Original Articles

Diabetes, Parental Diabetes, and Obesity in Oklahoma Indians

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The relationship between diabetes and parental diabetes status and obesity in Oklahoma Indians was studied. Data from 2095 adult Oklahoma Indians (1085 type II diabetic subjects and 1010 nondiabetic subjects) through a complete physical examination and personal interview showed a strong association between diabetes and parental diabetes status. Frequency of diabetes among siblings was significantly higher in families with affected parents than those without diabetic parents. No significant difference was found between families with one diabetic parent and those with two diabetic parents. The diabetic individuals were more obese than the nondiabetic individuals at age 18 and at interview. Obesity was defined as percent body mass index >120. After adjusting for possible age and sex effects, the risk of diabetes for the obese was estimated as almost twice that for the nonobese. DIABETES CARE 1985; 8:107–13.

There are many factors associated with the epidemiology of diabetes mellitus. Among these factors, heredity and obesity have received much attention in the past.[1–6] Diabetes has been considered a hereditary disorder in which obesity often plays a "precipitating" role[6] and diabetes was subdivided into two groups, juvenile and maturity onset, on the basis of the age of diagnosis. In 1979, the National Diabetes Data Group[7] reclassified diabetes into two major subdivisions: insulin-dependent diabetes mellitus (IDDM), type I, and non-insulin-dependent diabetes mellitus (NIDDM), type II. Type I diabetic individuals are "dependent on injected insulin to prevent ketosis and to preserve life, although there may be preketotic, non-insulin-dependent phases in the natural history of the disease."[7] Although the disease may occur at any age, the majority of IDDM patients are young at onset. Persons with type II diabetes are not insulin dependent or ketosis prone, although they may be treated with insulin and they can develop ketosis. The majority of NIDDM patients are over 40 yr of age at onset, but NIDDM may occur at any age.

Type II diabetes has been divided into two subclasses according to whether or not obesity is present. About 60–90% of NIDDM patients are obese. Kempner et al.[8] studied 106 very fat patients and found that 63% had elevated plasma glucose levels 2 h after a test meal, even though the average age was only 34 yr. It is believed that obesity alone can and does produce diabetes if sufficient in degree and duration.[6] Recently Knowler et al.[4] studied the diabetes incidence in Pima Indians and concluded that obesity and parental diabetes contributed significantly. The present project studied the relationship of parental diabetes, obesity, and other factors to diabetes in Oklahoma Indians.

METHODS

Dr. Kelly M. West became interested in the high rate of type II diabetes among the Oklahoma Indian tribes during his studies on the epidemiology of diabetes.[6,9,10] In 1972–79, he recruited 2095 adult Oklahoma Indians from 27 contiguous counties. They represent the Five Civilized Tribes (Cherokee, Choctaw, Chickasaw, Creek, and Seminole) and the Plains Tribes including Comanches, Kiowas, Caddos, Wichitas, Cheyennes, Osages, Shawnees, Arapahos, Apaches, Poncas, etc. Among them, 1085 (52%) were diagnosed as being diabetic and 1010 (48%) as nondiabetic. The diabetic subjects were recruited from the Indian Hospitals of the U.S. Public Health Service and their satellite clinics. All diabetic subjects had fasting venous plasma glucose concentrations ≥140 mg/dl or elevated venous plasma glucose value ≥200 mg/dl during the oral glucose tolerance test at 2 h after 75–100 g of oral glucose. Most of the subjects reside in predominantly rural Southwest areas (Lawton, Shawnee, and Clinton areas) and all have similar lifestyle and social and economic status. More than half of the subjects are from the Lawton area. In this subdistrict, more than 86% of eligible diabetic individuals were recruited.

