALIVE) __ __ (25,26)

S1-15. Are you currently never married, living with someone in a long-term relationship, married, separated, divorced, or widowed? — (27)

1 ☐ Never Married 3 ☐ Married 5 ☐ Divorced (BLANK 28,29)
2 ☐ Living Together 4 ☐ Separated 6 ☐ Widowed

Revision 1, 2/20/86

Form A6D, Page 2

Question Number			Record 1 Col:
S1-20.	What is the highest grade or level of schooling that you have completed? (CIRCLE LAST GRADE COMPLETED) Grade School. . . . . .0 1 2 3 4 5 6 7 8 High School . . . . . .9 10 11 12 College . . . . . .13 14 15 16 17+ = Graduate School	— —	(30,31)
-24.	Which of the followi - best describes your current work status? 1☐ Currently employed full or part-time 2☐ Currently retired 3☐ Currently full-time homemaker 4☐ Currently not working, but looking for work in past 4 weeks. 5☐ Currently not working, and not looking for work in past 4 4 weeks. 6☐ Other _____ (Please Specify) 7☐ Have never worked	—	(32)
-58.	What was the first language that you learned to speak? 1☐ English 2☐ Both English and Spanish 3☐ Spanish 4☐ Other--Please specify_____	—	(33)

NOW, I WOULD LIKE TO ASK YOU A FEW QUESTIONS ABOUT YOUR HEALTH.

M1-1.	Compared with other people your age, would you say that your health is: 1☐ Excellent? 2☐ Good? 3☐ Fair? 4☐ Poor?	—	(34)
-3.	How many times have you seen a doctor within the past 12 months? (99 = DK)	— — Times	(35,36)
			(BLANK 37,38)

Revision 1, 2/20/86

Form A6D, Page 3

Question Number

Record 1 Col:

MI-4. During the past 12 months, how many times have you been a patient overnight in a hospital? (00 = NONE, 99 = DK) — — Times (39,40)

-17. How much do you weigh now? — — — Pounds (41-43)

-28. Have you ever been told by a doctor that you had high blood pressure? — (44)

1☐ NO 2☐ YES 9☐ DK

-33. Are you now taking any medicine for your high blood pressure? — (45)

1☐ NO 2☐ YES 9☐ DK

-35. Have you ever been told by a doctor that you had a stroke? — (46)

1☐ NO 2☐ YES 9☐ DK

A4-10. Have you ever been told by a doctor that you had a heart attack? — (47)

1☐ NO 2☐ YES 9☐ DK

MI-115. Have you smoked at least 100 cigarettes in your lifetime? — (48)

1☐ NO (IF NO, SKIP TO QUESTION 127) 2☐ YES

-116. Do you smoke cigarettes now? — (49)

1☐ NO (FORMER SMOKERS) 2☐ YES (CURRENT SMOKERS) (INCLUDE OCCASIONALLY)

-117. How old were you when you started smoking cigarettes regularly?

— — Years Old

117. How old were you when you started smoking cigarettes regularly? — — (50,51)

— — Years Old

-118. How old were you when you stopped smoking cigarettes? — — (52,53)

— — Years Old

(BLANK 54,55)

Revision 1, 2/20/86

Form A6D, Page 4

Record 1 Col:

Question Number

M1-119. On average, how many cigarettes did you smoke a day?

__ __ Cigarettes (1 PACK = 20 CIGARETTES)

1☐ Per Day
2☐ Per Week
3☐ Per Month
4☐ Per Year
5☐ In Lifetime

119. On average, how many cigarettes do you smoke a day?

__ __ Cigarettes (1 PACK = 20 CIGARETTES) —— (56,57)

1☐ Per Day
2☐ Per Week
3☐ Per Month
4☐ Per Year
5☐ In Lifetime

— (58)

-127. Have you ever consumed beer, wine, or other alcoholic beverages? — (59)

1☐ NO 2☐ YES

-128. Are you still drinking any of these? — (60)

1☐ NO 2☐ YES

-135. Did any of your family have a heart attack?

Mother	1☐ NO	2☐ YES	9☐ DK	— (61)
Father	1☐ NO	2☐ YES	9☐ DK	— (62)
Brothers	1☐ NO	2☐ YES	9☐ DK	— (63)
Sisters	1☐ NO	2☐ YES	9☐ DK	— (64)
Children	1☐ NO	2☐ YES	9☐ DK	— (65)

Which, if any, of these members of your family had diabetes?

-138. Your mother?	1☐ NO	2☐ YES	9☐ DK	— (66)
-139. Either of your mother's parents?	1☐ NO	2☐ YES	9☐ DK	— (67)
-140. Your father?	1☐ NO	2☐ YES	9☐ DK	— (68)
-141. Either of your father's parents?	1☐ NO	2☐ YES	9☐ DK	— (69)
-142. Any of your brothers?	1☐ NO	2☐ YES	9☐ DK	— (70)
-143. Any of your sisters?	1☐ NO	2☐ YES	9☐ DK	— (71)
-144. Any children you may have had?	1☐ NO	2☐ YES	9☐ DK	— (72)

(BLANK 73,74)

Form A6D, Page 5

Question Number

Record 1 Col:

M1-146. Have you ever been tested for Diabetes? — (75)

1☐ NO 2☐ YES 9☐ DK

-147. At what age? — — Age (FOR FIRST TEST) — — (76,77)

-148. What was the reason you were tested for diabetes? — (78)

1☐ Routine Screening
2☐ Screened Because of Family History
3☐ Screened Because of Other Trouble
4☐ Symptoms of Diabetes
5☐ During Pregnancy
8☐ Other_____ Please Specify
9☐ DK

-149. Has a doctor ever told you that you had diabetes (Sugar Diabetes)? — (79)

1☐ NO
2☐ YES
3☐ YES, Pregnancy Only
4☐ YES, Borderline Only
5☐ YES, Prediabetes Only
6☐ YES, Chemical Diabetes Only
7☐ YES, Potential Diabetes Only
8☐ YES, Other_____
9☐ DK

(BLANK 80,81)

Form A6D, Page 6

Record 1 Col:

Question Number

Mi-150. At what age was your diabetes first diagnosed? — — (82,83)

— — Age 1☐ Remembered Well 2☐ Estimated

Over the first six months after you were told you had diabetes, what treatment was prescribed?

-151. Nothing (PROBE) 1☐ NO 2☐ YES 9☐ DK — (84)

-152. Special Diabetic Diet 1☐ NO 2☐ YES 9☐ DK — (85)

-153. Pills (Oral Hypoglycemics) 1☐ NO 2☐ YES 9☐ DK — (86)

-154. Insulin Shots 1☐ NO 2☐ YES 9☐ DK — (87)

↓

-155. How many total units did you take per day? (999 = DK)
— — — Units — — — (88-90)

-156. Other_____ 1☐ NO 2☐ YES 9☐ DK — (91)
Please Specify

-157. What treatment are you now taking for your diabetes?

-158. Nothing (PROBE) 1☐ NO 2☐ YES 9☐ DK — (92)
(SKIP TO QUESTION 166)

-159. Special Diabetic Diet 1☐ NO 2☐ YES 9☐ DK — (93)

-160. Pills (Oral Hypoglycemics) 1☐ NO 2☐ YES 9☐ DK — (94)

-161. Insulin Shots 1☐ NO 2☐ YES 9☐ DK — (95)

↓ ↓
(SKIP TO QUESTION 166)

-162. How many total units do you take per day? (999 = DK)
— — — Units — — — (96-98)

-163. How many times per day? — — Times — — (99,100)

-164. How long have you been on insulin? (Ignore breaks in treatment less than 1 month long) (01 = 1 Year or Less, 99 = DK)
— — Years — — (101,102)

-165. If you stopped taking your insulin for a week or more, would it make you feel sick? — (103)

1☐ NO 2☐ YES 9☐ DK
~~(SKIP TO QUESTION 168)~~

(BLANK 104)

Form A6D, Page 7

Question Number — Record 1 Col:

M1-166. Have you ever taken insulin for your diabetes? — (105)

1☐ NO 2☐ YES ↓

-167. About how many years did you take it? (99 = DK) — — Years — — (106,107)

(ASK EVERYONE:)

-168. Have you taken any other treatments? — (108)

1☐ NO 2☐ YES _____ 9☐ DK
Please Specify

-169. Would you say that over the past 12 months the control of your diabetes has been excellent, good, fair, poor, or very poor? — (109)

1☐ Excellent

2☐ Good

3☐ Fair

4☐ Poor

5☐ Very Poor

9☐ DK

Have you ever been told by a doctor that your diabetes was affecting:

			AGE WHEN TOLD	
-170.	Your Eyes?	1☐ NO 9☐ DK 2☐ YES	— —	— — — (110-112)
-171.	The Back of Your Eyes (Retina)?	1☐ NO 9☐ DK 2☐ YES	— —	— — — (113-115)
-172.	Your Kidneys?	1☐ NO 9☐ DK 2☐ YES	— —	— — — (116-118)
-173.	Your Nerves?	1☐ NO 9☐ DK 2☐ YES	— —	— — — (119-121)
-174.	Your Ability to Feel Hot or Cold Temperatures?	1☐ NO 9☐ DK 2☐ YES	— —	— — — (122-124)
-175.	Your Skin?	1☐ NO 9☐ DK 2☐ YES	— —	— — — (125-127) (BLANK 128,129)

Record 1 Col:

Question Number

M1-176. Have you ever had laser photocoagulation surgery of your eyes? — (130)

1☐ NO 2☐ YES 9☐ DK

↓

-177. How old were you? — — Age — — (131,132)

-178. Have you ever had an episode of ketoacidosis or diabetic coma? — (133)

1☐ NO 2☐ YES 9☐ DK

↓

-179. H w old were you the first time? — — Age — — (134,135)

-180. When was the last time you saw a doctor for your diabetes? — (136)

1☐ Less Than 3 Months Ago

2☐ 3-6 Months

3☐ 7-12 Months

4☐ 1-2 Years

5☐ Over 2 Years

9☐ DK

(BLANK 137,138)

Form A6D, Page 9

Record 1 Col:

Question Number

THAT'S THE END OF THE QUESTIONS. I ONLY HAVE ONE LAST QUESTION FOR YOU.

WE WOULD LIKE TO BETTER UNDERSTAND THE STEPS WE COULD TAKE THAT MIGHT ENCOURAGE PEOPLE TO COME TO OUR CLINIC. WOULD ANY OF THE FOLLOWING MAKE A DIFFERENCE IN YOUR DECISION NOT TO PARTICIPATE IN THE STUDY.

A shorter clinic	1 = NO	2 = YES	3 = MAYBE	9 = DK	__	(139)
A night clinic	1 = NO	2 = YES	3 = MAYBE	9 = DK	__	(140)
No blood drawing	1 = NO	2 = YES	3 = MAYBE	9 = DK	__	(141)
More money (10 vs 15)	1 = NO	2 = YES	3 = MAYBE	9 = DK	__	(142)
Your doctor's advice	1 = NO	2 = YES	3 = MAYBE	9 = DK	__	(143)
Other_____						
ANY OTHER COMMENTS?	1 = NO	2 = Yes			__	(144)

(BLANK 145-150)

THANK YOU VERY MUCH FOR YOUR TIME.

Editer ID: __ __

Edit Date: __ / __

Revision 1, 2/20/86

SAN LUIS VALLEY DIABETES STUDY
Control Refusal Interview
Form A6C

	ED/BNA	BG	HH	Person	Record 1 Col:
Household Number	____ /	__ /	____ /	__	(1-12)

First Name _____
Middle Initial _____
Last Name _____

Interviewer Code __ __ (13,14)

HELLO, MY NAME IS _____, AND I'M CALLING TO SEE IF YOU WOULD RECONSIDER YOUR DECISION NOT TO COME TO THE SAN LUIS VALLEY DIABETES STUDY CLINIC.

1 = NO (COMPLETE N1)
2 = YES (SCHEDULE CLINIC VISIT; STOP) __ (15)

WOULD YOU BE ABLE TO ANSWER ABOUT 5 MINUTES OF QUESTIONS NOW, INSTEAD OF COMING TO OUR CLINIC, TO HELP US WITH OUR STUDY? ALL YOUR ANSWERS WILL BE KEPT CONFIDENTIAL.

1 = Refusal _____ (Reason) __ (16)

DATE/TIME

2 = No contact (code only after 3 attempts)
1. _____
2. _____

3 = Continue interview
3. _____ (STOP)

Question Number

S1-1. How long, altogether, have you lived in the San Luis Valley? (IF YOU LIVED IN THE VALLEY FOR 5 YEARS, MOVED AWAY AND HAVE BEEN BACK FOR 4 YEARS, YOUR ANSWER WOULD BE 9 YEARS)

TOTAL: __ __ / __ __ (17-20)
Years Months

A5-1. How many brothers do you have? (INCLUDE DEAD AND ALIVE) __ __ (21,22)

-2. How many sisters do you have? (INCLUDE DEAD AND ALIVE) __ __ (23,24)

-3. How many children do you have? (INCLUDE DEAD AND ALIVE) __ __ (25,26)

S1-15. Are you currently never married, living with someone in a long-term relationship, married, separated, divorced, or widowed? __ (27)

1☐ Never Married 3☐ Married 5☐ Divorced
2☐ Living Together 4☐ Separated 6☐ Widowed

(BLANK 28,29)

Revision 2, 2/19/86

Form A6C, Page 2

Record 1 Col:

Question Number

S1-20. What is the highest grade or level of schooling that you have completed? (CIRCLE LAST GRADE COMPLETED) — — (30,31)

Grade School. 0 1 2 3 4 5 6 7 8

High School 9 10 11 12

College . . . . . .13 14 15 16 17+ = Graduate School

-24. Which of the following best describes your current work status? — (32)

1☐ Currently employed full or part-time

2☐ Currently retired

3☐ Currently full-time homemaker

4☐ Currently not working, but looking for work in past 4 weeks.

