as it dissociates from maternal hemoglobin and binds to fetal hemoglobin. The only source of fetal oxygen is the mother, and maternal treatment for CO poisoning reduces fetal COHb levels (Greingor et al. 2001). Clearance of CO in the fetus would be dependent on the mother's oxygen intake. Children have the same type of hemoglobin as adults but are more susceptible than adults because they breathe a greater amount of air per body weight than adults.

The binding affinities of embryonic hemoglobin suggest that fetuses are more susceptible to CO intoxication when compared with adult hemoglobin. Embryonic hemoglobins (Gower I, Gower II, and Portland) are present until about 8-12 weeks of gestation. Fetal hemoglobin is expressed from about 5 weeks of gestation until 9 months after birth. Adult hemoglobin starts being produced between 3-6 months after birth (Orville 2008). Under physiologic conditions, the binding constants of fetal and adult hemoglobin to CO are 0.09 μM and 0.13 μM, respectively, meaning that the binding affinity for CO is higher for fetal hemoglobin than for adult hemoglobin (Di Cera et al. 1989). The rate constants for the binding of CO to the three embryonic hemoglobins are 3.0×10^{-6} M/s (Gower I), 2.0×10^{-6} M/s (Gower II), and 3.5×10^{-6} M/s (Portland) compared with 4.0×10^{-6} M/s for adult hemoglobin at pH 6.5 (Hofmann and Brittain 1996). No data were located that reported on whether embryos are more susceptible than fetuses. Data on the susceptibility of embryos to CO are mostly qualitative.

2. Children are at higher risk because they develop acute neurotoxic effects (e.g., headaches and nausea), long-lasting neurotoxic effects (e.g., memory deficits) and impaired ability to escape (e.g., syncopes) at lower COHb concentrations than adults (see Section 2.2.2.1). Children also have developing organs (brain and lungs), which may be affected differently than the developed organs of adults (ATSDR 2002). Children tend to be more susceptible than adults because they breathe a greater amount of air per body weight than adults.

3. People are at higher risk who have pre-existing diseases, either known or unknown, that already decrease the availability of oxygen to critical tissues; this group includes those who have coronary artery disease (see Sections 2.2.1 and 2.2.1.1), chronic obstructive lung disease, chronic anemia, and hemoglobinopathies, such as sickle cell anemia. For example, in sickle-cell disease, the average lifespan of red blood cells with abnormal hemoglobin is 12 days compared with an average of 120 days in healthy individuals with normal hemoglobin. "As a result, baseline COHb levels can be as high as 4%. Presumably, exogenous exposure to CO, in conjunction with higher endogenous CO levels, could result in critical levels of COHb. However, it is not known how ambient or near-ambient air levels of CO would affect individuals with these disorders" (EPA 2000; see also WHO 1999a). Due to physiologic adaptation in these subpopulations, they are not considered more susceptible than patients with coronary artery disease.

4. People at high altitude are at higher risk, especially those not living there long enough for physiologic adaptation. "It is important to distinguish between the long-term resident of high altitude and the newly arrived visitor from