trial Research of the University of Pittsburgh: Dr. George E. Barker, senior industrial fellow; George E. Alter, industrial fellow, and Charles E. McKnight, fellowship assistant. Other specialists will be added to the staff as the research progresses. A broad survey of the problems involved is being made by Dr. Barker, with the cooperation of the Navy Department, various American instrument manufacturers and other Federal Government laboratories.

WARD'S NATURAL SCIENCE ESTABLISHMENT, INC., which for over eighty years has been conducted in Rochester, N. Y., has moved to a new site on a sixty-five acre tract of land overlooking Irondequoit Bay and Lake Ontario, outside the Rochester city limits. Thirty-five acres are planted with grapes, the remainder being in woodland and pasture. Numerous springs furnish an abundant supply of pure water for cultures and other living material. The departments of mineralogy, paleontology, biology, entomology, and microscope slides and models are housed in a two-story concrete building, and the offices, mailing and photography departments in a large residence near the main building. There are seven other houses on the property and these will eventually be occupied by the Ward employees.

DR. C. P. RHOADS, author with Professor Louis F. Fieser and others of the article entitled "Steroid Hormone Excretion by Normal and Pathological Individuals," in the issue of SCIENCE for May 22, wishes to acknowledge the indebtedness of the authors to the Commonwealth Fund and to the Jane Coffin Childs Memorial Foundation for Medical Research.

DISCUSSION

IN DEFENSE OF THE KALLIKAK STUDY

THE story of the Kallikaks was published in 1912. The larger book, "Feeble-Mindedness: Its Causes and Consequences,"[1] giving the data which seemed to establish the hereditary character of feeble-mindedness, appeared in 1914.

For a decade the data were accepted apparently without question. There seems to have been enough people who were familiar with the details to explain how the study was made, the methods used, the conditions existent, and to answer any questions that arose. But as time went on, the inevitable happened and writers appeared who did not know, who obviously had not read the originals, and who therefore thought they detected certain flaws in the techniques which did not exist.

The first of these appeared in 1925 in "The Inheritance of Mental Disease," by Abraham Myerson, M.D. (pp. 77 *ff.*).[2] To this I paid no public attention because I felt that it was so obviously prejudiced that it would do no harm.

However, fourteen years later, a second book appeared which repeated and added to the errors of the Myerson book. This occurred in 1939 in "You and Heredity," by Amram Scheinfeld (pp. 360 *ff.*).[3]

Again in 1942 the same errors are copied from the preceding sources in "Biology for Better Living," by Ernest E. Bayles and R. Will Burnett (pp. 610 *ff.*).[4]

It therefore seems necessary to correct the errors publicly and attempt to set the record straight.

[1] Henry H. Goddard, "Feeble-Mindedness: Its Causes and Consequences." Macmillan. 1914.

[2] Abraham Myerson, "The Inheritance of Mental Disease." Williams and Wilkins. 1925.

[3] Amram Scheinfeld, "You and Heredity." Frederick A. Stokes Company. 1939.

[4] Ernest R. Bayles and R. Will Burnett, "Biology for Better Living." Silver Burdett Company. 1942.

While much in the way of polish is lacking in this pioneer study, there are certain universal techniques which must be vigorously applied in any careful study; and to certain criticisms of these in Dr. Myerson's book, I wish to refer.

On page 77, Dr. Myerson, in a few lines, makes his only reference to "Feeble-Mindedness: Its Causes and Consequences"—the book which alone contains the data which led to the conclusion that feeble-mindedness is generally hereditary. The Kallikak family is merely a striking illustration.

Dr. Myerson says: "In this book, Goddard decides that feeble-mindedness is a Mendelian trait. He cites some 100 cases in which family studies have been made."

The record shows there are 327 cases carefully studied, charted and explained.

A few lines farther on, Dr. Myerson says: "The keystone of the arch of their results and laws is the field investigator and her surmises as to the mental and physical state of the dead and the quick."

Not understanding the purpose or the methods of the field-worker, Dr. Myerson makes his own assumptions. He argues that because he can not correctly diagnose feeble-mindedness, nobody can. Therefore, all our diagnoses must be guesses and "surmises."

The record shows that our field-workers were carefully trained (see "Feeble-Mindedness," pp. 22–46, 293 and 352). They spent weeks and months in the institution, talking with and observing all grades of defectives. It is well known that superintendents of such institutions quickly learn, and when a new arrival appears they not only know whether he is a fit subject for their institution or is normal and does not belong there, but they also know his *grade*. Even the attendants acquire this ability rather quickly. Dr. Fernald used to enjoy telling how his attendants

would spot a child on the train and report that a new case was on the way.