5☐ Currently not working, and not looking for work in past 4 4 weeks.

6☐ Other _____ Please Specify

7☐ Have never worked

-58. What was the first language that you learned to speak? — (33)

1☐ English

2☐ Both English and Spanish

3☐ Spanish

4☐ Other--Please specify_____

NOW, I WOULD LIKE TO ASK YOU A FEW QUESTIONS ABOUT YOUR HEALTH.

M1-1. Compared with other people your age, would you say that your health is: — (34)

1☐ Excellent? 2☐ Good? 3☐ Fair? 4☐ Poor?

-3. How many times have you seen a doctor within the past 12 months? (99 = DK) — — Times (35,36)

(BLANK 37,38)

Revision 2, 2/19/86

Form A6C, Page 3

Question Number

M1-4. During the past 12 months, how many times have you been a patient overnight in a hospital? (00 = NONE, 99 = DK) — — Times (39,40)

-17. How much do you weigh now? — — — Pounds (41-43)

-28. Have you ever been told by a doctor that you had high blood pressure? — (44)

1☐ NO 2☐ YES 9☐ DK

↓

-33. Are you now taking any medicine for your high blood pressure? — (45)

1☐ NO 2☐ YES 9☐ DK

-35. Have you ever been told by a doctor that you had a stroke? — (46)

1☐ NO 2☐ YES 9☐ DK

A4-10. Have you ever been told by a doctor that you had a heart attack? — (47)

1☐ NO 2☐ YES 9☐ DK

M1-115. Have you smoked at least 100 cigarettes in your lifetime? (48)

1☐ NO (IF NO, SKIP TO QUESTION 127) 2☐ YES

-116. Do you smoke cigarettes now? — (49)

1☐ NO (FORMER SMOKERS) 2☐ YES (CURRENT SMOKERS) (INCLUDE OCCASIONALLY)

↓ ↓

-117. How old were you when you started smoking cigarettes regularly?

— — Years Old

117. How old were you when you started smoking cigarettes regularly? — — (50,

— — Years Old

-118. How old were you when you stopped smoking cigarettes? — — (52,53)

— — Years Old

(54,55)

Revision 2, 2/19/86

Form A6C, Page 4

Record 1 Col:

Question Number

M1-119. On average, how many cigarettes did you smoke a day?

__ __ Cigarettes (1 PACK = 20 CIGARETTES)

1☐ Per Day
2☐ Per Week
3☐ Per Month
4☐ Per Year
5☐ In Lifetime

119. On average, how many cigarettes do you smoke a day?

__ __ Cigarettes (1 PACK = 20 CIGARETTES) — — (56,57)

1☐ Per Day — (58)
2☐ Per Week
3☐ Per Month
4☐ Per Year
5☐ In Lifetime

-127. Have you ever consumed beer, wine, or other alcoholic beverages? — (59)

1☐ NO 2☐ YES

-128. Are you still drinking any of these? — (60)

1☐ NO 2☐ YES

-135. Did any of your family have a heart attack?

Mother	1☐ NO	2☐ YES	9☐ DK	— (61)
Father	1☐ NO	2☐ YES	9☐ DK	— (62)
Brothers	1☐ NO	2☐ YES	9☐ DK	— (63)
Sisters	1☐ NO	2☐ YES	9☐ DK	— (64)
Children	1☐ NO	2☐ YES	9☐ DK	— (65)

Which, if any, of these members of your family had diabetes?

-138.	Your mother?	1☐ NO	2☐ YES	9☐ DK	— (66)
-139.	Either of your mother's parents?	1☐ NO	2☐ YES	9☐ DK	— (67)
-140.	Your father?	1☐ NO	2☐ YES	9☐ DK	— (68)
-141.	Either of your father's parents?	1☐ NO	2☐ YES	9☐ DK	— (69)
-142.	Any of your brothers?	1☐ NO	2☐ YES	9☐ DK	— (70)
-143.	Any of your sisters?	1☐ NO	2☐ YES	9☐ DK	— (71)
-144.	Any children you may have had?	1☐ NO	2☐ YES	9☐ DK	— (72)

Revision 2, 2/19/86 (BLANK 73,74)

Form A6C, Page 5

Record 1 Col:

Question Number

M1-146. Have you ever been tested for Diabetes? — (75)

1☐ NO 2☐ YES 9☐ DK

-147. At what age? — — Age (FOR FIRST TEST) — — (76,77)

-148. What was the reason you were tested for diabetes? — (78)

1☐ Routine Screening
2☐ Screened Because of Family History
3☐ Screened Because of Other Trouble
4☐ Symptoms of Diabetes
5☐ During Pregnancy
8☐ Other_____ Please Specify
9☐ DK

-149. Has a doctor ever told you that you had diabetes (Sugar Diabetes)? — (79)

1☐ NO
2☐ YES
3☐ YES, Pregnancy Only
4☐ YES, Borderline Only
5☐ YES, Prediabetes Only
6☐ YES, Chemical Diabetes Only
7☐ YES, Potential Diabetes Only
8☐ YES, Other_____
9☐ DK

(BLANK 80,81)

Form A6C, Page 6

Record 1 Col:

Question Number

THAT'S THE END OF THE QUESTIONS. I ONLY HAVE ONE LAST QUESTION FOR YOU.

WE WOULD LIKE TO BETTER UNDERSTAND THE STEPS WE COULD TAKE THAT MIGHT ENCOURAGE PEOPLE TO COME TO OUR CLINIC. WOULD ANY OF THE FOLLOWING MAKE A DIFFERENCE IN YOUR DECISION NOT TO PARTICIPATE IN THE STUDY.

A shorter clinic	1 = NO	2 = YES	3 = MAYBE	9 = DK	___	(82)
A night clinic	1 = NO	2 = YES	3 = MAYBE	9 = DK	___	(83)
No blood drawing	1 = NO	2 = YES	3 = MAYBE	9 = DK	___	(84)
More money (10 vs 15)	1 = NO	2 = YES	3 = MAYBE	9 = DK	___	(85)
Your doctor's advice	1 = NO	2 = YES	3 = MAYBE	9 = DK	___	(86)
Other_____						

ANY OTHER COMMENTS? 1 = NO 2 = Yes ___ (87)

(BLANK 88-125)

THANK YOU VERY MUCH FOR YOUR TIME.

Editer ID: ___

Edit Date: ___ / ___

APPENDIX E: Informed Consent

Form E1, Page 1

SAN LUIS VALLEY DIABETES STUDY

General Informed Consent

You are asked to participate in a study conducted by the University of Colorado School of Medicine to learn about diabetes (sugar diabetes) and heart trouble in persons in the San Luis Valley. We are interested in how your lifestyle and your family background may change your risks for diabetes and heart trouble.

We will ask you questions about where you live, your schooling, jobs, family history, medical history, diet, and your exercise. We will also ask questions about languages you speak, your religion, income, and your family background.

We will give you a sugar syrup to drink as part of the test for diabetes. We will also draw about four tablespoons of blood altogether from your arm at 3 times by venipuncture. This is the standard method used to obtain blood for routine hospital laboratory tests. You can expect to experience some pain at the moment the needle goes into your arm. In about 10% of cases, a small amount of bleeding under the skin will produce a bruise (hematoma). The risk of more serious complications, including temporary clotting of the vein, infection of a bruise, or significant external blood loss, is much less than one in 1,000.

We will do an electrocardiogram (ECG - heart test), and measure the blood pressure in your arm and also in your ankles to locate blood vessel blockage in your legs. These are standard medical tests that carry essentially no risk. You will feel a tightness in your arm or leg as the blood pressure cuff is inflated.

We will place drops in your eyes to open your pupils so that we may examine the inside of your eye (the retina). Approximately one time in 5,000 this will rapidly increase the pressure in the eye (glaucoma) and will require immediate treatment by an ophthalmologist. We will also take pictures of the inside of your eyes with a special camera and a flash (retinal photographs).

The only benefit from these procedures would be that all results will be sent to you and to a physician of your choice for his/her use in your care. You may ask questions about any of these procedures at any time. All personal data will be kept strictly confidential, and any presentation of results will be in summary form only. In the event your participation in this research supported by The National Institutes of Health directly results in injury to you, medical treatment will be available; but as of this time there is no compensation available for such injury. Further information about this treatment may be obtained by calling Dr. Richard F. Hamman at (303) 394-8811.

Authorization

I, _____, have been read the above, have received a copy, and understand the discomforts, inconveniences, and risks of this study. I agree to participate, and I understand that I may refuse to participate or withdraw at any time.

_____ _____

Respondent Signature Date

_____ _____

Interviewer Date

Revision 3, 5/15/84

APPENDIX F: 24-Hour Diet Recall Instructions and Documentation Form

San Luis Valley Diabetes Study
24-Hour Recall Interview
Form D1

INTRODUCTIONS...

"During the next 45 minutes or so, I want to ask you questions about the foods you eat. Right now, there is not a lot known about whether certain foods or patterns of eating may bring on diabetes and your answers along with answers from all the other people participating in the study will increase the understanding of how diet is involved in both the onset of diabetes and the later complications. If we do find that foods or patterns of eating are associated with diabetes, this new information may be helpful in preventing other people from getting diabetes or in helping people with diabetes to prevent the complications that may occur."

"First I would like you to tell me what you ate yesterday. It is important that you tell me everything you ate or drank including beer, wine, mixed drinks, and vitamins or pills. We are also interested in what you add to your foods like butter, gravy, sauces, including chile sauce and also dry chile (show chile models). We want to know what you really ate. So don't worry about what you think you should've eaten."

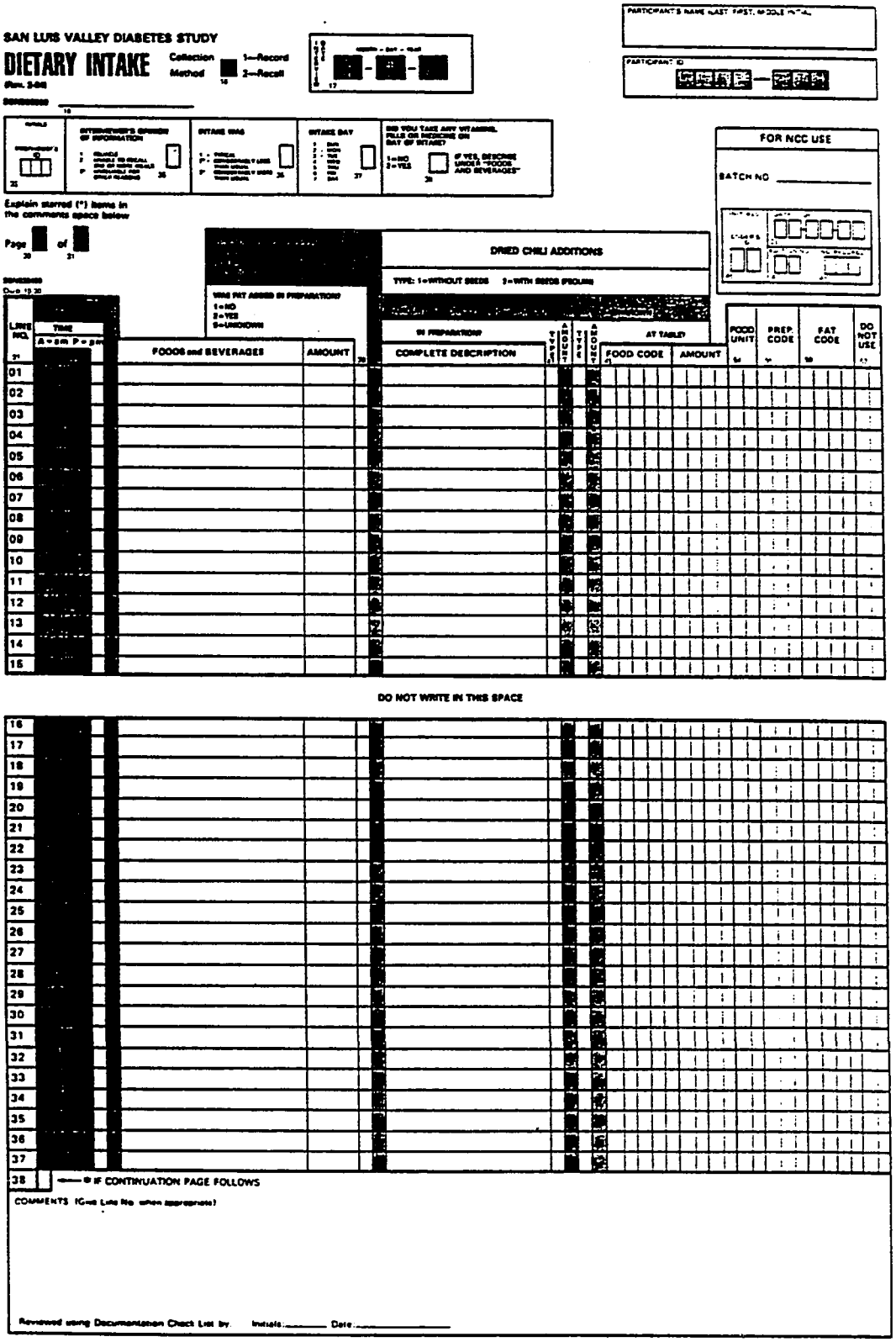
"Because yesterday you had to fast for the clinic, let's start with after your dinner two days ago. That would be..... So .... night did you have anything to eat, like snacks or drinks, between dinner and bedtime?

