Genetics and Public Health

N. C. MYRIANTHOPOULOS, Ph.D.

The epidemiological-genetic approach is ideally suited to investigations concerning the estimation of gene frequencies and mutation rates, the selective advantage of certain genotypes, the detection of heterozygous carriers, the metabolic etiology of genetic disease, and the practice of genetic counseling. Progress in any of these areas has numerous public health applications.

THE INCREASING application of human genetics in the fields of epidemiology and preventive medicine has brought about a necessary collaboration of these disciplines on a close and equal basis. Efforts to understand, control, prevent, or eradicate disease are greatly aided by the advances in genetics during the past 25 years and the growing appreciation of the human host as an important factor in the etiology of disease. The ensuing reevaluation and realinement of concepts regarding etiology are well illustrated by Shimkin (*1*), who, in a recent review on the etiology of cancer, writes:

"There are few, if any, simple or single causes in biology. There are, instead, complex situations and environments in which the probability of certain events is increased. The probability of the neoplastic event following exposure of an organism to a carcinogenic stimulus is modified by a large series of factors. In regard to the stimulus, among the more evident influences are those of dose, route of exposure, physical state of the material, and length and schedule of exposure. In regard to the host, the probability of the neoplastic reaction is influenced by the genetic background, age, sex, nutritional status, and intercurrent infections. The longer the period between initial exposure and the end point of the reaction, the more opportunities occur for the introduction of additional modifying factors."

It is not too farfetched to claim that all diseases have a genetic component. Heredity, of course, does not operate in a vacuum. The environment influences its expression to a greater or lesser degree, so that it is well nigh impossible to differentiate between "purely genetic" and "purely environmental" disease. In this context the human condition appears as a continuum formed by the interaction of genetic and environmental influences. At one end of the continuum are those diseases in which the genetic factors are most powerful and the influence of the environment negligible; these are illustrated by Huntington's chorea or Tay-Sachs disease. At the other end are those diseases in which environmental factors play the chief role while genetic elements are least influential; these are exemplified by infectious diseases whose occurrence depends on the presence of the infectious agent. Between these two extremes, the genetic and environmental influences operate with varying degrees of interaction in the causation of disease.

When diseases are observed to occur in familial aggregations, it is desirable to clarify the relative roles of heredity and common exposure to some environmental stress. Neel and Schull (*2*) advance four criteria by which the influence of genetic factors in the etiology of disease can be detected: (*a*) the occurrence of the disease in definite numerical proportions among individuals related by descent; (*b*) failure to "spread" to nonrelated individuals; (*c*) onset at a characteristic age without a known precipitating cause; and (*d*) greater concordance in identical twins than in fraternal twins. These

Dr. Myrianthopoulos is a geneticist in the Epidemiology Branch, National Institute of Neurological Diseases and Blindness, Public Health Service.

criteria must be applied with extreme caution and critical judgment, for the pitfalls of error are many. For years, for example, because syphilitic women tended to have syphilitic babies, the disease was considered hereditary, although it is now realized that the infant is infected during gestation. Even in a disease such as tuberculosis, although predisposition may be genetically determined, the aggregation of many cases in a family is generally conceded to be a result of common exposure.

In the study of the natural history of a disease, epidemiology is, in a sense, the intelligence service of medicine. It provides information on the extent and distribution of the disease by analyzing its prevalence and incidence, mortality, age and sex distribution, geographic distribution, periodic occurrence, and association with other diseases and environmental factors. Attempts to control, prevent, and eradicate disease depend largely on such epidemiological studies to guide the attack on the causative factors or to strengthen the host and his environment, or both.

Genetics has become an adjunct to both of these fundamental phases of public health: epidemiology and preventive medicine.

Genetics and Epidemiology

The circumstances which have brought genetics and epidemiology together result in part from increasing attention to those usually chronic diseases in which host or genetic factors appear to be relatively important. The incorporation of genetic concepts and techniques in epidemiological research has added a new dimension to medical demography: the assessment of the genetic endowments of populations.