But these are cases that were *seen*. How about cases in earlier generations, no longer living? Here again Dr. Myerson does not understand—although it is explained in the above references.

The field-worker does not make any "surmises," nor does she ask for anybody's "opinion." Such a method would be naïve indeed. She first asks the prospective informant: "Do you (or did you) know such a person?" If the answer is yes, she proceeds to ask many questions as to how he behaved, what he did, how he managed his affairs.

From this she gets an accurate picture of the kind of person he was. But she does not stop there. This informant may be prejudiced. His account must be corroborated. She hunts up everybody who knew the case. Finally she knows whether he was feeble-minded or normal. If she does not get enough information to decide without a reasonable doubt, the case is "undetermined," and is so marked. Dr. Myerson perhaps did not notice the many squares and circles that were not marked either "F" or "N." The figures are important. On the 327 charts there are 3,996 individuals marked normal (N), 1,946 marked feeble-minded (F): 476 doubtful (?): and 5,892 undetermined (left blank).

Moreover, we are not left without a check-up. After all the individual cases were finished and the charts made, we worked out the "expectation" according to the Mendelian principle, for five different kinds of matings; *e.g.*, if both parents were "F" all the children should be "F." We added all the children of such matings. The record shows 482 children whose status was determined. According to "expectation" they should all be "F." According to our determination, 476 were "F" and 6 were "N." Either we were in error to the extent of six cases, or there was some illegitimacy, or, as some biologists hold, two "F" parents may *rarely* have an "N" child.

The four other kinds of matings gave results equally close to the "expectation." This is all fully explained in "Feeble-Mindedness."

Dr. Myerson next turns his attention to the "Kallikak Family."[5] He ridicules the idea that we could know that the mother of the Kallikaks was feeble-minded, when we "did not even know her name," but had to put her down as "Nameless." I did not realize that it might mislead. All names are fictitious, and it occurred to me that "nameless" would identify her without any possibility of confusion. She is nameless to the reader only. We had her name; and not only her name but her history. We were fortunate enough to find an intelligent lady of advanced age, *who knew personally the "Nameless one."* That seems impossible until one realizes that if each of them had lived to be 80, they could have known each other for eight years or more; and if they had lived to be 90—not impossible—they could have been neighbors for 30 years! The nameless one was neither a prostitute nor a "bar-room habituée."

Perhaps Mr. Scheinfeld can explain how he acquired the information that "In 1898 Dr. H. H. Goddard, then director of an institution for mental defectives in New Jersey, chanced upon the strange fact, etc."

It is no misprint, because he repeats with emphasis on the next page "Remember, this study was begun in 1898, before there was any science of genetics."

The facts are these: I was never director of an institution for mental defectives and I did not begin the study of the Kallikaks in 1898. It was begun in 1910 and published in 1912.

Mr. Scheinfeld contributes one original idea—wrong like the rest. He says: "The comparison rests largely on *the assumption* (italics his) that the illegitimate child whom the feeble-minded mother chose to call Martin Kallikak, Jr., was indeed the son of the man she designated, which no court would accept as evidence."

A strange statement. Courts have always accepted such evidence and still do. In this case there was not even a doubt.

For the rest, Mr. Scheinfeld devotes a few paragraphs to prove the obvious.

Certainly Martin Kallikak, Sr., must have been a "Simplex," else his son by the "Nameless" would have been normal. But that is no argument. It is well known that a trait may remain recessive for generations as long as its possessors mate with "duplexes."

Finally we come to Bayles and Burnett. Their treatment of the matter is so much like Scheinfeld's that it has already been answered. With parrot-like accuracy they repeat Scheinfeld's error: "In 1898 Dr. H. H. Goddard began a study of two family lines that had a common ancestor," and on the next page, "Remember that the study was begun in 1898." They add, "Remember that Mendel's work was not known to the scientific world until 1900." And in the next paragraph, "Intelligence tests did not even exist when the study was made."

They are so sure of all this that they add "The persons in Goddard's study were classed as feeble-minded simply upon the basis of opinions of persons who had known them or who knew (however vaguely) of them." (Reminiscent of Dr. Myerson!)

The record shows that the Binet tests were extensively used by us in both studies. (See "Kallikak Family," pp. 71 and 88, also "Feeble-Mindedness," pp. 4 and 183, and see index.)