[wait for response and then probe for description and amounts]

"Now tell me about yesterday, starting with the first thing you had to eat after you got up and continuing until you started to fast last night."

[wait until the participant goes through the entire day and then go back and probe for descriptions and amounts]

Revision 5 5/8/85



SAN LUIS VALLEY DIABETES STUDY

DIETARY INTAKE

Collection Method: 1—Record, 2—Recall

PARTICIPANT'S NAME (LAST, FIRST, MIDDLE INITIAL)

PARTICIPANT ID

FOR NCC USE

BATCH NO

Explain starred (*) items in the comments space below

Page ___ of ___

DRIED CHILI ADDITIONS

TYPE: 1=WITHOUT SEEDS 2=WITH SEEDS (FIGURE)

LINE NO.	TIME A=am P=pm	FOODS and BEVERAGES	AMOUNT	COMPLETE DESCRIPTION (IN PREPARATION)	RECIPE	AT TABLE RECIPE	FOOD CODE (AT TABLE)	AMOUNT (AT TABLE)	FOOD UNIT	PREP CODE	FAT CODE	DO NOT USE
01												
02												
03												
04												
05												
06												
07												
08												
09												
10												
11												
12												
13												
14												
15												

DO NOT WRITE IN THIS SPACE

16												
17												
18												
19												
20												
21												
22												
23												
24												
25												
26												
27												
28												
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31												
32												
33												
34												
35												
36												
37												

38 — 9 IF CONTINUATION PAGE FOLLOWS

COMMENTS (Give Line No. when appropriate)

Reviewed using Documentation Check List by: Initials:_____ Date:_____

APPENDIX G: Food Frequency Questionnaire (FFQ)

San Luis Valley Diabetes Study
Food Frequency Interview
Form D2

"Now I'd like to ask you how <u>often</u> you eat certain foods items. For each food I'll ask how often you've eaten that food at meals or as a snack during the past month. And second, I'll ask how often you ate that food _____ years ago."

"Many people change the way they eat over the years. We realize it is difficult to remember what you ate a long time ago but it is important that you try to remember as well as possible. The next few questions are intended to help you better recall what you were eating at that time. First, I would like you to think about where you were living .... years ago. [pause]

Can you tell me where you were living? _____

Who were you living with at the time? _____

Were you in school? [ ]no [ ]yes

Did you have children in school? [ ]no [ ]yes

Were you working away from home at that time?

[ ]no [ ]yes, part-time [ ]yes, full-time [ ]yes, on-the-road

Do you remember who cooked most of the meals? _____

Where would you say you ate most of your meals? _____

"Now keeping that time in mind, ... years ago, let's go through each food. First I'll ask how often you ate it during the past month and then .... years ago. Some foods like fruits and vegetables, holiday foods and wild game may only be eaten during certain parts of the year. For those foods, think of the season we're in now, that would be _____ "

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Form D2, Page 2

Participant ID ☐☐☐ ☐☐☐

START TIME __ __ : __ __

"How often have you eaten ... at meals or as a snack, during the past month?"

"Now thinking back, how often did you eat ... ☐☐ years ago?"

FOOD	PAST MONTH: No. of times	Per: day	wk	mo	PRIOR INTAKE: SAME	If not the same: No. of times	Per: day	wk	mo	<1 mo
1. apples	__	2	3	4	1	__	2	3	4	5
2. bananas	__	2	3	4	1	__	2	3	4	5
3. other fresh fruit (list)	__	2	3	4	1	__	2	3	4	5
4. dried fruit (raisins, dates, figs, prunes)	__	2	3	4	1	__	2	3	4	5
5. avocados and guacamole	__	2	3	4	1	__	2	3	4	5
6. nuts and seeds (peanut butter, pinon, all)	__	2	3	4	1	__	2	3	4	5
7. corn, chicos, hominy, or posole	__	2	3	4	1	__	2	3	4	5
8. dried peas and beans (pintos, lima, lentils)	__	2	3	4	1	__	2	3	4	5
9. yams, sweet potatoes, pumpkin, winter squash	__	2	3	4	1	__	2	3	4	5
10. carrots	__	2	3	4	1	__	2	3	4	5
11. broccoli	__	2	3	4	1	__	2	3	4	5
12. green peas	__	2	3	4	1	__	2	3	4	5
13. lettuce or tossed salad	__	2	3	4	1	__	2	3	4	5
14. spinach, greens, verdolagos	__	2	3	4	1	__	2	3	4	5

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Form D2, Page 3

FOOD	PAST MONTH: No. of times	Per: day	wk	mo	PRIOR INTAKE: SAME	If not the same: No. of times	Per: day	wk	mo	<1 mo
15. other vegetables (list)	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
16. potatoes	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
Do you usually eat your potatoes: (if both equally, check both)	fried 1☐ or	boiled, baked or mashed 2☐?			fried 1☐ or	boiled, baked or mashed 2☐?				
17. rice, macaroni, noodles	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
18. corn tortillas or cornbread	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
19. white bread, rolls or tortillas	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
20. whole wheat bread, rolls tortillas	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
21. cold breakfast cereal	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
What kinds do you eat most often (in order of most often eaten)	1. _____ ☐ 2. _____ ☐					1. _____ ☐ 2. _____ ☐				
22. hot cereal	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
What kinds do you eat most often (in order of most often eaten)	1. _____ ☐ 2. _____ ☐					1. _____ ☐ 2. _____ ☐				
23. crackers or chips	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
24. pizza	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
25. enchiladas, tamales, tacos, tostadas, burritos	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐
26. vegetable soups/stews with meat	__	2☐	3☐	4☐	1☐	__	2☐	3☐	4☐	5☐

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Form D2, Page 4

FOOD	PAST MONTH: No. of times	Per: day	wk	mo	PRIOR INTAKE: SAME	If not the same: No. of times	Per: day	wk	mo	<1 mo
27. How often do you add green chile to foods or eat foods prepared with it?	__	2	3	4	1	__	2	3	4	5
28. How often do you add red chile to foods or eat foods prepared with it?	__	2	3	4	1	__	2	3	4	5
29. cold cuts (potted meats, liverwurst, franks)	__	2	3	4	1	__	2	3	4	5
30. game (venison, elk, rabbit)	__	2	3	4	1	__	2	3	4	5
31. pork (fresh pork, ham, bacon, sausage)	__	2	3	4	1	__	2	3	4	5
32. beef, veal or lamb	__	2	3	4	1	__	2	3	4	5
33. poultry (chicken, turkey, wild fowl, cold cuts)	__	2	3	4	1	__	2	3	4	5
34. fish or shellfish	__	2	3	4	1	__	2	3	4	5
35. eggs	__	2	3	4	1	__	2	3	4	5
36. cheese (cheddar, swiss, velveeta, jack...)	__	2	3	4	1	__	2	3	4	5
37. whole milk including on cereal but not in coffee	__	2	3	4	1	__	2	3	4	5
38. 2% or skim milk	__	2	3	4	1	__	2	3	4	5
39. butter or margarine	__	2	3	4	1	__	2	3	4	5
40. salad dressing or mayonnaise	__	2	3	4	1	__	2	3	4	5
41. ice cream	__	2	3	4	1	__	2	3	4	5
42. cookies, cakes or pies	__	2	3	4	1	__	2	3	4	5

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Form D2, Page 5

FOOD	PAST MONTH: No. of times	PAST MONTH: Per day	PAST MONTH: wk	PAST MONTH: mo	PRIOR INTAKE: SAME	PRIOR INTAKE: If not the same: No. of times	Per day	wk	mo	<1 mo
43. empanadas, sopapillas or doughnuts	__	2	3	4	1	__	2	3	4	5
44. chocolate candy	__	2	3	4	1	__	2	3	4	5
45. caffeinated coffee or tea	__	2	3	4	1	__	2	3	4	5
46. caffeinated cola beverages	__	2	3	4	1	__	2	3	4	5

Now I want to ask you a few questions about your use of alcoholic beverages.

(ASK Q.47 FOR BOTH TIME PERIODS)

47. Did you drink beer, wine, mixed drinks or distilled spirits during the past YEAR? 1 no 2 yes ... years ago? 1 no 2 yes

(IF YES, CONTINUE IN YES COLUMNS. IF NO TO BOTH, SKIP TO Q. 60)

ever? 3 skip

	PAST YEAR: About how OFTEN did you drink ..., in the past year? Number	Per day	wk	mo	yr	PRIOR INTAKE: ... years ago, how OFTEN did you drink...? SAME	If not the same: Number	Per day	wk	mo	yr
48. beer	__	2	3	4	5	1	__	2	3	4	5
49. wine	__	2	3	4	5	1	__	2	3	4	5
50. mixed drinks or distilled spirits	__	2	3	4	5	1	__	2	3	4	5

(continued on next page)

(continued on next page)

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Form D2, Page 6

	PAST YEAR	PRIOR INTAKE
	How MUCH do you usually drink at one time, when you drink ... ?	... years ago, how MUCH did you usually drink?
	Number	Number
51. beer	__ __ cans	__ __ cans
52. wine	__ __ glasses	__ __ glasses
53. mixed drinks or distilled spirits	__ __ drinks	__ __ drinks
People will sometimes drink MORE THAN THEIR USUAL. Thinking about occasions when you do drink more than your usual,	what is the MOST you would drink at one time of ... ?	... years ago, what would have been the MOST?
	Number	Number
54. beer	__ __ cans	__ __ cans
55. wine	__ __ glasses	__ __ glasses
56. mixed drinks or distilled spirits	__ __ drinks	__ __ drinks
	About how OFTEN do you drink this much...?	... years ago, how OFTEN did you drink this much ... ?
		If not the same:
	Number / Per: day wk mo yr	SAME / Number / Per: day wk mo yr
57. beer	__ __ 2 3 4 5	1 __ __ 2 3 4 5
58. wine	__ __ 2 3 4 5	1 __ __ 2 3 4 5
59. mixed drinks or distilled spirits	__ __ 2 3 4 5	1 __ __ 2 3 4 5

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Form D2, Page 7

The next few questions are about the types of fat used in preparing the food you eat.

60. People use different types of fat in baking, frying and at the table. Can you tell me what kind of fat you (or your spouse) use MOST often for:
(Emphasize most often and try to get respondent to pick one. If the respondent uses several types equally, then check all that apply.)

baking	frying	at the table	
1[ ]	1[ ]		lard or other meat fat
2[ ]	2[ ]		vegetable shortening (solid)
3[ ]	3[ ]	3[ ]	butter
4[ ]	4[ ]	4[ ]	tub margarine or squeeze bottle
5[ ]	5[ ]	5[ ]	stick margarine
6[ ]	6[ ]	6[ ]	oil
7[ ]	.....		other fat _____
	7[ ]	.....	other fat _____
		7[ ]..	other fat _____
8[ ]	8[ ]	8[ ]	don't use fat or does not bake
9[ ]	9[ ]	9[ ]	don't know what type is used

61. If you (or your spouse) use cooking oil, what type do you MOST often use?

1[ ] coconut, palm, or MCT
2[ ] olive oil
3[ ] sunflower or safflower oil
4[ ] other vegetable oil (corn, sesame, soybean, wheat germ, blended,...)
8[ ] none
9[ ] don't know

62. What type of salad dressing do you MOST often use?
(Emphasize most often and try to get respondent to pick one. If the respondent uses several types equally, then check all that apply.)