About 40% of the nondiabetic subjects were spouses of the diabetic subjects. Other nondiabetic subjects were volunteers recruited from the general population, including relatives and friends of the diabetic subjects and healthy relatives of other outpatients at the hospitals or clinics. A very small number of them were recruited from attendees at tribal meetings. Thus, the nondiabetic subjects were a quasi-random sample of the nondiabetic population. Although this sample is a crude representative, we believe the nondiabetic subjects are comparable in obesity and quantum of Indian blood to the population. We realize that a nondiabetic individual who has a diabetic parent or relative may be more likely to participate in the study. If we compare only the proportion of diabetic parents between the diabetic and nondiabetic individuals, we may overestimate the prevalence of diabetic parents among the nondiabetic population and thus underestimate the difference between diabetic and nondiabetic individuals in parental diabetes status. However, we also analyzed the data in a different way by identifying in the study 604 families and the number of diabetic and nondiabetic children in each family. It enabled us to compare frequencies of diabetes among siblings in the three parental diabetes status groups (both, one, and neither parent[s] diabetic). The quasi-random sample of nondiabetic subjects has little effect on this analysis.

The subjects were invited to visit the diabetes clinic for a complete physical examination. Detailed questionnaires on family history of diabetes and weight were administered by a registered nurse through a personal interview. Part of the data was used in the Multinational Study of Vascular Disease in Diabetes sponsored by the World Health Organization.

TABLE 1
Summary statistics

	Diabetic subjects		Nondiabetic subjects	
	N	%	N	%
Sex				
Male	406	37	386	38
Female	679	63	624	62
Age				
<40	139	13	503	50
>40	946	87	507	50
Quantum of Indian blood				
<50%	108	10	218	22
51–75%	83	8	64	6
76–100%	883	82	704	71
Diabetic parents				
Neither	108	44	347	53
Father only	46	19	104	16
Mother only	60	24	157	24
Both	32	13	47	7
PBMI				
<80 (lean)	2		0	
80–120 (normal)	170	16	295	30
120–139 (obese)	255	24	268	27
>140 (very obese)	651	60	423	43

TABLE 2
Number of subjects by tribe and diabetes status

Tribe	Diabetic	Nondiabetic	Total
Comanche	216	383	599
Kiowa	178	249	427
Choctaw	80	70	150
Caddo	52	66	118
Creek	77	15	92
Seminole	82	6	88
Wichita	46	25	71
Cheyenne	62	7	69
Chickasaw	31	22	53
Cherokee	19	28	47
Others	231	117	348
Total	1074	988	2062

More than 98% of Indians have a medical record in the Indian Health Service system and they receive more than 90% of their care from the system. This central record system and the relative immobility of the population provide excellent opportunities for collecting information on weight history. The Plains Indians of Southwestern Oklahoma have been weighed frequently for the last 40 yr. They have had good patient care in the Lawton Indian Hospital for half a century.

For all subjects, percent body mass index (PBMI), defined as 100 (BMI/standard BMI) was used as a measure of obesity, where BMI (body mass index) was defined as (weight in kg)/(height in m)^2 and standard BMI used for males and females were, respectively, 22.1 and 20.6.[12] West suggested that "obese" and "very obese" be defined, respectively, as $120 \leq$ PBMI ≤ 139 and PBMI ≥ 140. Individuals with PBMI < 80 are considered "lean" and those with PBMI of 80–120 are "normal."

To study the relationship of parental diabetes and obesity to diabetes, both descriptive and analytic methods were used, including contingency table analysis, two-sample tests, analysis of variance, and the Mantel-Haenszel chi-square method.[13,14]

RESULTS

Age, sex, and tribal distributions. Table 1 gives the age and sex distributions in diabetic and nondiabetic subjects. The sex distributions in the two groups are very similar. The nondiabetic subjects are younger on the average. Average ages at interview for the diabetic and nondiabetic individuals were 51.5 yr and 41.5 yr, respectively. More than two-thirds (67%) of the diabetic group were diagnosed after 40 yr of age. Average known duration of the disease for the diabetic group was 6.7 yr. Some patients were newly diagnosed and three had had the disease for >30 yr.

