vidual will throw some light on the nature of the mutation which produces *quercina*. Efforts are being made to secure self-pollinated nuts from each of these trees and in 1916 I hope to secure sesqui-hybrids from the cross *J. californica* mut. *quercina* ♀ × 08 H_1F_1P8c ♂. The completion of these breeding tests is especially desirable in view of the diverse behavior of certain of the original *quercina* trees. One of the type individuals breeds true, while a cotype individual produces both *quercina* and *californica* seedlings and another cotype individual produces nothing but *californica* seedlings.

The facts reported in this paper may be interpreted in part as meaning (1) that the mutation takes place in female flowers only and appears in the first generation after the mutation occurs but upon crossing with the species type it is completely recessive in the F_1 generation; (2) that the nature of the mutation is such that only certain genetic factors are affected without having the chromosome number disturbed.

Another walnut mutant has appeared in the form of a laciniate-leaved type of *Juglans regia*. Open pollinated nuts occasionally reproduce the new type. It is expected that self- and cross-pollinations will be made in 1916 and possibly cytological investigations.

[1] Babcock, E. B., Studies in Juglans I. Study of a New Form of *Juglans californica* Wats., *Univ. Cal. Pub. Agr. Sci.*, **2**, no. 1 (1913).

[2] Jepson, W. L., in *Bull. So. Cal. Acad. Sci.*, **7**, 23 (1908).

[3] Babcock, E. B., Studies in Juglans II. Further observations on a New Variety of *Juglans californica* Wats., and on Certain Supposed Walnut-Oak Hybrids, *Univ. Cal. Pub. Agr. Sci.*, **2**, no. 2 (1914).

HEREDITARY FRAGILITY OF BONE

By C. B. Davenport and H. S. Conard

CARNEGIE INSTITUTION OF WASHINGTON AND GRINNELL COLLEGE, IOWA

Presented to the Academy, August 30, 1915

While a weakness or brittleness of the long bones may arise from a number of pathological conditions affecting the bones, yet there has been recognized for 80 years a constitutional tendency to brittle bone that runs through families in such fashion as to demonstrate the presence of an hereditary factor. To it has been given the name fragilitas ossium or osteopsathyrosis.

In a classical case of osteopsathyrosis the femur is frequently found fractured at birth, but it mends quickly and smoothly. Repeatedly in life a slight knock causes a bone of the leg or arm to break; and sometimes before puberty the individual has suffered a score or more of

fractures. The tendency is often outgrown after puberty. Lovett and Nichols[1] have shown that in osteopsathyrosis the periosteal bone formation especially is imperfect, since the Haversian systems are not formed.

An examination of a large number of family histories shows clearly that heredity in osteopsathyrosis is typically direct—i.e., the factor that determines the irregular bone formation is a dominant one. The view that it is due to "some disturbance of metabolism of the mother" is disproved by the instance of twins, described by Schwarz and Bass,[2] one of whom showed osteopsathyrosis at birth while the other did not. The rare exceptions to the rule that a generation is not skipped are explained by the well known principle of imperfection of dominance or possibly to a lack of knowledge of an infantile condition of a parent.

An association of osteopsathyrosis with blue sclerotics has often been pointed out. The condition of blue sclerotics also appears to be a dominant trait. It is barely possible that the association is not a necessary one but on the other hand, there may be a linkage between these traits; pedigrees are not yet extensive and numerous enough to decide.

That osteopsathyrosis depends upon a single dominant factor is further indicated by the fact that the proportion of children in an affected fraternity that shows osteopsathyrosis is approximately half—actually 55%.

The presence of biotypes of osteopsathyrosis can be recognized. In some families the slightest pressure results in fracture; in other families the bones are fairly resistant. The commonest bone to break is the femur. But in some families the humerus seems especially weak and in still others the clavicle is frequently broken. Thus, associated with the imperfect bone development are special family traits that modify the result.

It appears, to summarize, that of a parent who was in early life osteopsathyrotic at least half of the children will be similarly affected. But if neither parent, though of affected stock, has shown the tendency then expectation is that none of the children will have brittle bones. Moreover, when a parent is affected it is possible that his children will show the tendency at about the same time of life, in the same bones and to the same degree as he himself showed it.

The full paper with numerous pedigree charts will appear in the Bulletin of the Eugenics Record Office.

[1] Lovett, R. W. and Nichols, E. H., *Brit. Med. J., London*, 1906, II, 915.
[2] Schwarz and Bass, *Medical Record*, 1912, p. 317.