1[ ] don't use salad dressings
2[ ] clear dressings like Italian
3[ ] blue cheese dressing
4[ ] other creamy dressings like thousand island or ranch
5[ ] lo cal dressings of any type
6[ ] other _____

63. What do you do with the visible fat on your meat?

1[ ] eat most of it
2[ ] eat some of it
3[ ] eat as little as possible
4[ ] do not eat meat

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Form D2, Page 8

64. Have you had an illness which caused you to permanently change your diet?

1[ ]no → THANK YOU (end of interview)

2[ ]yes ↓

65. If yes, what type of illness was it?

1[ ] diabetes
2[ ] cancer
3[ ] hypertension or heart related disease
4[ ] other, specify _____

66. When did you make this change in your diet? ____|____ month year

Thinking about this change, would you say you now eat more, less or about the same of the following:

	more	less	no change	don't understand
67. fat	1[ ]	2[ ]	3[ ]	4[ ]
68. cholesterol	1[ ]	2[ ]	3[ ]	4[ ]
69. fiber	1[ ]	2[ ]	3[ ]	4[ ]
70. starchy foods	1[ ]	2[ ]	3[ ]	4[ ]
71. sugar	1[ ]	2[ ]	3[ ]	4[ ]
72. salt	1[ ]	2[ ]	3[ ]	4[ ]
80. other, specify: _____	1[ ]	change	3[ ]	4[ ]

THANK YOU VERY MUCH FOR TAKING THE TIME TO PARTICIPATE IN THIS INTERVIEW

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Form D2, Page 9

INTERVIEWER DATA - COMPLETE AT CONCLUSION OF INTERVIEW

ID: __ __ FINISH TIME __ __ : __ __

1. Do you feel that the information provided by the subject was satisfactory?
1[ ]no 2[ ]yes if no, explain:

2. Please circle the number that best describes the subject's awareness level during the interview.

0 ...	1------2------3------4------5	
not observed	very alert	very confused

COMMENTS: 1[ ]no 2[ ]yes

Edit ID: __ __

Date: __|__|__

Computer Entry ID: __ __

Date: __|__|__

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APPENDIX H: FFQs Reviewed from Other Studies

In developing the SLVDS food frequency questionnaire, forms from the following studies were reviewed to determine what questions were asked (frequency, serving size, change over time), whether frequency was open ended or precategorized, individual foods and food groups included, use of serving size in the description, time period of coverage, and approaches to seasonality.

1. National Health and Nutrition Examination Surveys I and II
2. Sample interviews by Abramson et al (171)
3. San Antonio Heart Study
4. New Mexico Community Health Survey
5. Diabetes Control and Complications Trial (Form 029.1)
6. Channing Laboratories Nurses Study (178)
7. Women's Health Study, National Cancer Institute
8. Roswell Park's brief questionnaire to obtain indices of monthly intakes of nutrients (fiber, fat, vitamin A, vitamin C, vitamin E)
9. Jean Brender's dissertation questionnaire, University of Washington (fiber)
10. Joan Karkec's Food Intake Frequency, University of Washington (all nutrients)
11. Hypertension Study, Oregon Health Sciences Center (calcium)
12. Jim Mann's simplified questionnaire (dietary fiber) (29)

APPENDIX I: FFQ Formatting Issues

Food frequency questionnaires (FFQ) in other studies have used a variety of formats. Minimal data existed on the effects that these varying formats might have on study findings when the SLVDS FFQ was developed. Several of these issues are discussed below.

Individual versus grouped food items. It is reasonable that a subject could more accurately recall intake of an individual food item than intake of a grouping of foods. A grouping is more complex and subunits may more easily be omitted by the subject. However, due to the large number of foods containing fat and fiber, and the lack of knowledge on indicator foods, it was necessary to include many food items in order to adequately assess an individuals intake of these nutrients. The additional accuracy which might be obtained by individual food items had to be balanced against the accuracy lost with an increasingly lengthy interview. A compromise utilizing both individual food items, for more important nutrient sources or items that weren't readily associated (either due to context or nutrient content), and food groups was employed.

Portion size. Portion size was included on the pretest version of the SLVDS FFQ. However, including portion size, which often required a volume model for reference, was found to be cumbersome and to lengthen the interview. In addition, discrimination among individuals had been suggested to be more dependent on frequency than portion size. Portion size was omitted. SLVDS 24-hour diet recall data could potentially be reviewed for systematic differences in portion size across subgroups of interest.

Measure of frequency. The measure of frequency was open ended. This method was chosen in order to obtain a continuous measure of frequency. Experience collecting data in the SLVDS suggested that participants may have more easily responded to rough categories of frequency rather than having to give a specific (i.e. more exact) frequency.

Time period of inquiry. In order to assess diet at a time relevant to disease etiology, it was necessary to ask people about their past diet. A number of studies evaluating the accuracy of retrospective recall of diet have recently been published and are discussed in Chapter 4.

The past 30 days was chosen, instead of the past year, as a time period participants could more accurately average over. This seemed especially true when seasonal variation in the variety and frequency of foods eaten was considered.

Seasonality. Some foods are eaten more or less at different times during the year. It is possible that important differences in dietary patterns may be more evident in one season than another (i.e. fruit and vegetable intake may be comparable by ethnic group or disease status during the summer but may quite different during the winter). The 30-day food frequency allowed us to look for these differences.

APPENDIX J: SLVDS Diet Protocol Manual

1.0 General Information

The dietary interview consists of two components:

1. the 24-hour dietary recall - **Form D1** and
2. the food frequency interview - **Form D2.**

The **24-hour dietary recall** interview asks the participant to remember everything he/she ate or drank over the 24-hour period prior to starting to fast for the clinic visit. Recognizing that an individual's intake may vary from day-to-day, this data will be averaged over many individuals to estimate mean nutrient intakes for comparison between subgroups within the San Luis Valley Diabetes Study (SLVDS) and for comparison with other populations that have been studied. Variables of particular interest are percent of calories from carbohydrate and fat, the polyunsaturated to saturated fat ratio, percent of total fat intake from animal sources, mg of cholesterol, percent of carbohydrate intake as complex versus simple, gm dietary fiber, caffeine and alcohol. The 24-hour recall should take between 20 and 30 minutes of interviewing time depending on the subject and their diet. The Nutrition Coding Center (NCC) at the University of Minnesota has been contracted to code and process the 24-hour recall data and to train interviewers for collecting the recalls.

The **food frequency** interview asks the participants to estimate how many times in the past month they have eaten each food listed and how many times they ate it during a defined time in the past. Foods have been included based on their estimated contribution to dietary fiber, fat, caffeine and alcohol intake in the SLV population. Asking prior intake is important in order to investigate factors which may have led to the development of diabetes and heart disease. The food frequency requires about 20 minutes of interviewing time to complete.

1.1 Training Procedures

Training of the interviewers to administer the **24-hour recall** included:

- a two day training session by the NCC,
- NCC certification,
- supplemental training by the SLVDS,
- development of an expanded documentation checklist,
- video taping of interviews, and
- continuing education from the NCC.

These procedures are described below.

NCC training sessions - The basis of the initial training of clinic staff was a two-day training session January 23 and 24, 1984, directed by Joyce Wenz from the NCC. The session included an introduction to the NCC system with particular emphasis on good interviewing and documentation techniques. Trainees were sent materials ahead of time. This pretraining packet provided minimal acquaintance

with the system and coding exercises so that there could be optimal use of the time at the session. Coding, interviewing and documentation were covered.

There was an in-depth discussion of the NCC Codebook, the dietary collection forms and special forms. Trainees were taught the basics of coding and given practice coding menus. The purpose of this part of the session was to give trainees an appreciation of the type of documentation required for adequate data collection.

Time was spent in discussing the research interview. The principles of good interviewing were taught with the aid of a video tape prepared for this use. The video tape contained examples of both good and poor interviewing techniques which were discussed in the session. Trainees conducted a practice interview.

A major item on the agenda was discussion of the importance of good documentation to the gathering of high quality data. The goal of the sessions and the entire NCC education effort is to instill in the trainees good documentation skills within the confines of the research interview.

NCC Certification - It is the policy of NCC that only certified interviewers collect dietary data for coding. The requirements of certification are:

- attendance at the NCC training session described above,
- coding of six standard recalls with no more than nine errors in the set, and
- collection and coding of 10 recalls from study-similar subjects.

Supplemental training session by the SLVDS - A training session supplemental to the NCC training was held in mid-February to integrate SLVDS goals with the NCC training and to reinforce what was introduced at the NCC training. This session addressed the following issues:

- why is the SLVDS asking people about what they eat?
- why are we using a 24-hour recall?
- what happens between coding and data analysis?
- how can practice coding help?
- how will we estimate portion size in the SLVDS?
- how do we use the SLVDS 24-hour recall form?

This session also included review of feedback from NCC on coding of the six standard recalls for certification and practice interviews.

Documentation Checklist - In response to interviewer questions on the type and amount of detail required for proper documentation of the 24-hour recall, the interviewers worked with our consulting nutritionist, Marsha Jacobs, to develop an extended documentation checklist (Figure J.3). This was useful for training since interviewers had to question how items were classified, what specification or description was needed and the acceptable units for amount. The NCC codebook was used to answer questions whenever possible. The checklist developed was reviewed by the NCC for corrections and additions. The final checklist serves as a reference for interviewers when questions arise during an interview or in reviewing each others recalls following the interview.

Video taping - In April, 1984, after pilot interviews began, SLVDS staff came to Denver for further training. At this time, interviews were videotaped and rerun to examine interviewing style.

Continuing Education from the NCC - The continuing education program is designed to keep field interviewers current with new developments in the NCC system and to correct any inappropriate practices, especially in recall documentation. The NCC Communique is a newsletter sent to all studies bimonthly. It discusses actual or potential problems in dietary documentation, NCC changes in procedure or personnel and other points which may be of interest to the study staff such as system modifications. Individual problems that a particular interviewer has on a particular recall are addressed through the Inquiry. NCC will send an Inquiry to call attention to specific problems to try to prevent their recurrence.

Training of interviewers to administer the **food frequency** interview was addressed in the supplemental training where the interview format was presented and video taping for interview style mentioned above. The food frequency interview is a much more structured interview than the 24-hour recall and consequently less training was required. In the pilot, completed questionaires were reviewed to check for correct documentation of data, problems were solicited from the interviewers experiences and discussed.

1.2 Administration and Tracking Procedures

The **24-hour recall** is administered to the participant first, followed by the food frequency. Several references are recommended for frequent review to maintain good documentation and consistent probing in the 24-hour recall interview. These references include:

1. the Nutrition Coding Center (NCC) Documentation Checklist,
2. the SLVDS (extended) Documentation Checklist - Form D5,
3. the first section of the NCC codebook, pages 1-13,
4. the Food Portion Visual Manual, pages 7-11, 16-19, 30-33,
5. the NCC's Continuing Education Communiques, and
6. the logbook of documentation question and answers - Form D6.

and are available in the clinic dietary interview protocol manual.

It is important to emphasize at the beginning of the interview, the following paragragh from the inside front cover of the dietary interview.

> "I would like you to tell me what you ate yesterday. It is important that you tell me everything you ate or drank including beer, wine, mixed drinks, and vitamins and pills. We are also interested in what you add to your foods like butter, gravy, sauces, including chile sauce and also dry chile (show chile models). We want to know what you really ate. So don't worry about what you think you should've eaten."

Two types of aides are available to assist the participant in estimating portion sizes during the 24-hour recall. The primary reference is the two dimensional visual aid called a Food Portion Visual (FPV) developed by Boston Nutrition Associates. The FPV is poster size. It has an "A" side with shapes representing volumes and a "B" side with shapes and thicknesses for calculating weights. The FPV is described in detail in the FPV Manual. Three dimensional aides are available to estimate portion size if the participant has trouble relating to the two dimensional FPV. These three dimensional aides include:

- a 12 inch ruler
- two plates (10" and 7"), erasable pens, and a sponge for drawing dimensions of items like meat
- two bowls (1 3/4 cup low, flat bowl and 2 cup rounded bowl)
- three glasses (6 oz, 10 oz, 16 oz)
- one tea cup and one mug
- measuring spoons (1 TB, 1 TS, 1/2 TS, 1/4 TS)
- butter pat (1 teaspoon each)
- pinto beans for measuring "handfuls" of nuts, etc.
- three dimensional thickness measure

Following the dietary interview, the 24-hour recall should be reviewed carefully with the NCC and SLVDS documentation checklists. Following self-review, a second interviewer should also carefully review the recall and sign their initials at the bottom of the form. A two-part tracking form, Form D4 (Figure J.2), is to be filled out as recalls are completed each day. The first part of Form D4 is for SLVDS records of the current status of each recall. The second part is a replica of NCC's packing list and should be included with each batch of recalls sent to NCC. Recalls are to be mailed in batches of no less than 25 to:

ATTN: Data Flow
Nutrition Coding Center
2829 University Avenue, S.E.
Suite 526
Minneapolis, Minnesota 55414

Recalls which require additional documentation are returned to the SLVDS as an inquiry. These inquiries are addressed to Field coordinator who then distributes them to the appropriate interviewer. NCC requires a response within one week of receipt, otherwise a coding decision will be made at the NCC, and the intake form will be processed. NCC applies an additional charge if more than 5% of dietary intake forms require inquiries. The NCC will return nutrient summaries to the SLVDS for each participant's 24-hour dietary recall.

The **food frequency** requires assignment of prior intake time before the interview begins. Because we are studying existing cases of diabetes, time since diagnosis will vary from in the past year to 20 or 30 years ago. As stated in the previous section we are interested in learning about what people ate before they developed their disease. We also need to ask controls about a comparable time in the past. Assignment of prior intake time should be done for each parti-

cipant when their packets are assembled. For diabetics, prior times should be transcribed from the alphabetical listing of diabetics provided. For diabetics whose medical record did not contain information on date of diagnosis, a question about date of diagnosis is asked in the registration interview. Years since diagnosis plus one year is calculated and written onto the front of the frequency interview. For controls, the next prior time on the list (form D3 - Figure J.3) for the appropriate age group should be chosen and entered on the cover of the food frequency when packets are assembled.