The "Indianness" of each subject was measured by the quantum of Indian blood. All but two subjects had at least 50% Indian blood. Average quantum of Indian blood was 90%, and 73% were full-blood Indians. Table 1 gives the distribution of quantum of Indian blood in the diabetic and

nondiabetic subjects. Quantum of Indian blood was significantly ($P < 0.0001$) higher in the diabetic than in the nondiabetic subjects. It was also found that the proportion of obese subjects increases significantly ($P < 0.001$) as the quantum of Indian blood increases (66% of the subjects with ≤50% Indian blood and 80% of those with >50% Indian blood were obese).

Forty-one tribes were represented by the study subjects. Table 2 lists the top 10 tribes (according to the number of participants) by diabetes status. All 10 tribes were classified as having high or probably high (Wichitas) diabetes rates.[15] The Comanches and Kiowas constituted almost half of the study subjects. These two tribes have been closely related for a long time. They reside in the Lawton area. Table 3 gives the proportions of subjects in the top 10 tribes who had at least one diabetic parent and who were obese at time of interview. Obesity and parental diabetes are uniformly prevalent among all tribes. Overall, about 75% of the subjects were obese and 56% had at least one diabetic parent.

Family history of diabetes. The parental diabetes status in the diabetic and nondiabetic groups is summarized in Table 1. The data indicate a highly significant association between diabetes and parental diabetes ($P < 0.01$). As indicated in METHODS, this association may have been even more pronounced if our nondiabetic subjects had been a completely random sample.

Our data also show the familial aggregation of diabetes mellitus. Among the 1000 diabetic individuals who answered the question on relative diabetes status, 58% had one or more close relatives (excluding parents) with diabetes and 29% had no diabetic close relatives. Twenty-six percent of the diabetic individuals had diabetic brothers as compared with 13% of the nondiabetic individuals; 31% of the diabetic individuals had diabetic sisters, while only 15% of the nondiabetic individuals did. Using study subjects' names and parents' identification numbers, we were able to link the individuals of the same family. Six hundred four families were successfully identified. The number of diabetic siblings and parents' diabetes status reported by those included in our study are summarized in Table 4. Diabetes rates among the three parental diabetes status groups were significantly different ($P < 0.001$). The main contribution was the highly significant difference between individuals with no diabetic parents and those with at least one diabetic parent. The diabetes rate of individuals with one diabetic parent is only suggestively different from those with no diabetic parents ($P \cong 0.09$).

One hundred sixty-four diabetic patients knew their ages at diagnosis and their parental diabetes status (Table 5). Sixty-six percent of those whose mothers were diabetic were diagnosed before the age of 40. Fifty-three percent of the patients with two diabetic parents were diagnosed before 40 yr of age as compared with 18% of those with nondiabetic parents. The results indicate a significantly high ($P < 0.001$) association between parental diabetes and age of diagnosis. This is consistent with previous findings.[2]

Obesity. Table 6 summarizes eight weight-related variables in diabetic and nondiabetic subjects. Obviously, the majority of the study subjects were obese. On the average, they weighed 145% of standard. In the diabetic subjects, weight at diagnosis averged 160% of standard. The diabetic subjects had significantly higher PBMI—maximum lifetime PBMI and PBMI at 18 yr of age—than did the nondiabetic subjects. Subscapular and ulnar skinfolds were thicker in the diabetic than the nondiabetic subjects.

