



Figure 1. Chemical Dynamics of NO Interactions with Hemoglobin. This perspective envisions Hb as a programmable chemical reactor in which NO chemistry is modulated, as illustrated, by ambient conditions of NO, oxygen, and redox potentials. Allosteric effectors would likewise modulate the chemistry through effects on oxygen-saturation, oxidation-states, and spin-states. NO signal input, entailing NO, NO donors, nitrites, thionitrites are processed—as directed by effector levels, absolute and relative concentrations of reagents, and reagent mixing—to provide a disposition of NO products on the protein appropriate for signal output. For hypoxic vasodilation, output appears to involve dispensing of vasodilatory activity through formation of S-nitrosylated RBC membrane proteins and perhaps low-molecular-weight thiols. In the case of high levels of NO, for example in sepsis, the adaptive chemistry works to brake NO release. The species indicated should be taken as exemplary, but underscore the role of minority species and micropopulations in this chemistry.