Human genetics has developed in two areas; each stems from the same principles and laws, but operates on a different level. One, the so-called "pedigree genetics," deals with the individual case, the individual family, or a group of families, and is more directly concerned with preventive medicine. This will be discussed later. The other, "population genetics," transcends the individual case and operates at the population level. It deals with the dynamic balance between mutation and selection in relation to the inherited traits of man, that is, with population characteristics, which are the stock-in-trade of the epidemiologist.

Following are some of the fields where the geneticist and the epidemiologist together study the natural history of genetically determined disease.

Estimation of Gene Frequencies

Knowledge of the presence of a gene which determines a disease is only a first step toward the study of its natural history. We like to know next the prevalence rate, as this is an indispensable tool for the estimation of the gene frequency by genetic theory and method. Suppose a disease determined by a pair of autosomal recessive genes has a prevalence rate of 1 per 30,000 population in the United States. Such a disease is phenylketonuria (*3*). According to the Hardy-Weinberg law, the gene frequency of this disease (q) is 1 in 173 and the carrier frequency (2pq) is 1 in 86. In other words, 1 person out of 86 in the population is a carrier of the phenylketonuria recessive gene. In England the prevalence rate of phenylketonuria is half that of the United States, 1 per 60,000 population (*3*). The gene frequency of this disease in England is 1 in 215, and one person out of 122 is a carrier of the recessive gene for phenylketonuria. (Actually the frequencies of phenylketonuria as estimated by Jervis in the United States and Munro in England are 1 in 25,000 and 1 in 50,000 respectively. The figures 1 in 30,000 and 1 in 60,000 presented here were derived by excluding those cases known to have consanguineous parents.)

An estimated gene frequency may not apply to the whole population for which it was estimated because the gene may be present in greater frequency among some subgroups of the population than others. The study of the frequency of thalassemia in Rochester, N.Y., is an example (*4*). It was found that 11 out of 100,000 children born in the city between 1928 and 1942 were affected with the disease, and from this it would appear that the frequency of the heterozygous carriers would be about 1 out of 50 persons. It so happens, however, that there is a rather large isolate of Italian descent in Rochester, and the 11 cases were of children born within this group. The estimate of the thalassemia gene, therefore, would apply only

Custom-Built Heredity?

Recent advances in research in genetics and biochemistry are described in a series of three articles, which began publication in the September 30, 1959, issue of *Scope Weekly*, as offering high hopes for public health.

In effect, the report says the studies give promise of almost unlimited uses of chemistry to correct physical deficiencies and to strengthen the genetic strain. It notes that awareness of enzymatic defects in metabolism has been coupled with methods of repairing such chemical flaws. Moreover, it cites the discovery of the principle of transduction, the transfer of genetic material from one cell to another, as suggesting possibilities of strengthening resistance to parasites, as well as enlarging the mental and physical potentials of humanity.

Basic ideas that have become apparent from such research were defined by Dr. Edward Lawrie Tatum of Rockefeller Institute, who along with Prof. George W. Beadle of the California Institute of Technology and Dr. Joshua Lederberg, then at Stanford University, was a Nobel medical laureate in 1958. Tatum listed the following concepts:

All biochemical processes in all organisms are under genic control.

These processes are resolvable into a series of individual stepwise reactions.

Each single reaction is controlled in a primary fashion by a single gene: there is a one-to-one correspondence between gene and biochemical reaction.

Therefore, the mutation of a single gene results only in an alteration in the ability of the cell to carry out a single primary chemical reaction.

"Perhaps within our lifetime," he said, "the code of life processes tied up in the structure of proteins and nucleic acids will be broken. This may permit the improvement of all living organisms by processes which we might call biological engineering."

In the same article, Dr. Laurence H. Snyder, president of the University of Hawaii, said that biochemistry and genetics offer a sound approach to diagnosis, prevention, and therapy.

He said there are "reasons for believing that genetics is involved in one way or another in the development of all disease."

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to the Italian isolate. The total number of births in the isolate during the 15-year period was estimated to have been in the order of 26,000. The frequency of thalassemia in the Italian subpopulation, then, becomes 1 in about 2,400 and the frequency of the heterozygous carriers 1 in 25 persons.