Probably the most difficult part of the food frequency is helping the participant recall intake at the assigned time in the past. The 24-hour recall helps get the participant to think about food in general and questions are asked at the beginning of the food frequency to help orient them to the appropriate time in the past. The participants may need encouragement to estimate prior frequencies. This may be accomplished by letting them know this information is important and that we will be looking at whether a lot or a little is reported and we are less concerned with the exact numbers. When the participant can not remember and you have tried to encourage them about the importance of this information as stated above, then ask "Do you ever eat ... e.g. apples?" If they respond "yes", then ask how often again.

When the participant responds with a large number like 30 times per week. Ask them how many times per day. Using a smaller time interval like day versus month for foods eaten frequently should provide more accurate information.

It is important that both the "No. of times" and the interval (per day, week, month) be completed by the interviewer. At the end of the interview, before the participant leaves the room, check to make sure that all items in the food list are complete. The interviewer is encouraged to include anecdotal comments on page 9 of the questionaire when the participant volunteers additional information.

CODING RULES:

1. INTERVIEWER: If "No. of times" is "0", then leave the interval blank.
2. INTERVIEWER: Use "99" for "No. of times" when the participant can't remember a few items, but remembers the rest. Leave all fields blank if the participant can't remember prior time at all.
3. EDITER: If the interviewer completed
   - "no. of times" but the interval is blank, then write a "9" to the right of the month box.
   - interval but "No. of times" is blank, then write "99" as "No. of times".

   When possible, if the participant is still in the clinic, ask the participant to recall this information.

Editing of the food frequency by another staff member should include:

-verifying that all data items have been completed and are readable,
-scanning the food frequency to be sure
    1. a time interval (per day, week, or month) is checked whenever "No. of times" is not zero,
    2. "same" and "no. of times" are not both marked under prior intake,
-verifying that the appropriate skips have been made following question 47. on alcoholic beverages.

1.3 Forms D1-D7

Diet Protocol Forms

San Luis Valley Diabetes Study

Control Assignment of Prior Intake Time
for Food Frequency Questionaire
Form D3

Controls age 20 thru 44

Prior No. of years	Age	ID No.	Prior No. of years	Age	ID No.	Prior No. of years	Age	ID No.
10	35	GAL	13			14		
3	37	STE	4			2		
4	43	BUR	7			31		
5	44	MAR	9			9		
5	40	LUJ	16			11		
3	37	MAR	14			14		
7	40	OLI	1			23		
4			6			20		
1			11			8		
6			12			1		
22			14			22		
8			1					

Controls age 45 thru 74

Prior No. of years	Age	ID No.	Prior No. of years	Age	ID No.	Prior No. of years	Age	ID No.
2			4			4		
22			2			29		
12			11			4		
11			15			2		
13			7			3		
13			11			12		
1			1			2		
14			3			7		
2			12			6		
1			6			10		
1			3			8		
1			16			2		
12			3			1		
9			4			5		
3			1			2		
1			3			18		
2			9			15		
10			3			2		
12			1			1		
12			10			6		
11			13			11		
3			12			8		
2			3			11		
1			19			13		

Revision 1 7/2/84

Figure J.1. Prior intake assignment forms

Figure J.1. (continued)

San Luis Valley Diabetes Study

Control Assignment of Prior Intake Time
for Food Frequency Questionaire
Form D3

Controls age 45 thru 74 (continued)

Prior No. of years	Age	ID No.	Prior No. of years	Age	ID No.	Prior No. of years	Age	ID No.
15			17			4		
3			5			8		
2			23			6		
2			10			9		
2			23			8		
49			16			1		
10			30			17		
9			12			40		
4			4			2		
6			2			8		
2			15			12		
5			7			4		
4			1			1		
4			3			1		
10			1			3		
9			31					
6			3					
2			6					
6			10					
14			6					

Revision 1 7/2/84

San Luis Valley Diabetes Study
24-Hour Recall Tracking Form and NCC Packing List

Batch No. _____
No. of Pages _____
Total No. of Records _____

Date sent to NCC __|__|__ Postcard of receipt from NCC __|__|__

List each participant I.D. number and date of visit		1st response OK	1st response Inquiry	Final response OK	Final response date
1. _____	_____	[ ]	[ ]	[ ]	__|__|__
2. _____	_____	[ ]	[ ]	[ ]	__|__|__
3. _____	_____	[ ]	[ ]	[ ]	__|__|__
4. _____	_____	[ ]	[ ]	[ ]	__|__|__
5. _____	_____	[ ]	[ ]	[ ]	__|__|__
6. _____	_____	[ ]	[ ]	[ ]	__|__|__
7. _____	_____	[ ]	[ ]	[ ]	__|__|__
8. _____	_____	[ ]	[ ]	[ ]	__|__|__
9. _____	_____	[ ]	[ ]	[ ]	__|__|__
10. _____	_____	[ ]	[ ]	[ ]	__|__|__
11. _____	_____	[ ]	[ ]	[ ]	__|__|__
12. _____	_____	[ ]	[ ]	[ ]	__|__|__
13. _____	_____	[ ]	[ ]	[ ]	__|__|__
14. _____	_____	[ ]	[ ]	[ ]	__|__|__
15. _____	_____	[ ]	[ ]	[ ]	__|__|__
16. _____	_____	[ ]	[ ]	[ ]	__|__|__
17. _____	_____	[ ]	[ ]	[ ]	__|__|__
18. _____	_____	[ ]	[ ]	[ ]	__|__|__
19. _____	_____	[ ]	[ ]	[ ]	__|__|__
20. _____	_____	[ ]	[ ]	[ ]	__|__|__
21. _____	_____	[ ]	[ ]	[ ]	__|__|__
22. _____	_____	[ ]	[ ]	[ ]	__|__|__
23. _____	_____	[ ]	[ ]	[ ]	__|__|__
24. _____	_____	[ ]	[ ]	[ ]	__|__|__
25. _____	_____	[ ]	[ ]	[ ]	__|__|__

Total number listed on this page _____

Form D5
Revision 2:4/25/84

Figure J.2. 24-hour diet recall tracking forms

San Luis Valley Diabetes Study
24-hour Dietary Recall
Documentation Checklist
Form D5

CONTENTS Page

NOTE: UNKNOWN INFORMATION PLEASE DOCUMENT AS SUCH

Revision 4:7/25/84

Figure J.3. 24-hour diet recall documentation checklist

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
DAIRY PRODUCT		
Cheese	type of cheese? processed or natural? brand name if processed	
	shredded/grated	cups, tablespoons A side
	sliced/chunk	3-dimensions in inches, B side
	cheese spreads	tablespoons A side
	cottage cheese % fat	cups, tablespoons A side
	cream cheese % fat, flavor	3-dimensions B side tablespoons, A side
Milk	% fat	cups, A side
Milk shake	ice cream, ice milk or fast food? Choc. or other?	cups, A side
Flavored milk	kind of milk % fat, flavoring	cups, A side
Ice cream	Brand and/or % fat flavor if choc. or coffee	cups, A side
Ice milk		cups, A side
Sherbet	brand	cups, A side
Ice cream sandwich or bar	kind	3-dimensions B side

NOTE: Unknown information, please document as such.

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
DAIRY PRODUCT		
Yogurt	%fat, brand, flavored or plain	cups, fluid oz. A side
Cream	heavy, light, or half & half?	tablespoons A side
Whipping cream	aerosol? sweetened? dairy or non-dairy? brand name?	tablespoons A side
Sour cream	light, heavy, % fat if dairy brand name if imitation	tablespoons A side
Creamer	dairy or non-dairy? liquid or powder? brand name if non-dairy	tablespoon teaspoon A side
Dips	sour cream cottage cheese	tablespoons A side
Beverages mixes	brand name protein mix, cocoa mix, Ovaltine, milk % fat	amount of mix 1 pkg, tablespoons amount of milk cups, A side 1 pkg
Hot chocolate	brand or scratch water or milk % fat milk	cups A side
Eggs	preparation? type of fat? milk added? chile added? egg substitute? (brand name)	number of eggs cups, A side

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
FRUIT and JUICES		
	cooked, fresh, canned, frozen, dried	cups, B side diameter sm, med, large
	sweetened? unsweetened?	for canned fruit - amt liquid drunk (i.e. 1/2 canned peach & 1/4 cup juice)

NOTE: Unknown information, please document as such.

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
VEGETABLES	cooked or raw? canned, fresh or frozen? sauces added? kind of fat added?	cups, tablespoons A side dimensions 3-D, for stick vegetables use ruler
Potatoes	skin eaten? additions in preparation? (fat, cheese) gravy or fat added at table? Chile added?	cups, A side # of potatoes sm, med, or large
GRAVIES AND SAUCES		
Gravy	water, milk or cream, % fat milk fat type	tablespoons, cups A side
Sauces	white sauce - milk, % fat, type of fat, scratch or commercial?	tablespoons, cups A side
	Italian spaghetti - w/or w/out meat kind of meat, scratch, (fat?)mix	

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
MEATS		
	type-pork, beef, veal, lamb, etc. type of cut, fat trimmed? bone in? how cooked? % fat hamburger? fat added? Kind?	dimensions B side 3-D draw it & ms. in inches
Poultry	piece or parts eaten? skin eaten? how prepared? fat if used? dark or light meat?	B side (slices) standard servings (drumstick, etc.)
Fish	kind, how prepared breaded? battered? fat if used? oil or water packed?	B side or inches 3 dimensions cups
Shellfish	how prepared? fat if used?	size and number eaten, cups
Bacon		1 sl. if regular, otherwise dimensions
Sausage	patty or link? pork or beef? how cooked?	dimensions B side
Cold cuts	type of meat? brand	3 dimensions B side
Ground meat	type of meat? % fat, how cooked?	dimensions if patty B side cups, A side

NOTE: Unknown information, please document as such.

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
MEATS (continued):		
Hamburger	% fat bun? type? relish or other condiments?	dimensions (diameter unless McDonalds or Burger King)
Hot dog	kind? brand? relish or other condiments? bun? type?	standard, jumbo
BREADS	kind? principal fat? fat added at table?	average slice 3 dimensions
Rolls & Buns	type - pan, hard, sweet, etc. kind- white, whole wheat, rye, etc. principal fat? fat added at table? topping, frosting or glaze?	3 dimensions B side
Cereals	kind? brand? anything added, milk, sugar, raisins	cups, A side
Muffins	additions, mix or scratch, store bought? kind of fat used?	3-D diameter

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
CASSEROLES: 1) give a common name, e.g. beef stew, tamale pie, chicken chow mein etc. or 2) describe listing main ingredients & proportions or 3) use recipe specification form for regional or ethnic foods not in NCC Codebook.		
	meat or fish % fat meat, milk % fat, tomatoes, kind of soup added, cooking method? proportion of ingredients?	cups, A side
SALADS	w/egg, type of dressing? macaroni added?	cups, A side
SOUPS	milk or water? if milk, % fat? thick or chunky? dilution diff tomatoes?	cups, A side
DESSERTS	specify type of fat used in preparation	
Pies	1 or 2 crusts? filling type? scratch, mix or comm'l for crust & filling	draw the dimensions A side
Cakes	scratch or commercial? price bakery frosting	A or B side
Cookies	scratch or commercial, frosting, price range	diameter, inches B side
Cobblers	biscuit or piecrust, scratch	dimensions, inches B side, cups

NOTE: Unknown information, please document as such.

Figure J.3. (continued)

Food item	Specification Needed	Acceptable Units for amount
DESSERTS (continued)		
Donuts, longjohns	frosting, filling yeast or cake type	3-D, inches B side
Fruit crisp		cups, A side
CRACKERS	type & brand	number of crackers 2-D, B side if type and brand unknown
SNACK FOODS	kind & brand	cups, A side
BEVERAGES	sweetened? unsweetened? brand?	cups, A side fluid oz.
	with or without caffeine? sodium free? proportion ice?	
Koolaid	sugar? nutra-sweet or saccharin?	
Tea	herbal tea decaf, reg? sugar added, lemon	
Beer	regular or low cal brand name	
Wine, liquor, mixed drinks, liqueur	type of wine - table or dessert, flavor of liqueur	

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
MEXICAN DISHES - specify type of fat		
Beans	pinto? refried? with or without fat? kind of fat?	Give only amount of beans cups, A side
Burritos	beef, pork or chicken? chile? type? cheese?	length in inches amounts on filling, sauce, and addition
Calabacitas	cheese added? chile added?	cups, A side
Chile	red or green? sauce or salsa? with meat? kind?	cups, A side
Chicos or posole	anything added at table?	cups, A side
Enchiladas	filling? type? which sauce fried? kind of fat?	number of enchiladas
Empanadas	type of filling? type of fat fried in?	3 dimensions
Menudo	anything added at table?	cups, A side

NOTE: Unknown information, please document as such.

