Hemoglobin type	Composition	Notes
HbH	-Tetramer of Beta, **β4.**	-Alpha thalassemia intermedia -3 **α** genes deleted -mild-moderate symptoms -not fatal
Hb Barts	-Tetramer of Gamma, **γ4**.	-Alpha thalassemia major -hydrops fetalis -4 **α** genes deleted -fatal -stillbirth/early after-birth morbidity
HbE	-Defective beta globin which combines with alpha (α2, **βE).**	-Individuals with this point mutation make only 60% of the normal amount of **β globin** -common in southeast Asia **-**asymptomatic
HbS	-Point mutation in beta chain. -Sickled hemoglobin, via the valine, forms a hydrophobic protrusion (can clog into the pocket that is there even in normal Hb only when deoxygenated) -Anything that increases T state will worsen the sickling.	-mutant Hb is poorly soluble when deoxygenated -causes hemolytic anemia, less than 20 days rbc life span -irreversible sickling with repeated cycles, blocks capillaries, causing damage to organs via hypoxia
HbC	-point mutation in beta chain. -This hemoglobin is less soluble, so it crystallizes in RBCs, reducing their deformability. -Causes more water loss out of an RBC= higher Hb concentration.	-Mostly a minor issue -minor hemolytic anemia due to water loss (RBCs are rigid) -less deformability= reducing their ability to squeeze through capillary.
HbSC	-HbC from a parent, HbS from the other	-causes mild hemolytic anemia!
HbCS (constant spring)	-mutation in the termination of alpha2, causes longer mRNA (unstable) or unstable proteins. -Less quantity, less quality together	-if heterozygotes **(α α, α $α_{cs}$)** causes alpha thalassemia trait. -if coinherited with thalassemia **(_ _, α $α_{cs}$)**, causes alpha thalassemia intermedia. -common in southeast Asia and China.
Hb Hammersmith	-point mutation (most commonly in beta) that leads to formation of unstable hemoglobin.	-reduced hydrophobicity in heme pocket. -unstable heme-globin interactions. -causes cyanosis.

Hb Cowtown	-substitution of His146 that's responsible for the Bohr effect	-**INCREASED** oxygen affinity -more hemoglobin in **R** state -shifts the curve to the **LEFT**
Hb Kansas	-stabilization of the **T** state	-**DECREASED** oxygen affinity -more hemoglobin in **T** state -shifts the curve to the **RIGHT**
Hb Yakima	-stabilization of the **R** state	-**INCREASED** oxygen affinity -more hemoglobin in **R** state -shifts the curve to the **LEFT**
HbM – methemoglobin (in normal people it's available but only in very small amounts) (kept low via methemoglobin reductase enzyme). normal hemoglobin but with ferric iron.	-normally, oxyhemoglobin carries oxygen while in ferrous state, and during the release it's oxidized to ferric state, then reduced by methemoglobin reductase back to ferrous state. -methemoglobinemia causes: some mutant globins resist the reductase. or deficiency of this reductase. or drugs / drinking water with nitrates.	-normal enzyme uses **NADH** from **glycolysis** --- -TREATED BY methylene blue, it will get reduced to leukomethylene blue which will accept the electrons instead of the deficient enzyme. -uses **NADPH** from **PPP** -chocolate brown blood + lips
HbM Boston	-**Distal** histidine mutated to tyrosine; this tyrosine's oxygen causes oxidation of ferrous iron – Auto oxidation of iron.	-results in methemoglobinemia
HbM Iwate	-**Proximal** histidine mutated to tyrosine; this tyrosine's oxygen causes oxidation of ferrous iron – Auto oxidation of iron.	-results in methemoglobinemia