Another case in point is the frequency of the recessive gene for Tay-Sachs disease which has long been suspected of occurring more frequently among Jewish than among non-Jewish people. Kozinn(*5*) and his associates in a recent study in the New York City area estimated that the gene frequency for Tay-Sachs disease among Jews is about 1 in 100 and the frequency of carriers about 1 in 50, while for non-Jews the gene frequency is about 1 in 660 and the frequency of carriers about 1 in 300. Another study now being conducted by the National Institute of Neurological Diseases and Blindness, Public Health Service, shows the same trend. Since the homozygote in Tay-Sachs disease dies before reaching reproductive age, only matings between heterozygous carriers will produce affected children. The chance that two Jewish heterozygotes will marry is the product of their separate probabilities of carrying the gene, or 1 in 2,500. The chance, however, that two non-Jewish carriers will marry is only 1 in 90,000 which, admittedly, is very remote. The task of control and prevention of Tay-Sachs disease is quite different in magnitude and intensity in the two groups.

Estimation of Mutation Rates

Nature tends to maintain a balance of forces between selection and mutation. Selection against deleterious genes occurs either naturally or by eugenic practices. Mutation, however, reintroduces these deleterious genes in the population at a specific rate. It is of great importance, therefore, to know the rate at which normal genes mutate in the population if their control or elimination rate is to be ef-

fective. The tools needed here again are both epidemiological and genetic, for any attempt to arrive at an estimate of the mutation rate of a gene must stem from an accurate knowledge of the incidence rate of the disease determined by the gene in question.

Progressive muscular dystrophy of childhood may serve to illustrate the point. This type of muscular dystrophy is inherited in a sex-linked recessive manner; that is, it occurs only in males who receive the "defective" gene, located on the X (sex) chromosome, from their mothers. The mothers of affected boys act as genetic carriers but are not themselves affected because the defective gene is masked, so to speak, by the normal gene, or allele, located at the same position on the other X chromosome. (Females have two X chromosomes, while males have only one.) Thus mothers who are genetic carriers for childhood progressive muscular dystrophy pass on the gene to half of their sons, who develop the disease, and to half of their daughters, who in turn act as genetic carriers.

The onset is usually in about the third year of life and its progress is so rapid that the affected children are invalids by the time they reach 10 to 12 years of age. Few live past the age of 20 years. In spite of the fact that the gene is eliminated with the death of the affected individual before he reaches reproductive age, the frequency of the disease does not change appreciably in the population, and a rather high mutation rate would be required to maintain it at a constant level. Stephens (*6*) and his associates estimated the mutation rate by ascertaining the number of dystrophics born in Utah during the 10-year period 1931–41. There were 18 such cases, and 6 of these were considered as new mutations because there was good evidence that the gene had not been passed down through a line of female carriers to the affected individuals. Approximately 126,000 children were born in the State during that period. If half of them were males, then 6/63,000 gives the approximate mutation rate per X chromosome per generation. The estimate is 9.5 x 10^{-5}, or about 1 mutation per 10,000 male births.

The epidemiological-genetic cooperative approach during the last few years has been instrumental in enabling us to estimate the mutation rates of the genes responsible for many diseases. Some of these estimated mutation rates in man are summarized below:

Condition produced by gene	*Mutation rate per million genes per generation*
Autosomal dominant:	
Epiloia	8
Chondrodystrophy	42
Pelger's nuclear anomaly	80
Aniridia	5
Retinoblastoma	14
Autosomal recessive[1]:	
Albinism	28
Congenital total color blindness	28
Tay-Sachs disease	11
Ichthyosis congenita	11
Cystic fibrosis of the pancreas	700
Amyotonia congenita	20
Sex-linked recessive:	
Hemophilia	32
Pseudohypertrophic muscular dystrophy	95

[1] The general feeling among investigators currently is that mutation rate estimates for autosomal recessive genes may not be valid because of unknown heterozygote effects. If, for example, the heterozygote has a slight advantage sufficient to perpetuate a rare recessive lethal gene in the absence of mutation, then the estimate of the mutation rate would appear to be too high.

