

## SUMMARY

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The nitrogen oxides discussed in this document are nitric oxide (NO) and nitrogen dioxide (NO<sub>2</sub>). In combustion processes, NO is primarily formed and subsequently oxidized to NO<sub>2</sub> in the atmosphere. Traffic is the major source to human outdoor exposure to NO and NO<sub>2</sub> in urban areas. In homes with gas-fueled stoves or water heaters, the concentration of NO<sub>2</sub> is generally higher indoors than outdoors.

Upon inhalation, NO<sub>2</sub> penetrates deep into the lung. In animal experiments, NO<sub>2</sub> causes biochemical and morphological changes in lung tissue, affects host defense mechanisms, and causes decreased pulmonary function. The lowest observed effect level after prolonged exposure is 560 µg·m<sup>-3</sup>.

In short-term controlled studies on humans, NO<sub>2</sub> causes bronchoconstriction and an increase in bronchial responsiveness. A meta-analysis indicates a lowest observed effect level of 200 µg·m<sup>-3</sup> for increased bronchial responsiveness in asthmatics.

Epidemiologic studies on children living in homes with gas stoves suggest an increased risk of lower respiratory illness at average indoor NO<sub>2</sub> concentrations of about 40-80 µg·m<sup>-3</sup>, with short-term peaks exceeding 1000 µg·m<sup>-3</sup>. The studies focusing on outdoor exposure provide some evidence of increased respiratory disease rates for children and nonsmoking adults living in areas with long-term average NO<sub>2</sub> concentrations of 30-100 µg·m<sup>-3</sup>; however, the specific role of NO<sub>2</sub> is not clear.

Based on controlled studies on humans, a 1-h guideline value of 100 µg·m<sup>-3</sup> for NO<sub>2</sub> in ambient air (eg, as the 99th percentile) is recommended. This value would correspond to a long-term average (half-year mean) of about 40 µg·m<sup>-3</sup>. The epidemiologic evidence is not considered sufficient for a long-term guideline for NO<sub>2</sub>.

NO is formed endogenously for purposes such as signaling in the nervous system, the mediation of vasodilation, and the mediation of cytotoxicity in macrophages. After the inhalation of high concentrations of NO, lung vasodilator effects occur. No health-based guideline value for NO in ambient air is suggested.