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Call for Manuscript Reviewers for *The American Biology Teacher*

If you are interested in becoming a manuscript reviewer for *ABT*, please send the information below to Bill Leonard at leonard@clemson.edu. We especially need K-12 teachers. Thank you in advance for your contribution to biology education.

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Who First Reported a Genetic Disease?

I enjoyed reading the article by Heron et al. (2010) in *ABT* last September. On page 424, the authors state that “Sickle cell disease was the first disease to be recognized as having a genetic basis, and thus linked phenotype to genotype (Weatherall & Clegg, 2001).” But according to Darnell et al. (1990: p. 9), the first relation between a genetic defect and a biochemical abnormality was reported in the study of the human disease alkaptonuria by Archibald Garrod in his book *Inborn Errors of Metabolism* (1909). However, according to Sutton (1980: p. 5) and Dunn (1965: p. 242), Garrod's studies on alkaptonuria, published in 1902, state clearly the hypothesis that alkaptonuria is inherited as a Mendelian recessive trait. In 1910, the American physician James B. Herrick presented his findings of the blood disease (later to be called “sickle cell anemia”) at the annual meeting of the Association of American Physicians (Volpe, 1971: p. 145). J. V. Neel provided evidence in 1949 that sickle cell disease is inherited as a simple Mendelian autosomal recessive character (Volpe, 1971: p. 146). However, Pauling et al. (1949) correctly identified the codominant nature of the alleles in heterozygous individuals with sickle cell trait (SCT) because both normal hemoglobin A and abnormal hemoglobin S are expressed in heterozygotes. “Very few individuals homozygous for the sickling gene survive to reproductive age” (Volpe, 1971: p. 147). If a “recessive lethal mutation” is defined as a mutation that results mainly in the premature death of homozygous individuals, sickle cell anemia is a recessive lethal condition. Sickle cell trait (heterozygous genotype) could be classified as a “conditional lethal” because the sickling gene typically causes death “only in situations of dehydration or when the oxygen saturation falls to about one-third the normal level” (Herron et al., 2010: p. 424).

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