Selective Advantage of a Genotype

It has been known from experimental work in lower animals, especially in *Drosophila*, that the heterozygous condition of certain traits may have some selective advantage, that is, be reproductively superior, over either dominant or recessive homozygote. This phenomenon is known in genetic terms as "balanced polymorphism." Although it has been suspected for a long time that balanced polymorphism may operate in man, the first instance of such selective advantage in a human population was dramatically demonstrated by Allison (*7*) in 1954 with his work on sickle-cell anemia. This disease is genetically determined by a pair of incompletely dominant genes (genes which in the heterozygote produce detectable phenotypic effects). In the homozygous condition, the gene results in a severe, chronic hemolytic anemia which is often fatal before puberty.

In the heterozygous condition, the clinical picture is known as sickle-cell trait and results

in a variable syndrome which is compatible with survival. The gene is apparently much more frequent in Negroes than in whites. It is present in 10 percent of American Negroes, and 1 out of 500 of them is affected with sickle-cell anemia. Allison noted that in spite of the mass elimination of the recessive gene because of its lethality in the homozygous condition, its frequency was abnormally high in certain regions of Africa. This could be explained either on the basis of a high rate of mutation which would reintroduce the gene in the population or by attributing to the heterozygote some selective advantage. Indeed, Allison showed by both observation and experiment that in some regions of central Africa in which the frequency of the gene was very high, the heterozygous state afforded protection from falciparum malaria, which is endemic in those areas. Allison also observed that sickle-cell trait frequencies of 30–40 percent occur in geographically widely separated tribes in Africa and that these tribes are characteristically found in intensely malarious regions. He calculated that the reproductive fitness of the heterozygote must be 1.26 times that of the normal homozygote for an equilibrium to be maintained under these conditions.

Investigations along these lines have barely begun. It is possible that the distribution of many other genetic diseases and the gene frequencies that perpetuate them can be explained by balanced polymorphism. The question should be pursued by epidemiological-genetic methods.

Effects of Geographic Factors

It is well known and accepted as axiomatic that heredity alone is incapable of producing any character; the character in question will appear only if an optimal environment makes its expression possible. A study of environmental conditions, therefore, is essential for the complete understanding of the natural history of a genetically determined disease.

One environmental factor is geographic. Recently Mackay and Myrianthopoulos (*8*) presented evidence favoring genetic determination of multiple sclerosis. Although they could not postulate a mode of inheritance, they noted, after preliminary analysis of the affected siblings and first cousins of their propositi, that the hereditary factors involved must operate with greatly reduced penetrance. Earlier, Kurland and Westlund (*9*) demonstrated that multiple sclerosis was more prevalent in cold northern climates than in warm southern ones. It is possible that a correlation exists between penetrance and geographic distribution in multiple sclerosis.

Geographic isolation continues to be one of the most important factors in differential distribution of gene frequencies. On the island of Guam, for example, amyotrophic lateral sclerosis is responsible for approximately 10 percent of the deaths, while in the United States amyotrophic lateral sclerosis is estimated as the cause of death in only 0.1 percent of the population. There is good reason to believe that the majority of cases on Guam are genetically determined, while in the United States only a small proportion of cases are found to have positive family history. Whether the geographic isolation, the population size, or other environmental conditions are responsible for the high prevalence of amyotrophic lateral sclerosis on Guam, it is the natural responsibility of the epidemiological-genetic team to investigate.

Genetics and Preventive Medicine

The contributions which genetics has made to many facets of preventive medicine, especially diagnosis and treatment, are well recognized. For example, understanding of the genetics of the Rh factor by physicians and parents has aided in saving thousands of newborn babies that might have perished from erythroblastosis fetalis. Most physicians now routinely include a "family history" in the anamnesis of events of their patients' histories, and some even consider it a deciding factor in differential diagnosis.

Other possible applications of genetics to preventive medicine are not as widely appreciated. In many respects they constitute an untapped source of valuable information in the fight against disease.

Detection of Heterozygous Carriers

Heterozygous carriers, as the term is used here, means not only the carriers of recessive

genes in the heterozygous condition but also the carriers of dominant genes for diseases which have a late onset, such as Huntington's chorea or progressive muscular atrophy. The importance of being able to recognize these individuals who have the potentiality either to produce affected children or become affected themselves is self-evident.

