MALARIA:

HbS has low oxygen binding capacity, inhibiting malarial growth in red blood cells.

100 million people infected, 1 million die annually in Africa alone.
About 1,000 cases per year in the USA. Prevalent in southern Asia (most of India, southern China), central and south america.

More than 1/4 of all hospital admissions during the Civil and Spanish-American wars were for malaria. Serious problem in Pacific in 2nd WW, Korean war, Vietnamese wars.

Female *Anopheles* mosquito -> liver -> red blood cells -> lysis (chills and fever, paroxysms) and infection of new cells. Anemia, splenomegaly, hepatomegaly.

Notes (not necc. for lecture):

Ref: Lange/Harper's Biochem, Rodwell's Myoglob/Hemoglob article.

Myoglobin has a higher oxygen affinity and stores oxygen in muscle for periods of high exertion.

Dissolution of CO2 releases protons. Hb aids in buffering (unlike myoglobin) by absorbing two protons for every 4 oxygen molecules released. The CO2-generated acid thus helps force oxygen off the Hb. When oxygen binds in the lungs, it releases these protons, which helps force CO2 release to the gas phase. This reversible phenomenon is called the Bohr effect.

Partially oxygenated Hb is in the taut or T state; fully oxygenated, in the R or relaxed state. In the T state, there is a cavity between all 4 chains which can be occupied by 2,3-biphosphoglycerate, which increases during exercise. Thus, exercise and production of BPG tends to force oxygen off by stabilizing the T state.