Figure J.3. (continued)

Food Item	Specification Needed	Acceptable Units for amount
Relleno	with meat?	length in inches
Spanish rice	green pepper? fried in what? any meat?	cups A side
Spaghetti, macaroni,	fried in what? with meat? kind?	cups, A side
Fideo	code as Spanish rice, fried in what? any meat?	cups, A side
Tacos and Tostados	type and amount of meat	number of tacos (amount on additions)
Tortilla	corn or flour? fried or not? fried in what? homemade or store-bought?	diameter and thickness
Tamales	type of sauce? cheese and/or meat filling?	tablespoon cups, A side
Biscochitos		diameter

Figure J.3. (continued)

Include brand name for:
- granola
- process cheeses
- crackers
- cookies
- margarine
- shortenings
- oils
- frozen entrees
- non-dairy creamers and toppings
- salad dressings
- cereals

Note: In instances where brand might indicate a special processing or ingredient (such as low sugar or low sodium) the brand name should be recorded. For example, S & W's red, blue or green label specialties or any of the Batter-Lite line of products.

Exclude brand name for breads, dairy products (except processed cheeses), cold cuts, canned fruits and vegetables, pastas and rice, peanut butter, bacon.

If food is eaten (or prepared) outside the home situation, the place should be identified. For a commercial establishment, record the type of restaurant as expensive, moderate, inexpensive or fast food. When necessary, look up type of fat used in restaurant surveys.

Was fat (or chile sauce) used in preparation?

If the question is not asked leave it [blank]

If the question is asked and the answer is:

a. no fat added in preparation, enter [1] (no)

b. fat added in preparation, enter [2] (yes)

If fat was added (and 2 was entered), on the next line enter amount and description (type of fat or chile sauce).

c. If participant doesn't know whether fat was added, enter [9] (unknown)

The End

Page ___

San Luis Valley Diabetes Study
Log for Questions and Answers about 24-Hour Recall Documentation
Form D6

Please record your initials, the initials of your contact at NCC, the date, your questions and the answer received each time you make a call to NCC. This will provide a way for all of us to learn from inquiries made to NCC and it will also be useful in evaluating changes that arise in documentation over time due to new information. When questions arise call 1. Mary Stevens (612) 376-1830, 2. Joyce Wenz (612) 376-1690, or 3. Gayle Smith (612) 376-1886.

Your Init	NCC Init	DATE	Questions and Answers (clearly mark NCC's answer i.e. Q. and A.)
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________
___	___	__|__|__	________________

Revision 3:5/24/84

Figure J.4. Nutrition Coding Center question and answer log

NUTRITION CODING CENTER

LRC – Lipid Research Clinics | NHLI – National Heart and Lung Institute | MRFIT – Multiple Risk Factor Intervention Trial

Nutrition Coding Center
Suite 528
2829 University Ave. S.E.
Minneapolis, Minnesota 55414
(612) 376-4562

DATE RECEIVED | BY | SEQUENCE NO.

RECIPE SPECIFICATION FORM

DATE SUBMITTED | BY | CLINIC

DIRECTIONS:
1. The form is printed on NCR (No Carbon Required) paper. Use ball point pen only. Press firmly so the copies are clear.
2. Write clearly. Typed forms will be appreciated, but are not required. Do not write in shaded areas.
3. Complete Date Submitted, Recipe Name, Portion Size and Yield boxes fully.
4. If item is to be APF, specify the ACOM, BCOM, CCOM and HOME fats if known.
5. Give a specific reference (Document, date of publication or edition, page no.) if at all possible.
6. Specify each ingredient in the recipe in the space provided. If the recipe does not contain a Principal Fat, "None" should be entered in the "Household Measure" column. If there are more than 15 ingredients, use a second Recipe Specification form. Write "continuation" in the "Comments and References" box of the second form.
7. Send the white and canary copies of the form to the NCC, attached to the Dietary Recall on which the recipe is recorded. Retain the pink copy for your records.

NCC ACTION | BY | DATE

RECIPE NAME(S) OR DESCRIPTION

PORTION SIZE(S) FOR FOOD TABLE (explain SV):

TS TB CP OZ SV _____

COMMENTS AND REFERENCES:

YIELD (give both number and size of servings):

APF RECOMMENDATIONS (if appropriate):

ACOM	BCOM	CCOM	HOME

LINE NO.	INGREDIENT DESCRIPTION	HOUSEHOLD MEASURE	DO NOT USE
1	Principal Fat		
2			
3			
4			
5			
6			
7			
8			
9			
10			
11			
12			
13			
14			
15			

NCC FORM 550 (1-1) JULY 74

Recipes for biscochitos, burritos, calabacitas, chicos, green chile sauce without meat, green chile sauce with meat, green chile salsa, hamburger gravy, menudo, posole, red chile sauce without meat, red chile sauce with meat, red chile salsa, rellenos, and flour tortillas were submitted to NCC from the SLVDS.

Figure J.5. SLVDS Recipes

APPENDIX K: Fiber Composition of SLVDS FFQ Food Items

Table K.1. Total dietary fiber by FFQ food items

Food or Food Group	Portion Size	Weight[1]	TOTAL DIETARY FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)	MM[4] Median	MM[4] Range (N)
1. apples	1 fruit	3.35	3.05	2.82-3.28 (2)	3.65	3.58-3.72 (2)	2.6	2.6 (1)
2. bananas	1 fruit	2.59	2.77	2.77 (1)	2.38	2.38 (1)	3.9	3.9 (1)
3. other fresh fruit		1.42	1.46	0.76-4.47	1.38	0.33-4.75		0.5-3.8
1 fruit - pear, nectarine, peach, tangerine, orange 1/2 fruit - grapefruit 1/2 cup - apricots, cherries, plums, strawberries, pineapple, honeydew, watermelon, canteloupe				(14)		(13)		(14)
4. dried fruit- raisins, dates, fig, prunes	1/2 cup	8.27	8.16	5.44-8.64 (4)	8.37	5.44-19.96 (5)	10.4	5.0-24 (4)
5. avocados and guacamole	1/2 cup	3.06	2.26	2.26 (1)	3.85	3.24-4.46 (2)	2.3	2.3 (1)
6. nuts and seeds	1 ounce	2.24	2.15	2.15-2.60 (3)	2.32	1.21-4.00 (14)	2.3	1.5-4.0 (8)
7. corn, chicos, hominy, and posole	1/2 cup	3.89	3.82	3.73-3.90 (2)	3.96	1.93-4.73 (4)	4.9	3.9-6.0 (2)
8. dried peas and beans	1/2 cup	7.15	9.40	4.70-10.89 (6)	4.91	3.35-12.70 (8)	4.4	0.9-7.0 (9)
9. sweet potatoes and winter squash	1/2 cup	2.98	2.40	2.40 (1)	3.56	2.80-4.99 (3)	4.0	2.9-5.0 (2)
10. carrots	1/2 cup	2.65	2.88	2.28-2.88 (3)	2.41	2.28-2.88 (3)	2.3	1.6-2.9 (5)
11. broccoli	1/2 cup	3.04	3.00	2.80-3.19 (2)	3.08	2.96-3.19 (2)	3.0	2.8-3.2 (2)
12. green peas	1/2 cup	4.51	4.33	3.84-5.35 (3)	4.68	3.36-6.00 (2)	5.7	3.8-9.6 (7)

Table K.1. (continued)

Food or Food Group	Portion Size	Weight[1]	TOTAL DIETARY FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)	MW[4] Median	MW[4] Range (N)
13. lettuce or tossed salad	1 cup	0.94	1.03	1.03 (1)	0.85	0.57-1.12 (2)	0.8	0.8 (1)
14. spinach, greens, verdolagos	raw-1 cup ckd-1/2 cup	1.89	0.99	0.99 (1)	2.77	1.63-3.81 (4)	3.1	2.7-5.7 (4)
15. other vegetables	1/2 cup	1.05	0.84	0.55-2.80 (12)	1.27	0.55-2.80 (18)	1.2	0.2-3.2 (22)
16. potatoes	1 cup	4.24	4.66	3.72-5.44 (3)	4.24	3.08-6.85 (10)	1.6	0.8-3.8 (7)
17. rice, macaroni, noodles	1 cup	1.70	2.23	1.20-2.80 (3)	1.17	0.35-4.68 (15)	1.6	1.6 (1)
18. corn tortillas or cornbread	1 serving	1.01	ND	ND	1.01	1.00-1.91 (3)	ND	ND
19. white bread, rolls or tortillas	1 slice/sv	0.64	0.67	0.67 (1)	0.61	0.18-0.95 (12)	0.7	0.7 (1)
20. whole wheat bread, rolls or tortillas	1 slice/sv	1.69	1.65	1.27-2.83 (3)	1.72	1.06-4.65 (9)	1.3	1.2-2.1 (3)
21. cold breakfast cereal (see Table 4.)								
22. hot breakfast cereal (see Table 4.)								
23. crackers or chips	1 ounce	1.20	ND	ND	1.20	0.25-3.48 (9)	ND	ND
24. pizza	2 slices	1.91	ND	ND	1.91	1.63-2.19 (2)	ND	ND
25. enchiladas, tamales, tacos, tostadas, burritos	1 NCC sv	1.16	ND	ND	1.16	0.26-1.56 (3)	ND	ND
26. vegetable soups/stews with meat	1 cup	2.02	3.25	3.25 (1)	2.02	0.25-2.92 (6)	ND	ND
27. green chile	1/2 cup	0.20	ND	ND	0.20	0.20 (1)	ND	ND

Table K.1. (continued)

Food or Food Group	Portion Size	Weight[1]	TOTAL DIETARY FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)	MW[4] Median	MW[4] Range (N)
28. red chile, salsa	1/2 cup	0.97	ND	ND	0.97	0.75-1.18 (2)	ND	ND
42. cookies, cakes or pies	1 serving	0.59	ND	ND	0.59	0.13-3.40 (25)	ND	ND
43. empanadas, sopapillas,	1 serving	0.36	ND	ND	0.36	0.23-0.60 (3)	ND	ND

[1]Weight - was calculated by averaging the median values from USDA and NCC.

[2]USDA - United States Department of Agriculture, Human Nutrition Information Service, Federal Building, Hyattsville, Maryland. Computer listings were obtained from Ruth Matthews and dated 7/15/85.

[3]NCC - Nutrition Coordinating Center, University of Minnesota, 2829 University Avenue, SE, Suite 526, Minneapolis, Minnesota, 55414. Computer listings were obtained from NCC data base version 13, dated 8/8/86. Listings included both elemental and recipe food items from NCC's nutrient data base.

[4]MW - Paul, A. A., Southgate, D. A. T. McCance and Widdowson's The Composition of Foods 4th edition. New York, Elsevier/North-Holland Biomedical Press, 1978.

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Table K.2. Insoluble fiber by FFQ food items

Food or Food Group	Portion Size	Weight[1]	INSOLUBLE FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)
1. apples	1 fruit	2.89	2.79	2.41-3.17 (2)	2.99	2.80-3.17 (2)
2. bananas	1 fruit	1.68	1.70	1.70 (1)	1.66	1.66 (1)
3. other fresh fruit 1 fruit - pear, nectarine, peach, tangerine, orange 1/2 fruit - grapefruit 1/2 cup - apricots, cherries, plums, strawberries, pineapple, honeydew, watermelon, canteloupe		1.11	1.26	0.21-3.28 (7)	0.95	0.08-4.03 (12)
4. dried fruit- raisins, dates, fig, prunes	1/2 cup	3.38	2.37	1.36-3.37 (2)	4.39	1.36-16.96 (4)
5. avocados and guacamole	1/2 cup	2.53	ND	ND	2.53	2.05-3.00 (2)
6. nuts and seeds	1 ounce	1.38	1.54	1.54 (1)	1.22	0.82-2.50 (8)
7. corn, chicos, hominy, and posole	1/2 cup	1.43	1.74	1.74 (1)	1.11	0.68-1.74 (4)
8. dried peas and beans	1/2 cup	3.62	3.87	2.25-5.20 (20)	3.36	2.00-4.84 (8)
9. sweet potatoes and winter squash	1/2 cup	1.58	1.69	1.20-2.17 (2)	1.47	1.37-2.30 (3)
10. carrots	1/2 cup	1.21	1.21	0.85-1.44 (6)	1.21	0.93-1.48 (3)
11. broccoli	1/2 cup	1.32	1.24	1.09-2.04 (3)	1.40	1.09-1.71 (2)
12. green peas	1/2 cup	2.93	2.60	1.84-3.65 (6)	3.26	3.04-3.48 (2)

Table K.2. (continued)

Food or Food Group	Portion Size	Weight[1]	INSOLUBLE FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)
13. lettuce or tossed salad	1 cup	0.61	0.69	0.44-0.66 (3)	0.62	0.57-0.67 (2)
14. spinach, greens, verdolagos	raw-1 cup ckd-1/2 cup	1.63	1.44	1.04-4.01 (7)	1.83	1.05-2.88 (4)
15. other vegetables	1/2 cup	0.94	0.97	0.25-2.77 (20)	0.92	0.26-1.94 (17)
16. potatoes	1 cup	2.10	1.70	1.55-2.55 (3)	2.50	2.50 (1)
17. rice, macaroni, noodles	1 cup	0.98	0.98	0.91-1.44 (4)	0.98	0.14-3.53 (13)
18. corn tortillas or cornbread	1 serving	1.05	1.08	1.08 (1)	1.01	1.01 (1)
19. white bread, rolls or tortillas	1 slice/sv	0.39	0.55	0.21-1.12 (7)	0.23	0.07-0.64 (12)
20. whole wheat bread, rolls or tortillas	1 slice/sv	1.41	1.52	0.99-3.88 (9)	1.30	0.72-2.93 (9)
21. cold breakfast cereal (see Table 4.)						
22. hot breakfast cereal (see Table 4.)						
23. crackers or chips	1 ounce	0.53	ND	ND	0.53	0.14-1.06 (6)
24. pizza	2 slices	1.03	ND	ND	1.03	0.88-1.17 (2)
25. enchiladas, tamales, tacos, tostadas, burritos	1 NCC sv	1.05	ND	ND	1.05	1.05 (1)
26. vegetable soups/stews with meat	1 cup	0.71	ND	ND	0.71	0.40-1.32 (6)
27. green chile	1/2 cup	ND	ND	ND	ND	ND

Table K.2. (continued)

Food or Food Group	Portion Size	Weight[1]	INSOLUBLE FIBER (grams/portion) USDA[a] Median	USDA[a] Range (N)	NCC[b] Median	NCC[b] Range (N)
28. red chile, salsa	1/2 cup	0.90	ND	ND	0.90	0.90 (1)
42. cookies, cakes or pies	1 serving	0.27	ND	ND	0.27	0.05-2.50 (25)
43. empanadas, sopapillas, doughnuts	1 serving	0.13	ND	ND	0.13	0.09-0.20 (3)

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[1]Weight - was calculated by averaging the median values from USDA and NCC.

