HEALTH EFFECTS ASSOCIATED WITH OZONE AND NITROGEN DIOXIDE EXPOSURE

DAVID J. MCKEE and ROSALINA M. RODRIGUEZ

Ambient Standards Branch (MD-12), Air Quality Management Division, Office of Air Quality Planning And Standards, Office of Air and Radiation, U.S. Environmental Protection Agency, Research Triangle Park, NC 27711, U.S.A.

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Abstract. Of the oxidants and nitrogenous compounds found in ambient air, O3 and NO2 are among those most thoroughly investigated. Large health data bases have been compiled in air quality criteria documents, which serve as the basis for review of primary (health) national ambient air quality standards (NAAQS) for each pollutant. When comparisons can be made, O3 is generally more toxic than other photochemical oxidant species at or near ambient levels, although NO2 does not appear to be of more concern with respect to carcinogenesis. Public health effects of concern for O3 range from acute symptoms (e.g., cough) and decreased lung function to persistent functional changes and permanent scarring of lung tissue, a possible precursor to chronic lung disease. Impairment of immune defenses and increased susceptibility to lung infection have also been associated with O3 exposure. Populations at greatest risk include exercising healthy persons, children, and those with preexisting lung disease. Nitrogen dioxide exhibits similar health effects but at higher concentrations. These effects include acute symptoms and lung function impairment, increased susceptibility to acute respiratory infection, and possibly conditions leading to chronic lung disease. Populations potentially at risk include children, asthmatics, and individuals who exercise in the presence of NO2. Health effects data for O3 and NO2 are under consideration in the current review of both primary NAAQS.

1. Introduction

Ozone and NO2 are two of the most ubiquitous oxidants found in the ambient air and exhibit documented adverse health and welfare effects. For these reasons both have been designated criteria pollutants, and primary (health) and secondary (welfare) national ambient air quality standards (NAAQS) have been promulgated for each pollutant. Identical primary and secondary NAAQS for O3 were originally set in 1971 at 0.08 ppm total oxidants (1-hr average) and revised in 1979 to be 0.12 ppm O3 (1-hr average, daily maximum) not to be exceeded more than one time per year. For NO2 both primary and secondary NAAQS are also identical but were set in 1971 at 0.053 ppm NO2 as an annual average of all hours and retained in 1985. NAAQS for O3 and for NO2 are currently under review. While recognizing that welfare effects associated with these oxidants have important indirect effects on public health, this paper will focus exclusively on the direct public health effects of O3 and NO2 which form the basis for primary NAAQS.

2. Public Health Effects of Ozone

Ozone has probably been studied for its acute health effects as much or more
than any other air pollutant. When comparisons have been possible, O₃ is more
toxic than other photochemical oxidants at ambient or near-ambient concentrations.
Because over $110 \times 10^6$ people in the U.S. live in areas which have been designated
non-attainment for the O₃ NAAQS, O₃ is generally recognized as a major envi-
ronmental problem in the U.S.

Public health effects of concern for O₃ include lung function impairment, symptoms
(e.g. cough), aggravation of asthma, pulmonary inflammation, increased suscep-
tibility to respiratory infection, and lung structure damage (e.g. centriacinar lesions).
Controlled human exposure, epidemiology, and field studies provide most of the
significant human data on functional impairment, symptoms, inflammation and
respiratory infection, effects which are generally associated with acute (1 to 3 hr)
and prolonged (6 to 8 hr) O₃ exposures. Level of exercise has been identified as
a major determinant of acute and prolonged exposure effects. In contrast evidence
of chronic (months to years) O₃ exposure effects has come almost exclusively from
animal toxicology studies due to obvious limitations of exposing human subjects
for such extended periods under controlled conditions. Because chronic exposures
to O₃ can produce permanent functional and structural changes in animals, it is
important to extrapolate these findings to human health effects. This process of
extrapolation is an important step in the chronic risk assessment for O₃ which
is being conducted by USEPA and will be discussed later in this paper.

PULMONARY FUNCTION AND SYMPTOMS

Pulmonary function changes are represented by decrements in forced expiratory
volume (FEVI) and symptoms are the best documented acute health effects of O₃,
although the first effect of O₃ is to decrease the forced vital capacity (FVC), i.e.
restrict the inspiratory capacity (Hazucha et al., 1989). Numerous studies, as
summarized in Table I, provide indisputable evidence that acute O₃ exposures produce
transient pulmonary function decrements which results in rapid, shallow breathing
and decreased ability to breathe deeply.

The following conclusions can be drawn from these studies, which are discussed
in more detail in the Ozone Criteria Document (USEPA, 1986), Ozone Criteria
Document Supplement (USEPA, 1988), Ozone Staff Paper (USEPA, 1989) and the
NAPAP States of Science Document (NAPAP, 1990). (1) Analysis of FEVI data
shows a consistent concentration-dependent reduction in lung function, which is
further exacerbated by increased respiratory ventilation associated with exercise.
(2) The lowest O₃ concentration reported to produce statistically significant group
mean FEVI decrements in heavily exercising persons after 1 hr of exposure is 0.12
ppm. (3) The relationship between exercise and rate of FEVI decline with increasing
O₃ exposure is graphically displayed in Figure 1 (Hazucha, 1987). (4) There is a
wide range of individual responsiveness among healthy adults, with some individuals
being significantly more responsive than the group mean; however, there is no
clear evidence that smokers, elderly, young people, or individuals with asthma,
allergic rhinitis, or chronic respiratory disease are substantially more sensitive to