number of children ever born from affected persons was compared with the number ever born to their unaffected sibs, for all Huntington's chorea pedigrees worked out by us. The average number of children from affected individuals was 6.07 ± 0.9, and from unaffected sibs 3.33 ± 0.5. The difference between these means is 2.74 ± 1.03 and is statistically significant. A similar difference was also present in the entire material from the literature. It was present, consistently, when the sexes of the affected and unaffected persons were treated separately, when surviving children were compared with those ever born, both for the Dight Institute material and the literature. Eight different comparisons all gave excesses for the affected. (Our explanation for these differences is not well enough established to include here.)

Let us return to the effect of social class upon the spread of a medically deleterious, but reproductively advantageous, gene such as Huntington's chorea. Brother A and 18 of his descendants have already had the disease in extreme stages, usually accompanied by commitment to a state institution. A number of others are in early and moderate stages of the disease. It is a simple matter to calculate the number of expected descendants of A, now living, who will eventually develop the disease, which has a late onset in this family. In addition to the 19 obvious cases, we expect 101 additional cases if those persons with the gene reach their age of onset. This would make a total of 120 cases, a remarkable rate of increase for the gene introduced by this one man. These patients will cause great trouble and expense before their deaths, which are usually the result of exhaustion. It is practically impossible to care for them at home during the last few years of the disease; consequently, the state assumes the burden.

Patterson, Bagchi, and Test (*4*) have indicated that it is possible to detect a potential case of Huntington's chorea before the reproductive age, by means of the electroencephalograph. The descendants of A offer excellent material for testing the usefulness of the electroencephalogram in detecting Huntington's chorea early enough so that a voluntary eugenics program could be undertaken. Even partial success would be of value, although the psychological approach to the potential victims of the disease would have to be skillful.

An extensive program of testing the descendants of A with the electroencephalograph has been begun. They are also submitting most graciously to the Wechsler-Bellevue intelligence test and to the Rorschach and the Minnesota Multiphasic Personality tests. It is hoped that these latter tests may give some hint as to personality differences that would account for the greater fecundity of persons who will later acquire the disease.

This study demonstrates the way in which the dominant gene for Huntington's chorea has spread, not only because it increases the fecundity of the affected person compared with unaffected sibs, but also because the social stigmata connected with the disease confine the close relatives to the lower social strata. Persons with little education (and usually lower social fitness) have higher than average reproductive fitness, as shown by the U. S. census. This situation favors the spread of the gene for Huntington's chorea.

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A Rich Source of γ-Carotene[1]

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In 1940, Emerson and Fox (*1*) reported the occurrence of a high concentration of γ-carotene in the male gametangia of the Phycomycete water-mold *Allomyces*. These workers concluded that the synthesis and storage of carotenoids are usually associated with the processes involved in the metabolism of reproduction. Later, Smits and Peterson (*2*) showed that the orange color of the expanded telial galls of the rust fungus (*Gymnosporangium juniperi-virginianae* Lk.) was due in part to carotenoids, of which 36% was the rare γ-isomer.

The heteroecious rust fungus (*Gymnosporangium juniperi-virginianae* Lk.) produces telial galls on the common juniper (*Juniperis virginiana* L.) and pycnia and aecia on the leaves, young twigs, and fruit of apple species and related genera. The telial spores produced by the telial galls in the spring infect the apple and produce two types of injury on the leaves, the epiphyllous pycnidial lesions and the hypophyllous aecia (Stevens, *3*).

An investigation of the carotenoid pigments of the pycnidial lesions of crab apple leaves caused by this rust fungus was undertaken in the spring of 1950. Owing to the unusual distribution of rainfall, there was an abundance of leaves that had reached full maturity before they had become infected. The leaves used in this work were harvested June 10, 1950, at the first appearance of a deterioration of the pigments as indicated by the darkening of the pycnia. Approximately 80% of the leaf surface was covered with these lesions. The leaves were placed in pint fruit jars, tightly sealed, and held at –17.4° C until analyzed June 17, 1950.

The pigments were extracted by blending 2 g of leaves in a Waring blendor for 5 min in a mixture of 60 ml ethanol and 150 ml of a mixture of petroleum hydrocarbons[2] consisting principally of hexane (bp 60°–70°) according to the Wall-Kelley (*4*) method. It was obvious from examination that the extraction

[1] Contribution No. 420, Department of Chemistry.
[2] Skellysolve B, Skelly Oil Co., Lyman, Okla.

was incomplete, and consequently the leaf residue, freed from solvent by suction, was again extracted by refluxing 30 min with 40 ml of a 10% solution of potassium hydroxide in methanol. The two extracts were then shaken with the petroleum naphtha to remove the carotenoids. The petroleum naphtha extracts were combined and further shaken with 90% methanol to extract free and esterified xanthophylls. After washing to remove traces of alcohol the petroleum naphtha extract was dried over anhydrous sodium sulphate.