[a]USDA - United States Department of Agriculture, Human Nutrition Information Service, Federal Building, Hyattsville, Maryland. Computer listings were obtained from Ruth Matthews and dated 7/15/85.

[b]NCC - Nutrition Coordinating Center, University of Minnesota, 2829 University Avenue, SE, Suite 526, Minneapolis, Minnesota, 55414. Computer listings were obtained from NCC data base version 13, dated 8/8/86. Listings included both elemental and recipe food items from NCC's nutrient data base.

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Table K.3. Soluble fiber by FFQ food items

Food or Food Group	Portion Size	Weight[1]	SOLUBLE FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)
1. apples	1 fruit	0.81	0.96	0.96 (1)	0.67	0.55-0.78 (2)
2. bananas	1 fruit	0.77	0.83	0.83 (1)	0.71	0.71 (1)
3. other fresh fruit 1 fruit - pear, nectarine, peach, tangerine, orange 1/2 fruit - grapefruit 1/2 cup - apricots, cherries, plums, strawberries, pineapple, honeydew, watermelon, canteloupe		1.21	2.01	2.01 (1)	0.40	0.09-2.01 (12)
4. dried fruit- raisins, dates, fig, prunes	1/2 cup	3.77	3.96	3.96 (1)	3.58	3.00-4.64 (4)
5. avocados and guacamole	1/2 cup	1.26	ND	ND	1.26	1.06-1.45 (2)
6. nuts and seeds	1 ounce	0.71	ND	ND	0.71	0.09-2.68 (8)
7. corn, chicos, hominy, and posole	1/2 cup	1.74	1.49	1.49 (1)	1.98	1.49-2.98 (3)
8. dried peas and beans	1/2 cup	3.03	4.05	3.33-4.37 (3)	2.01	0.68-5.44 (8)
9. sweet potatoes and winter squash	1/2 cup	1.60	1.10	1.10 (1)	2.09	1.09-2.68 (3)
10. carrots	1/2 cup	0.96	0.89	0.89 (1)	1.06	0.93-1.95 (3)
11. broccoli	1/2 cup	1.75	1.87	1.87 (1)	1.63	1.24-2.02 (2)
12. green peas	1/2 cup	0.91	0.40	0.40 (1)	1.42	0.32-2.52 (2)

Table K.3. (continued)

Food or Food Group	Portion Size	Weight[1]	SOLUBLE FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)
13. lettuce or tossed salad	1 cup	0.41	0.37	0.37 (1)	0.45	0.45 (1)
14. spinach, greens, verdolagos	raw-1 cup ckd-1/2 cup	0.54	0.33	0.33 (1)	0.75	0.44-1.44 (4)
15. other vegetables	1/2 cup	0.49	0.44	0.20-1.09 (8)	0.54	0.05-1.52 (17)
16. potatoes	1 cup	1.82	1.39	1.39 (1)	2.25	2.25 (1)
17. rice, macaroni, noodles	1 cup	0.31	ND	ND	0.31	0.12-1.14 (11)
18. corn tortillas or cornbread	1 serving	ND	ND	ND	ND	ND
19. white bread, rolls or tortillas	1 slice/sv	0.28	ND	ND	0.28	0.02-0.64 (11)
20. whole wheat bread, rolls or tortillas	1 slice/sv	0.69	1.02	1.02 (1)	0.37	0.26-1.59 (8)
21. cold breakfast cereal (see Table 4.)						
22. hot breakfast cereal (see Table 4.)						
23. crackers or chips	1 ounce	0.24	ND	ND	0.24	0.02-0.67 (6)
24. pizza	2 slices	0.80	ND	ND	0.80	0.70-0.89 (2)
25. enchiladas, tamales, tacos, tostadas, burritos	1 NCC sv	0.11	ND	ND	0.11	0.11 (1)
26. vegetable soups/stews with meat	1 cup	0.55	ND	ND	0.55	0.47-0.85 (4)
27. green chile	1/2 cup	ND	ND	ND	ND	ND

Table K.3. (continued)

Food or Food Group	Portion Size	Weight[1]	SOLUBLE FIBER (grams/portion) USDA[2] Median	USDA[2] Range (N)	NCC[3] Median	NCC[3] Range (N)
28. red chile, salsa	1/2 cup	0.29	ND	ND	0.29	0.29 (1)
42. cookies, cakes or pies	1 serving	0.31	ND	ND	0.31	0.05-1.03 (25)
43. empanadas, sopapillas,	1 serving	0.20	ND	ND	0.20	0.13-0.31 (3)

[1]Weight - was calculated by averaging the median values from USDA and NCC.

[2]USDA - United States Department of Agriculture, Human Nutrition Information Service, Federal Building, Hyattsville, Maryland. Computer listings were obtained from Ruth Matthews and dated 7/15/85.

[3]NCC - Nutrition Coordinating Center, University of Minnesota, 2829 University Avenue, SE, Suite 526, Minneapolis, Minnesota, 55414. Computer listings were obtained from NCC data base version 13, dated 8/8/86. Listings included both elemental and recipe food items from NCC's nutrient data base.

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Table K.4. Total, insoluble and soluble dietary fiber for hot and cold breakfast cereals

Cold Breakfast Cereals	Portion Size	FIBER CONTENT (grams/portion) Total	Insoluble	Soluble
100% Bran	1 cup	17.62	18.90	0.00
100% Natural Cereal (all types)	1 cup	5.51	3.84	1.66
40% Bran Flakes	1 cup	6.00	5.49	0.50
All Bran	1 cup	22.42	25.11	0.00
Almond Delight	1 cup	0.66	0.44	0.22
Alpha-bits	1 cup	0.92	0.33	0.58
Apple Jacks	1 cup	0.92	0.29	0.58
Apple Raisin Crisp	1 cup	4.20	3.84	0.36
Body Buddies	1 cup	0.92	0.33	0.58
Boo Berry	1 cup	0.92	0.33	0.58
Bran Buds	1 cup	22.42	23.26	0.00
Bran Chex	1 cup	8.18	7.93	0.24
Bran, raw	1/4 cup	5.97	5.56	0.40
Buc Wheats	1 cup	2.24	2.04	0.19
C.W. Post w/raisins	1 cup	5.45	2.88	1.64
C.W. Post wo/raisins	1 cup	5.14	2.90	1.55
Cap'n Crunch	1 cup	0.66	0.44	0.22
Cap'n Crunch w/crunchberries	1 cup	0.63	0.40	0.21
Cap'n Crunch w/peanut butter	1 cup	0.66	0.44	0.22
Cheerios	1 cup	1.95	1.18	0.41
Cocoa Krispies	1 cup	1.18	0.30	0.75
Cocoa Pebbles	1 cup	1.05	0.22	0.67
Cocoa Puffs	1 cup	0.92	0.33	0.58
Cookie Crisp	1 cup	0.99	0.31	0.63
Corn Bran	1 cup	6.01	6.33	0.18
Corn Chex, Shredded Corn	1 cup	0.50	0.50	0.00
Corn Flakes	1 cup	0.45	0.27	0.17
Count Chocula	1 cup	0.92	0.33	0.58
Country Cornflakes	1 cup	0.45	0.27	0.17
Cracklin Bran	1 cup	9.60	8.07	2.52
Cracklin' Oat Bran	1 cup	12.28	9.06	3.22
Crazy Cow, choc. or strawberry	1 cup	0.92	0.33	0.58
Crispix	1 cup	0.50	0.50	0.00
Crispy Rice	1 cup	0.50	0.34	0.19
Crispy Wheats'n Raisins	1 cup	3.82	2.57	0.64
Crunch Peanut	1 cup	0.63	0.40	0.24
Crunchy Granola	1 cup	5.98	4.18	1.80
Donkey Kong	1 cup	0.92	0.33	0.58
Fiber One	1 cup	14.95	16.74	0.00
Fortified Oat Flakes	1 cup	0.43	0.81	0.04
Franken Berry	1 cup	0.92	0.33	0.58
Froot Loops	1 cup	0.92	0.27	0.58
Frosted Flakes,sugar or banana	1 cup	0.92	0.20	0.58

Table K.4. (continued)

Cold Breakfast Cereals	Portion Size	FIBER CONTENT (grams/portion) Total	Insoluble	Soluble
Frosted Mini Wheats	4 biscuits	3.10	2.61	0.21
Frosted Rice Krinkles	1 cup	0.57	0.19	0.22
Frosted Rice Krispies	1 cup	0.92	0.19	0.58
Fruit'n Fiber (all types)	1 cup	8.94	5.98	2.96
Fruitful Bran	1 cup	8.94	5.98	2.96
Fruity Pebbles	1 cup	1.05	0.38	0.67
Germaid	1/4 cup	1.68	0.75	0.92
Golden Grahams	1 cup	0.70	0.54	0.27
Good Morning Cereal	1 cup	0.45	0.27	0.17
Graham Crackos	1 cup	1.83	1.83	0.00
Granola, plain	1 cup	5.98	5.25	1.80
Granola w/apples and cinnamon	1 cup	5.98	3.72	1.80
Granola w/honey and almonds	1 cup	5.98	4.74	1.80
Granola w/raisins	1 cup	5.98	5.16	1.80
Granola, homemade	1 cup	4.69	2.12	1.85
Grape-Nuts	1 cup	7.34	5.42	1.92
Grape-Nuts Flakes	1 cup	2.24	2.04	0.19
Heartland Natural Cereal	1 cup	5.83	4.62	1.76
Honey Bran	1 cup	5.36	3.62	1.73
Honey and Nut Corn Flakes	1 cup	0.66	0.40	0.25
Honey-nut Cheerios	1 cup	1.29	1.01	0.27
Honeycomb	1 cup	0.72	0.27	0.46
Ideal 100% Natural Cereal	1 cup	5.51	3.84	1.66
King Vitaman	1 cup	0.69	0.25	0.44
Kix, Puffed Corn (unsweetened)	1 cup	0.34	0.23	0.13
Life, plain and cinnamon	1 cup	2.64	1.40	1.23
Lucky Charms	1 cup	1.05	0.51	0.67
Marshmellow Krispies	1 cup	0.92	0.33	0.58
Most	1 cup	7.33	6.44	0.88
Muesli type cereal, Familia	1 cup	7.00	5.81	0.01
Muffets	1 NCC SV	2.40	2.23	0.16
Nature Valley Granola	1 cup	5.98	4.18	1.80
Nutrigrain Barley	1 cup	2.59	2.37	0.22
Nutrigrain Corn	1 cup	2.94	2.64	0.25
Nutrigrain Rye	1 cup	2.80	2.56	0.24
Nutrigrain Wheat	1 cup	3.08	2.79	0.26
Nutrigrain Wheat & Raisins	1 cup	4.55	4.16	0.39
O.J.'s	1 cup	0.66	0.44	0.22
Oatmeal, dry/uncooked	1 cup	9.06	4.50	6.23
Product 19	1 cup	0.56	0.36	0.19
Puffed Rice, Quaker Rice	1 cup	0.12	0.07	0.05
Puffed Wheat	1 cup	0.52	0.40	0.12
Quisp	1 cup	0.54	0.37	0.18
Raisin Bran	1 cup	7.53	5.11	2.49

Table K.4. (continued)

