Hemoglobin D

There are a number of hemoglobins termed **Hemoglobin D** (D-Los Angeles or D-Punjab, and D-Ibadan being the most common). These are all inherited variants of normal adult hemoglobin (hemoglobin A) and all have similar clinical implications. The genes for Hemoglobin D have the highest frequency among people of Asiatic Indian heritage. However, they are also found in people of European descent, especially British and Irish. Summarized below are the four most commonly encountered hemoglobin patterns that involve hemoglobin D.

**Hemoglobin D Trait** (phenotype: FAD in infants and AD in adults)
Hemoglobin D trait results when the gene for hemoglobin D is inherited from one parent and a hemoglobin A gene from the other. This carrier state does not usually result in health problems. For an infant identified with hemoglobin D trait on two newborn screening specimens, no further testing is indicated for the child. However, it is strongly recommended that the parents have hemoglobin testing to determine if they may be at risk for having subsequent children with hemoglobin sickle D disease, a clinically significant disease (described below), which is inherited in an autosomal recessive fashion.

**Homozygous Hemoglobin D** (phenotype: FDD in infants and DD in adults)
Homozygous hemoglobin D results when the gene for hemoglobin D is inherited from both parents. A mild hemolytic anemia develops in the first few months of life as the amount of fetal hemoglobin decreases and hemoglobin D increases. Hemoglobin levels are usually normal; however, there may be increased target cells and decreased osmotic fragility.

**Hemoglobin Sickle D Disease** (phenotype: FSD in infants and SD in adults)
Compound heterozygotes with hemoglobin sickle D disease result when the gene for hemoglobin D is inherited from one parent and the gene for hemoglobin S (commonly known as sickle cell) from the other. A mild to moderate hemolytic anemia develops in the first few months of life as the amount of fetal hemoglobin decreases and hemoglobin S and D increases. Although a form of sickle cell disease, most individuals with hemoglobin sickle D disease have fewer problems with infections and spleen involvement, fewer pain episodes and less organ damage than the other more common forms of sickle cell disease.

**Hemoglobin D/β Thalassemia** (phenotype: FDA or FD– in infants and DA or D– in adults)
Co-inheritance of the gene for hemoglobin D and beta (β) thalassemia, termed hemoglobin D/β thalassemia, has clinical manifestations ranging from mild to moderate, depending upon the degree of the thalassemia affecting the hemoglobin A gene. A mild hemolytic anemia usually develops in the first few months of life as the amount of fetal hemoglobin decreases and hemoglobin D increases. Splenomegaly and other complications can occur but in general it is considered a fairly benign condition. Also, because the red blood cell indices are abnormal in hemoglobin D/β thalassemia, iron deficiency, if suspected, may need to be assessed more directly through serum iron levels, iron binding capacity, and percent saturation.

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*Genetic counseling is advisable for families affected by these conditions to promote understanding of the significance for themselves and future offspring. A list of genetic counselors and hemoglobin consultants was included with this fact sheet (additional copies are available from our office).*

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