In our search for signs or symptoms by which we would be able to identify the heterozygote, we are presently limited to the observation of recognizable morphological deviations or to the identification of detectable biochemical deviations from the normal. More often than not, these are elusive either because they are so minute as to be considered insignificant or because they occur in areas in which they are inaccessible and unsuspected. In spite of these difficulties, progress toward the recognition of the heterozygous state has been considerable for a good number of diseases. In some cases the heterozygote can be detected with 100 percent accuracy. In others, the degree of accuracy is much less.

On the basis of our ability to recognize the heterozygous state, genetic diseases can be classified in three categories.

First are those conditions in which the heterozygous state produces a recognizable clinical picture, albeit not as severe as that produced by the homozygous conditions. In this category belong the by now classic sickle-cell anemia which in the heterozygote is manifested as a sickle-cell trait; and thalassemia major which in the heterozygote is manifested as thalassemia minor.

Second are those conditions in which the carrier state is characterized by a subclinical effect with corresponding slight phenotypic change which can be detected with appropriate known and available tests. In this category belong gout whose carrier state shows only hyperuricemia; hereditary hemolytic jaundice which in the carrier state is characterized by asymptomatic sperocytosis; xanthomatosis in which the heterozygote shows hypercholesterolemia.

Finally are those conditions for which the carrier state can be recognized in some but not in all cases. Dystrophia myotonica, Huntington's chorea, galactosemia, Friedreich's ataxia, hemophilia, and a score of other conditions belong in this group. For some of these the rate of the detection of the heterozygote comes close to the genetic expectation while for others the degree of accuracy is small. This is not surprising since, as previously mentioned, for most of these conditions the detection of the heterozygote probably depends on the recognition of minute or subtle biochemical deviations which presently available techniques cannot assess accurately.

Neel and Schull (*2*) compiled a list of diseases in which it may be possible to recognize the carrier state. They devised a grading system of 1–4 to indicate the reliability of recognition of the carrier state, grade 1 being the most reliable and grade 4 the least reliable.

Whatever information is available now, even for diseases in the less reliable grades, can be very useful, when interpreted by an experienced worker, in preventive medicine and especially in genetic counseling. With the perfection of such methods as tolerance tests and the refinement of bioassay methods, it may become possible to detect accurately the heterozygote for many genetic diseases.

Genetics and Metabolism

Modern genetics has particularly studied the ways in which genes act. There is abundant accumulated evidence that genes produce their effects through metabolic pathways. The original investigations in this field by Beadle and Tatum with the mold Neurospora won them the Nobel Prize for medicine in 1958. Actually, Sir Archibald Garrod can be regarded as the prodrome of biochemical genetics, for he first drew attention to what he called "inborn errors of metabolism" as far back as 1908. The inherited diseases based on metabolic abnormalities which Garrod described were: alcaptonuria, cystinuria, albinism, porphyrinuria, and pentosuria.

Today a large number of diseases are believed to result from hereditary flaws in protein, carbohydrate, or fat metabolism. For a good number of these diseases, investigators have identified the enzymatic level where occur the metabolic blocks controlled by genes. It is easy to see the significance of such precise knowledge for now the problem can be attacked

at its roots. Two examples cited here illustrate the possibilities.

Phenylketonuria is a metabolic disorder in which, due to metabolic deficiency, there is a failure of transformation of phenylalanine to tyrosine. This block is controlled by a pair of autosomal recessive genes. The molecule whose dysfunction is responsible for this failure is a liver enzyme concerned with the oxidation of phenylalanine. The disturbance is present at birth and is characterized by elimination of phenylpyruvic acid in the urine, various neurological signs, and mental deficiency. Several attempts are now being made to treat children suffering from phenylketonuria with a special diet, free of phenylalanine. Although it is early yet for full evaluation of this treatment, encouraging results have been reported. The present status of the situation has recently been reviewed by Knox and Hsia (*10*).

Galactosemia, or idiopathic galactosuria, is another metabolic disorder of infants apparently dependent on a pair of recessive genes. It is the result of an inability to convert galactose, a component of milk sugar, into glycogen due to a decrease or absence of the hepatic enzyme Gal-1-P uridyl transferase. The disease is accompanied by severe clinical symptoms such as failure to gain weight, hepatomegaly, jaundice, diarrhea, vomiting, albuminuria, and zonular cataract. When galactosemia is correctly diagnosed, it is often enough to remove galactose from the diet and the clinical manifestations, which otherwise may have serious consequences, disappear.