Table 1 gives the distribution of PBMI for the diabetic and nondiabetic subjects. Among the nondiabetic subjects, the

TABLE 3
Obesity and parental diabetes by tribe

Tribe	Subjects with a diabetic parent		Obese subjects	
	N	%	N	%
Comanche	67.0	227	75.1	598
Kiowa	50.3	197	71.7	226
Choctaw	57.4	94	66.0	150
Caddo	41.5	65	69.5	118
Creek	64.0	25	88.0	92
Seminole	50.0	6	77.3	87
Wichita	59.1	22	69.0	71
Cheyenne	60.0	10	85.5	69
Chickasaw	58.8	17	76.9	52
Cherokee	44.0	107	78.1	347
Others	47.7	107	78.1	347
Total	55.6	795	74.9	1857

TABLE 4
Parental diabetes and frequency of diabetes among siblings

Diabetes status of parents	No. of families	Sibling		
		Total	Diabetes	
			Number	%
Both diabetic	48	307	55	17.92
One diabetic	242	1248	187	14.98
Neither diabetic	314	1557	154	9.89
Total families	604	3112	396	12.72

TABLE 5
Frequency of diabetes by age at diagnosis and parental diabetes status

Age at diagnosis	Parental diabetes status								
	Both diabetic		Diabetic father		Diabetic mother		Neither diabetic		
	N	%	N	%	N	%	N	%	Total
20–39	9	53	12	39	25	66	14	18	60
≥40	8		19		13		64		104
Total	17		31		38		78		164

TABLE 6
Summary obesity statistics

Variable	Diabetic					Nondiabetic					P-value
	N	Mean	SD	Maximum	Minimum	N	Mean	SD	Maximum	Minimum	
PBMI at interview	1078	150.6	34.0	498.5	72.4	986	138.5	31.3	333.5	82.0	0.0001
PBMI at diagnosis	764	160.1	33.7	307.8	78.7	—	—	—	—	—	—
Maximum lifetime PBMI	828	177.0	33.7	327.7	75.1	517	151.0	34.4	478.7	92.3	0.0001
PBMI at 18 yr of age	820	111.27	25.1	246.6	63.3	519	104.7	19.5	233.9	70.6	0.0001
Age of maximum weight	821	40.1	11.7	80	15	517	37.4	14.1	88	14	0.0001
Skinfold (in/mm)											
Triceps	767	26.6	11.8	67	5	523	25.9	11.1	80	0	0.2561
Subscapular	765	37.5	13.6	76	8	523	34.6	16.2	140	8	0.0009
Ulnar	385	7.1	4.3	30	0	454	6.4	3.4	28	2	0.0085

overall obesity rate, though very high (70%), was significantly ($P < 0.001$) lower than the diabetic subjects (84%). The obesity rate in the women (81%) was significantly ($P < 0.001$) higher than that in the men (71%). For the diabetic subjects, the average PBMI was much higher at diagnosis (160.1) than at interview (150.6, $P = 0.0001$). PBMI at diagnosis was available for 764 diabetic subjects. A strikingly high 71% were very obese, 19% were obese, and only 10% normal. Some patients obviously had lost weight after diagnosis.

Since weight is highly related to age and there are a greater number of young people in the nondiabetic group, it is important to study the association between obesity and diabetes, adjusting for possible effects connected with age and sex. Table 7 gives the distribution of diabetic and nondiabetic study subjects by obesity, sex, and age. The Mantel-Haenszel chi-square value of 28.66 (1 degree of freedom) indicates a highly significant ($P < 0.001$) association between obesity and diabetes after adjusting for possible age and sex effects. The odds ratio computed from this table is 1.93.

We also examined the fasting plasma glucose and 2-h value of the nondiabetic subjects in various PBMI and skinfold groups. Table 8 gives the average values. Note that both values increased significantly as the PBMI increased. Subjects with subscapular skinfold ≥30 mm had significantly higher fasting plasma glucose and 2-h value. No significant differences were found between the two ulnar skinfold groups. If tricep skinfold is used, only the 2-h values are significantly different between the two groups.

