state of Maryland during 1966-1971 showed COHb levels in the 40-79% range for 98% of lethal cases (Nelson 2006a). The Institute of Forensic Medicine in Oslo reported a study of COHb levels in 54 automobile exhaust victims. The mean fatal COHb level was 70%, and 40% was the minimum COHb level exhibited by less than 2% of the cases (Nelson 2006a). Another forensic study (Nelson et al. 2006) examining 2,241 fatalities between the years of 1976-1985 found that the mean COHb level of all the cases was 64.20% with an SD of 17.47. The data showed that 34% of victims had COHb levels of less than 60%. Of those who died in fires, 41% had COHb levels of less than 60% compared with 22% of the nonfire deaths.

The 40% COHb level is also supported by experimental studies performed in healthy human subjects. Studies by Chiodi et al. (1941), Henderson et al. (1921), and Haldane (1895) suggest that a COHb of about 34-56% does not cause lethal effects in healthy individuals. Further support comes from the studies by Kizakevich et al. (2000), Stewart et al. (1970), and Nielsen (1971) that reported headache as the only symptom when subjects were exposed to 20-33% COHb. Several case reports indicate that in patients with coronary artery disease, CO exposure can contribute to myocardial infarction. In the published cases of myocardial infarction, the following COHb values were measured after transport to the hospital: 52.2% (Marius-Nunez 1990), 30%, 22.8% (Atkins and Baker 1985), 21% (Ebisuno et al. 1986), and 15.6% (Grace and Platt 1981). A level of 40% COHb was used as the basis for AEGL-3 derivation. This point of departure is further supported by studies in animals reporting minimum lethal COHb levels in rats and mice of about 50-70% (Rose et al. 1970; E.I. du Pont de Nemours and Co. 1981).

Another uncertainty of the human reports used to support a lethality threshold level of 40% COHb was that they did not address whether the COHb measurement was derived from a peripheral site (e.g., femoral vein) or from central blood. This type of information is missing in many of the CO poisoning reports. Although it remains uncertain where the blood samples were taken, data from Levine et al. (2002) and Dalpe-Scott et al. (1995) ruled out significant postmortem changes in COHb levels that were demonstrated by similar heart blood to peripheral blood (H:P) ratios between central and peripheral blood.

Using the CFK model (Coburn et al. 1965; Peterson and Stewart 1975), exposure concentrations were calculated that would result in a COHb of 40% at the end of exposure periods for 10 and 30 min as well as for 1, 4 and 8 h (see Appendix B).

AEGL-3 values calculated with an intraspecies uncertainty factor of 10 would lead to an approximate 4% COHb level in exposed healthy adults. The values would be conservative and more protective of susceptible populations, including the developing fetus, children, and those with compromised circulatory systems, especially at longer exposure durations. However, 4% COHb is the approximate background level in smokers (WHO 1999a). At that level, healthy individuals have decreases in work capacity and decrements of neurobehavioral functions (WHO 1999a; EPA 2000; Hazucha 2000). Furthermore, workers com-