An opinion shared by many geneticists and biochemists is that all genetically determined diseases are metabolic in origin. If this be true, then as biochemical techniques become more refined and precise, research should be able to uncover the underlying metabolic defect not only in the affected individuals but also in the heterozygous carriers.

Genetic Counseling

Genetic counseling not only has potential importance in the prevention of genetic disease, but also helps to dispel false fears about hereditary traits. The primary function of genetic counseling is to provide people with information regarding their genetic problems. The physician should be the person most qualified to give genetic advice. He is thoroughly versed in medical matters and also knows intimately the personalities and needs of his clients. Unfortunately, few physicians are trained to give professional advice in genetic problems. Medical schools seldom offer formal training in medical genetics. Neither do they ask for courses in general or human genetics as entrance requirements.

Some of the institutions which have given serious consideration to the teaching of human genetics and the training of scientists in this field have established genetic counseling centers where physicians can refer their patients for counseling and where genetic problems per-

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Genetic Counselors and Counseling Centers in the United States and Canada

P. DAVID: University of Oklahoma, Norman.
F. C. FRASER: Department of Medical Genetics, Montreal Children's Hospital, Canada.
E. J. GARDNER: Department of Zoology, Utah State Agricultural College, Logan.
C. N. HERNDON: Department of Medical Genetics, Bowman Gray School of Medicine, Winston-Salem, N.C.
F. KALLMANN: New York State Psychiatric Institute, New York City.
H. W. KLOEPFER: Tulane University, New Orleans, La.
N. C. MYRIANTHOPOULOS: Genetic Counseling and Research Center, George Washington University Hospital, Washington, D.C.
J. V. NEEL: Heredity Clinic, University of Michigan, Ann Arbor.
C. P. OLIVER: Genetics Foundation, University of Texas, Austin.
S. C. REED: Dight Institute, University of Minnesota, Minneapolis.
F. E. STEPHENS: Laboratory of Human Genetics, University of Utah, Salt Lake City.
C. STERN: University of California, Berkeley.
K. A. STILES: Zoology Department, Michigan State College, East Lansing.
N. F. WALKER: Hospital for Sick Children, Toronto, Canada.

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taining to the individual, the family, and the community can be handled. There are about a dozen or more such centers in the United States, less than 1 per 10 million people (see list).

No two of the problems which confront the genetic counselor are the same even if they involve identical conditions. Obviously, the people who seek genetic counseling differ in each case with respect to physical and mental makeup, social and economic background, educational level, religious upbringing, and emotional content. Requests for counseling may be anticipatory, as with people contemplating marriage who know that a certain disease exists in one of the families. They would want to know what the chances are that their future children may inherit this disease. Some persons seek genetic counseling inevitably "after the fact," that is, after they have had one or more affected children. In these cases, the condition is usually recessive and the parents not affected. They find that they are genetic carriers when they have one affected child, and they are concerned about the chances of repeating their misfortune.

Many requests for counseling concern children to be placed for adoption. The adoption agency or the prospective parents want to know whether the child is a good adoptive risk, especially if it is known that there has been some undesirable trait in the child's family, such as epilepsy or mental deficiency.

The counselor's job is often complicated by situations which result from irregular action and expression of genes. Some conditions, such as peroneal muscular atrophy, show reduced penetrance; others, such as Friedreich's ataxia and retinitis pigmentosa, are inherited in more than one way; in still others, such as central nervous system malformations, the complex interaction between heredity and environment distorts the genetic ratios to such a degree that a precise evaluation of each component is impossible. The counselor has to consider all of these, and at times he has to resort to empiric risk figures in order to give effective advice. As Reed (*11*) put it, "It has been our lot to struggle with such complicated traits as schizophrenia, in which the heredity seems to be that of an incomplete dominant with incomplete penetrance. This sounds too incomplete for words and the impatient soul may decide to toss out the genes altogether."