Simultaneous effect of parental diabetes and obesity. The univariate analyses above indicate that parental diabetes and obesity are individually associated with diabetes. It would be of interest, then, to examine the simultaneous effect of these

TABLE 7
Distribution of diabetic and nondiabetic subjects by age, sex, and obesity

Sex	Age	Diabetic subjects			Nondiabetic subjects		
		Obese	Nonobese	Total	Obese	Nonobese	Total
Men	20–29	4	2	6	59	33	92
	30–39	40	9	49	73	26	99
	40–49	117	29	146	46	24	70
	50–59	103	30	133	38	16	54
	≥60	39	31	70	37	24	61
Total		303	101	404	253	123	376
Women	20–29	9	1	10	99	69	168
	30–39	64	8	72	96	37	133
	40–49	204	19	223	100	32	132
	50–59	185	15	200	88	19	107
	≥60	141	28	169	55	15	70
Total		603	71	674	438	172	610

TABLE 8
Average fasting plasma glucose values and 2-h values by PBMI and skinfolds, among nondiabetic subjects

Variable	Fasting plasma glucose	N	2-H variable	N
PBMI				
<120	90.70	272*	96.08	271*
120–139	95.10	264*	108.31	264*
≥140	97.93	396*	119.92	395*
P-value	0.0001		0.0001	
Subscapular skinfold				
<30 mm	93.17	182	101.62	181
≥30 mm	96.27	287	119.06	286
P-value	0.005		0.0001	
Tricep skinfold				
<20 mm	95.01	150	101.77	149
≥20 mm	95.01	318	117.07	317
P-value	0.99		0.0001	
Ulnar skinfold				
<5 mm	94.88	145	111.97	145
≥5 mm	96.62	255	116.17	253
P-value	0.15		0.22	

*Significantly different (at 0.05 level) subgroups from pairwise multiple comparisons.

variables. With respect to obesity in women, the diabetic group had a significantly higher ($P < 0.002$) PBMI than the nondiabetic group in both parental diabetes groups. PBMI at 18 yr of age (PBMI 18) was significantly higher ($P = 0.0001$) in the diabetic than in the nondiabetic individuals only in women without diabetic parents. Both men and women with at least one diabetic parent reached their maximum weight at an earlier age than those with nondiabetic parents. Particularly, the average age of maximum weight for the male and female diabetic subjects with at least one diabetic parent were, respectively, 9 yr and 5½ yr younger than the male and female diabetic subjects with nondiabetic parents ($P < 0.005$).

The proportion of diabetic individuals in siblings of obese and nonobese diabetic and nondiabetic subjects were compared. The subjects had a total of 3450 living siblings; 453 (13%) were diabetic. Table 9 gives the numbers of living and diabetic siblings of the obese and nonobese subjects by their diabetes status. Siblings of the diabetic subjects had a significantly higher diabetic rate (211/812 = 0.26) than those of the nondiabetic subjects (242/1838 = 0.13), regardless of their obesity conditions. Siblings of the lean and normal (nonobese) nondiabetic subjects had the lowest diabetes rate (7.2%), followed by siblings of the obese nondiabetic subjects (10%). Siblings of the nonobese diabetic subjects had a slightly higher diabetes rate (31.4%) than the obese diabetic subjects (25.2%). However, the difference was not statistically significant. The slight difference could have been due to the relatively small number of nonobese diabetic subjects. We further classified the nonobese subjects into two subgroups: (PBMI < 100 and 100 ≤ PBMI < 120) and found that the siblings' diabetes rate increased with the subjects' PBMI. Siblings of the nondiabetic subjects with PBMI < 100 had the lowest diabetes rate (6%).

Table 10 classifies the subjects according to sex, obesity status, and parental diabetes status. In both men and women, the proportion of diabetic individuals is highest in obese subjects with one or two diabetic parent(s) and is lowest in nonobese subjects with nondiabetic parents. Obese subjects with no diabetic parents had a much higher diabetes rate than the nonobese subjects with diabetic parents.