Cold Breakfast Cereals	Portion Size	FIBER CONTENT (grams/portion) Total	Insoluble	Soluble
Raisin Life	1 cup	2.64	1.40	1.23
Raisin Squares	1 cup	5.70	5.30	0.39
Raisin, Rice and Rye	1 cup	3.22	2.94	0.27
Rice Chex, Shredded Rice	1 cup	0.45	0.30	0.00
Rice Krispies	1 cup	0.50	0.17	0.19
Shredded Wheat n'Bran	1 cup	5.60	5.35	0.25
Shredded Wheat-large/spoonsize	1 cup	4.20	3.90	0.29
Shreddies	1 cup	4.30	3.99	0.30
Special K	1 cup	0.18	0.16	0.02
Sugar Corn Pops, Puffed Corn-s	1 cup	0.47	0.27	0.08
Sugar Frosted Flakes	1 cup	1.15	0.40	0.73
Sugar Smacks, Honey Smacks	1 cup	0.62	0.51	0.11
Sugar Sparkled Flakes	1 cup	0.68	0.41	0.26
Sun Country Granola w/raisins	1 cup	5.98	4.18	1.80
Sun Flakes Crispy Wheat & Rice	1 cup	0.50	0.35	0.19
Sunflakes	1 cup	0.50	0.50	0.00
Super Sugar Crisp	1 cup	0.59	0.46	0.13
Tasteeos	1 cup	1.56	0.99	0.40
Team	1 cup	0.75	0.41	0.29
Toasted Shredded Wheats&Raisin	1 cup	4.30	3.99	0.30
Toasties	1 cup	0.39	0.30	0.15
Total	1 cup	2.93	2.37	0.56
Trix, Corn Puffs, flav/sweet	1 cup	0.92	0.23	0.58
Vita Crunch	1 cup	5.98	4.18	1.80
Waffle O's	1 cup	0.99	0.34	0.63
Wheat Chex	1 cup	4.09	3.40	0.69
Wheat Flakes	1 cup	2.58	2.21	0.55
Wheat Germ w/br sugar & honey	1 cup	6.78	3.05	3.72
Wheat Germ, plain, toasted	1 cup	6.78	2.59	3.72
Wheat'n Raisin Chex	1 cup	4.80	3.77	0.81
Wheaties	1 cup	2.58	2.03	0.55
NONE		0.00	0.00	0.00
If the following cereals cannot be coded, with additional probing, from the list above, then the following codes are used.				
Bran Cereal	1 cup	6.00	5.49	0.50
Bran Mix	1 cup	0.41	0.25	0.16
Bran, old fashioned	1/4 cup	5.97	5.56	0.40
Granola, 7 grain	1 cup	5.98	4.18	1.80
High Fiber	1 cup	0.41	0.25	0.16
Wheat, cracked	1 cup	2.40	2.23	0.16
Wheat, ground	1 cup	0.41	0.25	0.16
Whole Grains	1 cup	0.41	0.25	0.16
UNKNOWN	1 cup	0.41	0.25	0.16

Table K.4 (continued)

Hot Cereals	Portion Size	FIBER CONTENT (grams/portion) Total	Insoluble	Soluble
Atole	3/4 cup	1.89	0.84	1.04
Bear Mush	3/4 cup	1.09	0.49	0.60
Bulgar	3/4 cup	3.95	1.77	2.18
Corn grits	3/4 cup	0.54	0.54	0.01
Cornmeal	3/4 cup	1.89	0.84	1.04
Cream of Rice	3/4 cup	0.45	0.20	0.25
Cream of Wheat, instant	3/4 cup	0.54	0.25	0.28
Cream of Wheat, mix&eat	3/4 cup	0.90	0.40	0.49
Cream of Wheat, regular	3/4 cup	0.56	0.26	0.30
Farina	3/4 cup	0.52	0.24	0.28
Hominy	3/4 cup	0.53	0.41	0.12
Kasha/Buckwheat Groats/Millet	3/4 cup	0.98	0.98	0.01
Maltex	3/4 cup	1.12	0.50	0.61
Maltomeal, plain or chocolate	3/4 cup	0.54	0.25	0.28
Maypo	3/4 cup	3.60	1.62	1.98
Oat Flour & Soy Protein Cereal	3/4 cup	10.32	4.64	5.67
Oatbran	3/4 cup	5.83	2.89	2.93
Oatmeal, instant/apples,raisin	1 pkt	2.23	1.30	0.92
Oatmeal, instant/plain or flav	1 pkt	2.19	1.28	0.91
Oatmeal, inst/maple & br sugar	1 pkt	2.01	1.17	0.83
Oatmeal, instant/raisin, spice	1 pkt	2.05	1.20	0.85
Oatmeal, unspecified type	3/4 cup	3.50	1.57	1.92
Ralston	3/4 cup	7.18	3.23	3.95
Red River	3/4 cup	3.62	1.62	1.99
Roman Meal	3/4 cup	3.62	1.62	1.99
Sun Maid (all types)	3/4 cup	2.01	1.17	0.83
Wheat, rolled	3/4 cup	1.09	0.49	0.60
Wheatena	3/4 cup	0.54	0.25	0.29
Whole Wheat Natural Cereal	3/4 cup	1.09	0.49	0.60
Zoom	3/4 cup	1.12	0.50	0.62
NONE		0.00	0.00	0.00
If the following cereals cannot be coded, with additional probing, from the list above, then the following codes are used.				
Oat and Barley Meals	3/4 cup	3.50	1.57	1.92
Old Fashioned Oats	3/4 cup	3.50	1.57	1.92
Rolled Oats	3/4 cup	3.50	1.57	1.92
Whole Grains	3/4 cup	0.90	0.40	0.49
UNKNOWN	3/4 cup	0.90	0.40	0.49

DATA SOURCES: United States Department of Agriculture, Human Nutrition Information Services, printouts dated 7/15/85. And Nutrition Coding Center, University of Minnesota, nutrient data base version 13.

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CURRICULUM VITAE

Julie Ann Marshall

Personal

Born:	January 7, 1951, Omaha, Nebraska, U.S.A.
Home Address:	1060 Holly Street Denver, Colorado 80220 Phone: (303) 388-0360
Business Address:	Department of Preventive Medicine and Biometrics School of Medicine, Box C-245 University of Colorado Health Sciences Center Denver, Colorado 80262 Phone: (303) 270-8358
Social Security No:	524-72-2483

Education

B.S. (1973)	Colorado State University Major: Microbiology
M.S. (1975)	School of Public Health, University of Hawaii Major areas: Epidemiology and Biostatistics
Continuing Education (1979)	School of Public Health, University of Minnesota Course Titles: "Clinical Trials", "Advanced Statistical Methods in Epidemiology", "Epidemiological Basis in Health Services Planning and Evaluation"
Continuing Education (1984)	University of Massachusetts at Amherst, Course Titles: "Nutritional Epidemiology", "Cardiovascular Disease Epidemiology", "Advanced Methods in Epidemiology"
Ph.D. (1987) Epidemiology	School of Public Health, University of Washington Department of Epidemiology. Dissertation title: "The Role of Dietary Fiber in the Etiology of Non-insulin Dependent Diabetes Mellitus"

Honors

1972:	Institute of Telecommunication Sciences Superior Performance Award
1973:	Colorado State University Graduated with Honors
1973-75:	East-West Center Population Institute, Hawaii Degree Scholar Award
1986:	Society for Epidemiological Research Student Workshop Participant

Professional Record

1976:	Research Assistant, Hawaii Tumor Registry Cancer Center of Hawaii
1976-78:	Coordinator of Data Management and Analysis, Cervical Cancer Screening Program, Cancer Center of Hawaii
1978-80:	Research Assistant, Department of Preventive Medicine, University of Colorado School of Medicine
1980-82:	Instructor, Department of Preventive Medicine, University of Colorado School of Medicine
1982-83:	Leave of Absence from University of Colorado, Graduate student in Epidemiology, University of Washington
1983-85:	Instructor, Department of Preventive Medicine and Biometrics, University of Colorado School of Medicine
1985-present:	Senior Instructor, Department of Preventive Medicine and Biometrics, University of Colorado School of Medicine

Professional Societies

Member, Society for Epidemiological Research
Member, American Diabetes Association

Teaching

1979-81:	Lectures on "Data Sources" and "Methods of Rate Adjustment" for Health Sciences I and Epidemiology
1979-80:	Methods of Inquiry in Medicine, 4.5 quarter hours Member of Faculty Teaching Team
1980-81:	Methods of Inquiry in Medicine, 3 quarter hours Coordinator of Faculty Teaching Team
1983-84:	Epidemiology, 4 quarter hours Co-instructor with Richard F. Hamman, M.D., Dr.P.H.
1983-87:	Lecture on "Dietary Assessment and Nutritional Epidemiology" for Nutrition in Public Health, Chronic Disease Epidemiology, and Nutrition and Cancer
1984-86:	Epidemiology, 4 quarter hours Course Director
1986:	Quantitative Methods in Medicine, 4 quarter hours Discussion Leader
1987:	Chronic Disease and Injury Epidemiology, 3 quarter hours Course Co-director

Research

San Luis Valley Diabetes Study (Co-investigator, 85% at present) Funded by NIH 5/83-4/86, $1,090,020 and 5/86-4/89, $1,849,265, Principal Investigator - Richard F. Hamman

Mortality Patterns from Selected Diseases Funded by the Colorado Department of Health, $1500, and the Department of Preventive Medicine, $1000, 12/81-8/82, Principal Investigator - Julie A. Marshall

Study of Birth Defects Among Oil, Chemical, and Atomic Worker's Union Members (Research Assistant) Funded by the March of Dimes 1/81-12/82, $75,088, Principal Investigator - Miriam Orleans

Diagnostic Ultrasound Study (Data base design and management, 9/81-12/82) Funded by the Bureau of Radiologic Health, 1/78-12/82, Principal Investigators - Miriam Orleans and Albert Haverkamp

Study of Infant Diarrhea in Western Samoa Funded by the East-West Center Population Institute, 4/75-7/75, $3000, Principal Investigator - Julie A. Marshall

Service

Data Sources Collection - Department of Preventive Medicine, UCHSC
Development and maintenance of health and population data resources for departmental research and teaching (1978-present)

Graduate Program - Master of Science in Public Health, UCHSC
Member, Admissions Committee (1983-1986)
Member, Academic Policy Committee (1983-present)

Annual Report - Department of Preventive Medicine, UCHSC
Compiled and edited (1980-1982)

Publications

Green LA, Reed FM, Martini C, Warren PS, Simmons RL, Marshall JA, Differences in morbidity patterns among rural, urban, and teaching family practices: A one year study of twelve Colorado family practices. Journal of Family Practice 9(6): 1975-1980, 1979.

Marshall JA, "Data Sources" Publication Series of the Department of Preventive Medicine No. 80-100, University of Colorado Health Sciences Center, September, 1980.

Poole SR, Morrison JD, Marshall JA, Simmons R, Pediatric health care in family practice. Journal of Family Practice 15(2): 945-952, 1982.

Publications - continued

Marshall JA, Hamman RF, "Mortality Patterns from Selected Diseases in Colorado" Publications Series of the Department of Preventive Medicine and Biometrics, University of Colorado Health Sciences Center, No. 82-100, August, 1982.

Savitz DA, Harley B, Krekel S, Marshall JA, Bondy J, Orleans M, Survey of Reproductive Hazards Among Oil, Chemical, and Atomic Workers Exposed to Halogenated Hydrocarbons. American Journal of Industrial Medicine 6: 253-264, 1984.

Jones RH, Marshall JA, Hamman RF, Adjusting logistic regression for different sampling fractions across strata. Submitted to the American Journal of Epidemiology, July, 1987.

Hamman RF, Marshall JA, Baxter J, Kahn LR, Mayer EJ, Orleans M, Murphy JR, Lezotte DC, The San Luis Valley Diabetes Study: Methods and prevalence of non-insulin dependent diabetes mellitus (NIDDM) in a biethnic Colorado population. Submitted to the American Journal of Epidemiology, October, 1987.

Marshall JA, The role of dietary fiber in the etiology of non-insulin dependent diabetes mellitus. Doctoral dissertation, University of Washington, 1987.

Abstracts

Hamman RF, Marshall JA, Baxter J, Kahn L, Ulibarri V, Prevalence of diabetes in rural Colorado. Diabetes, 34(Suppl 1): 131a, 1985.

Hamman RF, Baxter J, Marshall JA, Murphy J, Design and methods of the San Luis Valley Diabetes Study. Diabetes Research in Clinical Practice, Suppl 1: 5219, 1985.

Hamman RF, Gay E, Ulibarri V, Carosene-Link P, Baxter J, Kahn LR, Marshall J, Murphy JR, Undiagnosed NIDDM in Hispanics: Prevalence and sociodemographic characteristics. Research Symposium on Diabetes in Hispanic Americans. The National Coalition of Hispanic Health and Human Services Organizations. New York, New York, September, 1986.

Hamman RF, Marshall JA, Baxter J, Mayer E, Orleans M, Murphy JR, Lezotte DC, Jones RH, The San Luis Valley Diabetes Study: Prevalence of NIDDM in a biethnic Colorado population. Seventh Annual Epidemiological Research Exchange, University of Colorado School of Medicine, October, 1987.