These difficulties are listed here only to show that the quality of genetic advice depends on thorough mastery of the subject matter in all its known intricate details. Genetic counseling without competent and scientific training in this field may be disastrous to the individual, defeating the whole objective of the profession.

Comment

It is evident that genetics has become an important adjunct to public health practice, teaching, and research. Even so, in some of the areas, such as the detection of the heterozygote or the understanding of the metabolic etiology of disease, the surface has barely been scratched.

The manpower needs are now acutely felt. There is a serious shortage of well-trained human and medical geneticists. In the United States and Canada hardly a dozen institutions offer adequate training in human genetics. There is need for the establishment and support of many more training institutions. There is need for the introduction of courses in human and medical genetics in our medical and public health schools. There is need for the establishment of genetic counseling centers at convenient locations to serve all the people. Only such efforts can realize the potential of genetics in contributing toward the reduction of chronic disease and improvement of public welfare.

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Signs and Symptoms

Farm women in Maryland received instruction this summer in safe handling of gasoline engines, tractors, and other farm equipment, at a series of classes conducted throughout the State by Guy W. Gienger, agricultural engineer with the University of Maryland.

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Foster homes for New York City's aged have been provided for nearly 15 years by the New York City Department of Welfare. A few voluntary agencies also have foster home programs on a limited scale.

Foster homes meeting prescribed standards are needed for those older or handicapped individuals who require a semiprotected living arrangement but who neither desire nor need the institutional setting for congregate living.

Proprietors of foster homes in the Department of Welfare's supervised program currently receive $100 a month for single accommodations and $90 a month when two boarders share a room. The fee includes room, board, and personal service which may be required by the older or handicapped person.

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Yellow fever can be diagnosed in experimental animals within 24 hours after infection by means of histopathological examinations and use of the fluorescent antibody technique, Dr. H. F. Smetana of the Armed Forces Institute of Pathology, Washington, D.C., told a joint meeting of the American Society of Clinical Pathologists and the College of American Pathologists in Chicago.

A full report of papers delivered for the Council on National Defense at the American Medical Association meeting, June 6, 1959, appears in the September 12, 1959, issue of the *Journal of the American Medical Association*. The same issue carries a report on the epidemiology of mental health by Milton Olin, which concludes with a quotation from Dr. Will Menninger: "We live in a turbulent world, a crazy world, with many evidences of man's hostility to man . . . . But it is our world; it is what we are making it; and its course depends on the responsibility that you and I assume for it."

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Automobile accidents caused approximately 35 percent of all deaths of persons aged 15 through 24 years in the United States during 1957, according to the accident prevention program of the Public Health Service.

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The first patients have been admitted to New York State's research unit in narcotic addiction at Manhattan State Hospital, Ward's Island, New York City. According to Commissioner of Mental Hygiene Paul H. Hoch, the unit is the first full-time narcotics research unit in the State combining laboratory investigations and outpatient and inpatient operations. There are 55 beds for inpatients, and about 150 outpatients can be treated.

Organized for research purposes, the unit's staff will concentrate on basic investigations in an effort to determine causes of addiction and to improve treatment methods.

A wristwatch with a band too tight for the wearer was found by Dr. Howard R. Bierman to be the cause of a progressive unilateral neuritis of the thumb and index finger in an otherwise healthy woman, it was reported in the *New England Journal of Medicine*, July 30, 1959.

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Foresighted Florida physicians started in 1873 to campaign for a State board of health, but it took 16 years and Jacksonville's paralyzing yellow fever epidemic in 1888 to bring the board into being. This and other events in the history of public health in Florida came to light recently when a wooden packing case labeled "for posterity" and containing correspondence from the early days was found under the eaves of a State building. The origins and growth of public health in Florida were reviewed in the September 1959 issue of *Florida Health Notes*, introduced in July 1892 as a personal project of Dr. Joseph Y. Porter, the first State health officer, and his assistant, Dr. Hiram Byrd.

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The art of screening for glaucoma, a combined effort of health officials and the Lions Club of Brookline, Mass., is described by John G. McCormick in *Health Educators at Work*, May 1959. From the basic planning and preparatory information campaign, through the personal courtesies provided at the clinic and evaluation, this is a lucid, blow-by-blow community case history.