DISCUSSION AND CONCLUSION

The project studied the relationship between diabetes mellitus and familial diabetes, obesity, and other possible risk factors in Oklahoma Indians. Quantum of Indian blood is very high in this study group. Our data showed a strong association between diabetes and parental diabetes status even though our estimate was conservative. Frequency of diabetes among siblings was significantly higher in families with one or two diabetic parent(s) than those without affected parents. However, no significant difference was found between families with one diabetic parent and those with two diabetic parents. The results are consistent with other reports.[16] Though all diabetic subjects were non-insulin-dependent, there seemed to be a greater proportion with diabetic parents in those who were diagnosed before 40 yr of age.

Seventy-seven percent of all subjects and 84% of the diabetic subjects were obese. The diabetic group was more obese than the nondiabetic group at the age of 18 yr and at interview. A significantly large proportion of the diabetic group was also obese in childhood. In the diabetic subjects, mean

TABLE 9
Numbers of living siblings and diabetic siblings of subjects in different obesity and diabetes categories

	Subject's status					
	Nonobese			Obese		
	Diabetic	Nondiabetic	Total	Diabetic	Nondiabetic	Total
No. of living siblings	105	803	908	707	1035	2542
No. of diabetic siblings	33	58	91	178	184	362
Diabetes rate	0.314	0.072	0.1	0.252	0.1	0.142

TABLE 10
Proportion of diabetic subjects by sex, obesity, and parental diabetes status

Parental diabetes	At least one diabetic parent				No diabetic parents			
Obesity	Obese		Nonobese		Obese		Nonobese	
Status*	D	ND	D	ND	D	ND	D	ND
Men	35	77	10	38	29	85	12	44
Proportion of diabetic subjects	31%		21%		25%		21%	
Women	80	147	8	42	57	147	3	65
Proportion of diabetic subjects	35%		16%		28%		4%	
Men and women	115	224	18	80	86	232	15	109
Proportion of diabetic subjects	34%		18%		27%		12%	

*D = diabetic; ND = nondiabetic.

weight was 150% of ideal at time of interview and 160% at time of diagnosis. The obesity rate in the women was higher than that in the men. After adjusting for possible age and sex effects, the risk of diabetes at interview for the obese (PBMI > 120) was estimated as almost twice that for the nonobese.

The association of diabetes with the location of the adipose tissue in women has been reported by several authors.[17–20] Evidence is accumulating that women with upper-body obesity (or proximal obesity) are at higher risk of developing diabetes than women with lower-body obesity (pear-shape obesity). It is suggested that women with upper-body obesity have a greater number of large fat cells, which have a greater glucose intake and fewer insulin receptors than do small fat cells. On the other hand, women with lower-body obesity have a greater number of small cells, which are less likely to resist the effect of insulin. We do not have data on body build or fat distribution. Our observation is that Oklahoma Indian women usually have upper-body obesity. This might explain why diabetes is so prevalent among Indian women.

Our data also showed that fasting plasma glucose and the 2-h value increased significantly as PBMI increased in both diabetic and nondiabetic subjects. The two variables were significantly higher in nondiabetic individuals with subscapular skinfold ≥30 mm. Evidently, obesity is highly associated with elevated fasting plasma glucose level and 2-h value and is certainly a major contributing factor.

Diabetes was rare in Oklahoma Indians before 1940. They were very lean before the 20th century. Most of them were very active in outdoor activities; many were nomadic hunters and meat eaters. In the last several decades, the Indian lifestyles have changed completely. Outdoor activities have reduced drastically; however, their diets, concepts of ideal weight, and metabolic system still remain pretty much the same. Apparently, their consumption of calories is excessive in relation to energy expenditure. This excess is substantially derived from high-fat diet, which was necessary in the past. Attitudes toward adiposity also play an important role. It was reported that middle-aged Plains Indian women believe they should weigh about 50 lb more than what would be considered ideal by the upper-class white women.[21] Particularly, Comanche Indians expect women to gain considerable weight between 20 and 40 yr of age. This concept could have come from the fact that they were all very lean in the past and fat or abundant women were considered beautiful. It is interesting to find that diabetes rate and obesity level increase significantly as the degree of "Indianness" increases. Our conjecture is that "Indianness" is directly related to the Indian diet, concept of ideal weight, and metabolic system that they have established to suit their earlier lifestyle.