That these authors copied, uncritically, Scheinfeld's errors, including the absurd date, seems ob-

vious. Even a cursory reading of the "Kallikak Family"[5] or "Feeble-Mindedness" would have shown them that intelligence tests were in constant use.

Perhaps it is possible to regard these glaring errors as natural mistakes; but it is difficult not to feel that some, at least, result from wishful thinking.

In these days, one can not read everything. But if one feels it necessary to publicly criticize, it would seem that he should be sure that he understands what he is condemning. This would be not only for his own protection, but for the far more important consideration, the preservation of truth and the advancement of science.

HENRY H. GODDARD
THE OHIO STATE UNIVERSITY

THE GRAYING OF HAIR

DR. ALEŠ HRDLIČKA'S[1] recently publicized explanation that as one of the functions of the hair is to excrete melanin, graying of the hair is, therefore, a quantitative expression of the total amount of melanin to be excreted by the body, which in some way, not explained, depends on the metabolism. Thus, according to Dr. Hrdlička, graying is an automatic expression of the dying fires of metabolism, and no drug or chemical can be expected to have more than a temporary effect.

The color pattern of the hair is not only generally, but also somatically inherited. If this were not so we might be startled to find our leopard losing her spots and the tiger his stripes. Moreover, we would be at a complete loss to explain the white tips on black fox fur or the reverse on ermine.

That the coloring of the hair is functional in character and not automatically dependent on some generalized bodily change or growth is most clearly shown by the fact that certain animals, as the Arctic fox, change the entire color of the hair from winter to summer season, which is coupled with the moulting function; also shown in birds, like the ptarmigan. Moreover, in birds, and to some extent in mammals, the pattern of the hair coloring changes with sex activities, remarkably confirmable by experiment. In humans, both the hair coloring and its time-duration is a hereditary matter and independent of general health, virility or age.

Moreover, much evidence goes to show that melanin formation is a local matter. Commonly, for example, one finds the scalp hair white over the site of a former injury, although there is a good hair growth continuing. This is also true in other pigmented tissues, such as the skin. Pregnancy, lice, irritation, etc., cause marked localized pigmentary changes. On the other hand, as yet unknown factors cause marked patch withdrawal of pigmentation (Vitiligo) peculiarly striking in colored people. The Negro's hair grays with age; his skin doesn't, which fact I find impossible to coordinate with Dr. Hrdlička's idea of general melanin excretion.

While sex seems to play an important role in hair coloring, nevertheless, albinos breed quite freely, as laboratory rats and mice amply demonstrate.

That the matter is not a simple concomitant of growth or nutrition is well illustrated by the fact that the "bald" area rarely becomes markedly gray primarily, and often completes its own peculiar function without any graying whatever. The lateral margins of the scalp, on the other hand, most commonly gray first and they, on the contrary, rarely become bald.

All the above facts are but a small selection of the large number illustrating the same matter; namely, that while there are many outside controls reacting on the actual machinery of pigmentation, nevertheless, this is a separate entity. Moreover, each cell shows a quantitative difference in its reactivity to such controls so that hairs growing side by side may show very marked pigmentary contrasts. Such facts leave little doubt that the fiber pigmentation is a special function, and unless this be a solitary exception to the general rules of physiology, it is capable of being altered in a quantitative manner by pharmacological agents.

DR. OWEN S. GIBBS,
Director of Research
MEDICAL RESEARCH DIVISION,
PLOUGH, INC.,
MEMPHIS, TENNESSEE

SOME FACTORS AFFECTING APPLE SCALD DISEASE

THE scald disease of the apple is a storage disorder which causes tremendous wastage in storage wherever apples are stored the world around. The cause of this disease was found by workers in the U. S. Department of Agriculture to be accumulations of certain volatiles around the fruit in storage.[1] They devised a method of control in which these volatiles were absorbed by paper wraps impregnated with mineral oil.

Two years' results on the Rhode Island Greening variety indicate that coating the fruit with a wax emulsion (Brytene 489 AM) gave considerable promise in scald control. On prematurely picked apples, the wax treatment did not give as good control as the oiled paper treatment, but on pickings made at the normal time it gave as good control as oiled paper. Waxing has the advantage over the oiled paper treatment in that it keeps the fruit in a more green, crisp

5 Henry H. Goddard, "The Kallikak Family." Macmillan. 1912.

1 *Jour. Am. Med. Asn.*, March 14, 1942, p. 918.

1 C. Brooks, J. S. Cooley and D. F. Fisher, *Jour. Agr. Res.*, 18: 211–240, 1919.