The dried petroleum naphtha extract was chromatographed on a column consisting of equal parts by weight of Hyflo Super Cel and magnesia (No. 2641 Westvaco). Two prominent zones separated; the lower, less strongly adsorbed, was removed by eluting with a 4% solution (by vol) of acetone in the petroleum naphtha. The upper strongly adsorbed zone required an 8% solution of acetone in the petroleum naphtha for elution.

The pigment in the lower zone subsequently was shown by spectrum analysis to be β-carotene. The washings of the upper zone were evaporated to dryness *in vacuo* and taken up in the petroleum naphtha. This solution showed absorption maxima in the petroleum naphtha at 4,600 A, 4,900 A, and 5,340 A, with a minimum at 4,800 A. The absorption maxima and minimum of this pigment, together with its behavior on the adsorbent column, indicated that it was identical with γ-carotene.

The total carotene content of the infected leaves was 83.2 mg/100 g of dry material, of which 28.7 mg, or 34.5%, was the γ-carotene isomer. The uninfected leaves had 32.6 mg of β-carotene/100 g of dry material. No γ-carotene was detected in the normal leaves. Moisture content of the normal leaves was 62.1% when harvested, and infected leaves contained 68.7%.

γ-Carotene is relatively rare in plants, constituting about 0.1% of the total carotene extracted from ordinary sources (*5*). Small amounts have been found in apricots (*6*). MacKinney (*7*) has reported that the marsh dodder (*Cuscuta salina*) is a relatively rich source. A considerable concentration has been found in the fruit of *Pyracantha augustifolia* Schnied (*8*). However, the concentration of γ-carotene in leaves of the crab apple (*Malus ioensis* Britt) infected with the pycnidial lesions of the common rust fungus (*Gymnosporangium juniperi-virginianae* Lk.) is the highest that has come to the attention of the authors.

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Cavitomic Cotton

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An increasing and now large proportion of the spinnable good grades of cotton received by the mills is observed to contain an abundance of cellulose-destroying microorganisms. Deterioration of cotton during warehouse storage has also been noted. The full significance is not known, but the implications are apparent.

Within the limits of our experience, the effects produced by these microorganisms include an effectively shorter staple caused by weakening of the fibers, an

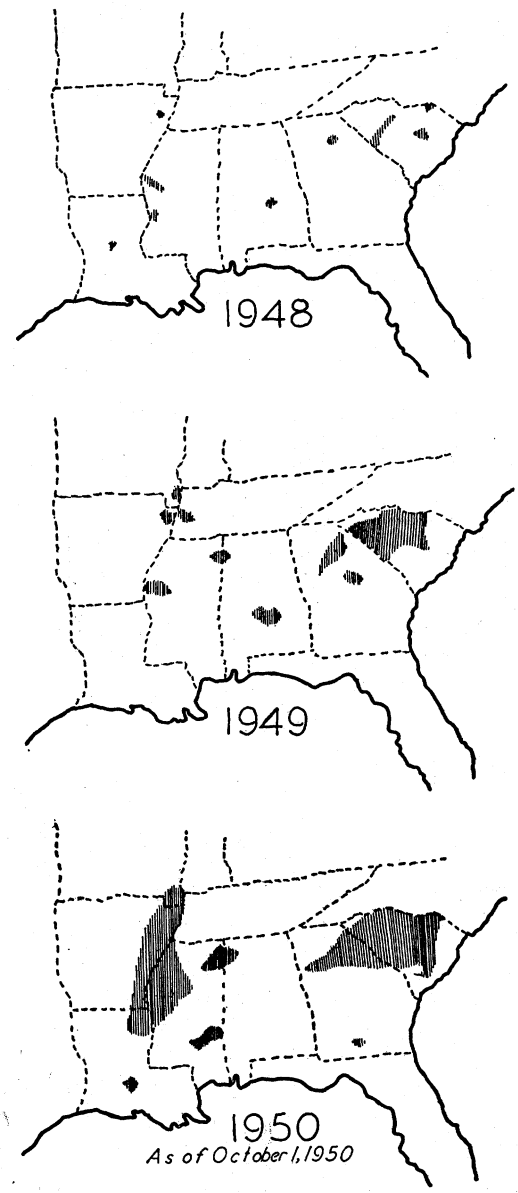

Fig. 1.