There is no doubt that both genetic and environmental factors contribute to the high prevalence of diabetes in Oklahoma Indians. The diets and opinions and attitudes on obesity are essential environmental factors. Thus, in addition to basic research on genetic, metabolic, and biochemical characteristics that are related to diabetes, the importance of cultural effects cannot be ignored.

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Age of Onset and Type of Diabetes

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The age of onset and the clinical type of diabetes mellitus were evaluated on the basis of a cross-sectional study of medical records of 14 municipal health centers in East Finland. Altogether 281 patients were classified as having insulin-dependent (IDDM) and 2713 as having non-insulin-dependent (NIDDM) diabetes. Nearly all patients diagnosed before the age of 19 had IDDM, but a large proportion (37%) of all diagnoses of IDDM were made after that age. Six percent of all diabetic subjects in the age group 15–19 yr were classified as NIDDM and the proportion increased rapidly in older age groups. Half of the patients with NIDDM were diagnosed over the age of 64. DIABETES CARE 1985; 8:114–17.

Very little is known about the occurrence of insulin-dependent diabetes mellitus (IDDM) in older and non-insulin-dependent diabetes mellitus (NIDDM) in younger age groups.[1] This is evidently caused by difficulties in collecting relevant data with good ascertainment in whole populations. As far as we know the only population-based study of this kind is that of Melton et al.[2] on the incidence of IDDM and NIDDM in the population of Rochester, Minnesota.

The aim of this study was to examine age relationships of IDDM and NIDDM in a cross-sectional study based on medical records of 14 municipal health centers in East Finland.

STUDY POPULATION AND METHODS

The Kuopio University Central Hospital district comprises a population of 250,000 inhabitants. In this district, as in all of Finland, the municipal health center is a functional unit providing basic health services for the population, including diabetes care. There are 23 such health centers in the Kuopio University Central Hospital district. Hospital services, including all main clinical specialties, are also provided by municipalities, and in the Kuopio University Hospital district this is done at the university hospital and three other small hospitals. Only a small number of physicians are in private practice, and in the Kuopio University Central Hospital district almost all of them have their practice in the town of Kuopio.

All medical records of living patients of 14 health centers, covering a total population of 151,000 inhabitants (13 rural health centers, population base of 77,000; the Kuopio town health center, population base of 74,000), were analyzed to identify persons with a diagnosis of diabetes up to 1981. The population living in the municipalities served by these 14 health centers was representative of the whole population of the Kuopio University Central Hospital district with respect to age and sex structure and other relevant demographic characteristics.

The WHO diagnostic criteria for diabetes mellitus were applied.[3] A person was classified as having diabetes if the fasting blood glucose concentration was at least 127 mg/dl on more than one occasion and/or the 2-h blood glucose concentration was at least 180 mg/dl (venous whole blood).

To classify diabetes according to type, the following criteria were applied: IDDM: (1) a person of any age treated initially and thereafter with insulin and (2) ketonuria or ketoacidosis at the time of diagnosis; NIDDM: (1) a person of any age treated initially with diet and/or oral drugs and thereafter with diet only, oral drugs, or insulin (insulin treatment permitted 1 yr or more after the diagnosis) and (2) no ketonuria or ketoacidosis at the time of diagnosis or later.

The degree of ascertainment of diabetic patients in this study was evaluated on the basis of a national register of drug-treated diabetic patients. All diabetic patients in Finland needing drug therapy are provided with medication free of charge according to the Sickness Insurance Act. The Social Insurance Institution maintains a central register of diabetic persons receiving drug reimbursement. In the 13 rural health centers 95% of the diabetic persons identified as treated with drug therapy on the basis of the national register were found. In the town of Kuopio the corresponding figure was only 64% due to a greater proportion of diabetic persons under the care