



EDITORIALS

HEREDITY IN DIABETES

Two recent studies, one by Thompson and Watson¹ in this issue of *DIABETES*, the other by Steinberg and Wilder² in the June issue of *American Journal of Human Genetics*, lend support to the conclusion of Pincus and White³ that predisposition to diabetes, in the great majority of pedigrees, is a simple Mendelian recessive.

The Thompson and Watson data were obtained in London, Ontario, in a study of 1631 diabetics, their parents, sibs and offspring. The data of Steinberg and Wilder were obtained at the Mayo Clinic from a consecutive series of 1981 "new" diabetic patients, i.e., patients who previously had not visited the Clinic with detectable diabetes.

Both of these sets of data, like the data earlier reported by Harris,⁴ disclose a tendency for the diabetic sibs of diabetic patients to become diabetic at the same age as the patients. This, however, as is pointed out in both of the reports, may be a statistical rather than a biological phenomenon. Neither of these newer studies reveals significant association between the sex of the patient and that of his diabetic parent or sib, failing in this to confirm the earlier data of Penrose and Watson⁵ which suggested such association.

The data of Steinberg and Wilder, as analyzed by them, prompted additional interpretations, as follows: 1. The average age of diabetics is unaffected by the sex of the patient. 2. The average age at onset is unaffected by the existence of diabetes in the parents of the patients. It was 47, 48 and 45 years respectively when none, one, or both parents were diabetic. 3. The birth order of the patient is without significance.

In contrast with what might be suspected, these data (as well as those of Pincus and White and of Harris) reveal a slightly greater average family size when diabetes occurs in one or even in both of the parents. The data, however, as is emphasized, relate only to those matings which have yielded at least one diabetic child.

The details of the genetic analysis by Steinberg and Wilder of their data and the data of others, including investigators who had proposed other hypotheses, cannot be presented here. They support the view, as has been said, that predisposition to diabetes is inherited as a Mendelian recessive character. This means that if *d* represents the condition of the gene which leads to a susceptibility to diabetes and *D* its normally functioning alternative condition, diabetics and potential diabetics are genetically *dd* and those not genetically liable to diabetes are either *DD* or *Dd*. The partners of *matings which yield diabetic offspring* may both be nondiabetic

(genetically such a mating is Dd x Dd) or one partner may be diabetic and the other nondiabetic (genetically the mating is dd x Dd) or both partners may be diabetic (genetically the mating is dd x dd). The relative frequency of diabetic offspring from these three types of matings (assuming essentially the same age distribution for the three sets of offspring) is as 1:2:4. The absolute values will depend on the age distribution of the sample; however, on the average *one-quarter* of the sibs of a diabetic patient with *nondiabetic parents* will be genetically predisposed to diabetes; on the average *one-half* the sibs of a diabetic patient with *one diabetic and one nondiabetic parent* will be genetically predisposed, and *all* offspring of matings in which *both parents are diabetic* will be predisposed to diabetes.

The knowledge that all of the offspring of a mating of two diabetics inherit the predisposition to diabetes is of great importance. If all such persons must develop diabetes, given time, they provide the material needed for studies of the prediabetic state. Also, diabetics obviously ought never to marry diabetics. It would be even safer, if they marry, to choose as mates the offspring of persons who are not the children of diabetics.

Steinberg and Wilder, by genetic analysis of their data, arrive at estimates of the prevalence of diabetes and prediabetes. They conclude that diagnosed diabetics constitute about one per cent of the population of the United States, a conclusion in close agreement with those of others, based on surveys. They estimate that those who are genetically dd, that is, the diagnosed, undetected and potential diabetics, approximate five per cent of the population. As the average age of the population increases more prediabetics will have time to manifest their diabetes, and so the prevalence of frank diabetes must continue to increase as undoubtedly it has increased in the last few decades. Here is a challenge to preventive medicine of more significance even than detection of the undiscovered diabetic.

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REFERENCES

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⁴ Harris, H. The familial distribution of diabetes mellitus: A study of the relatives of 1,241 diabetic propositi. *Ann. Eugenics* 15:95-119, 1950.
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FINANCIAL SUPPORT OF RESEARCH

A recent issue of *Science*¹ contained a detailed report on the financial support of medical research in the United States during the past six years. Many readers of *DIABETES* will be interested in certain data contained in the report, and also in information concerning the organization responsible for its preparation.

The Medical Sciences Information Exchange is a cooperative endeavor established in July 1950 within the Division of Medical Sciences of the National Research Council. It is jointly supported by six agencies of the federal government—the Army, the Navy, the Air Force, the Atomic Energy Commission, the Public Health Service, and the Veterans Administration. They maintain it as a clearing house for information on grant and contract support in the medical and allied fields. It compiles data concerning awards for research made by foundations, industries and others, in addition to the government. It is reported that investigators throughout the country, as well as granting agencies, make daily use of the Exchange, an indication of its success in meeting a definite need.

In the report, an analysis was presented of 12,923 research grants registered with the Medical Sciences Information Exchange during the period 1946 through 1951. In these six years \$135,044,125 was awarded, \$83,110,671, or 61.5 per cent, from government and \$51,933,454, or 38.5 per cent, from private sources. The annual funds increased from 4 million dollars in 1946 to 33 million in 1949, and have remained at approximately this level. Government support, 44 per cent in 1946, has steadily increased until in 1951 it provided nearly 66 per cent of the total support of medical research. Funds from private sources increased from 2 million in 1946 to 11 million in 1951; apparently increasing government support of medical research has not diminished the efforts of the private foundations.

Of special interest is the information concerning re-

TABLE I Expenditures for research in diabetes

Year	Government	Private	Total
1946	\$ 0	\$ 6,700	\$ 6,700
1947	25,580	24,800	50,380
1948	141,908	45,411	187,319
1949	242,622	54,841	297,463
1950	309,985	70,771	380,756
1951	353,544	74,505	428,049
1946-1951	\$ 1,073,639	\$ 277,028	\$ 1,350,667