

relate to uncertainties in the relevant action spectra and BAFs, as well as in factors related to characterizing the severity of the different types of NMSC. Based on the record information, for the other effects, the role of UV-B radiation is less well understood (e.g., as to relevant action spectra, BAFs, the nature of exposures of concern), although cumulative exposure to UV-B radiation is thought to play a causal role. These characterizations are derived from the large body of epidemiologic and toxicologic evidence that served as the basis for the reference document by EPA (1987).

The record includes a quantitative assessment conducted by EPA (1987, App. E) of the health risks associated with changes in exposure to UV-B radiation attributable to changes in the stratospheric O₃ layer. This assessment models the relationship between wide-scale changes in global/regional levels of stratospheric O₃, resulting from emissions of O₃ depleting substances with long-atmospheric lifetimes, and changes in UV-B radiation flux as a function of latitude for three broad regions across the United States.⁶⁷ As discussed above (section II.B.2), because changes in the stratospheric O₃ layer are relatively uniform across broad regions, varying across the U.S. primarily with latitude, information on localized spatial and temporal patterns of exposure-related variables (e.g., changes in ground-level O₃, meteorological conditions, human activity patterns) are not relevant in producing credible estimates of risk associated with changes in stratospheric O₃. This is in sharp contrast to the nature of the information necessary to produce credible estimates of risk associated with changes in exposures to UV-B radiation projected to result from changes in ground-level O₃ that would be associated with attainment of alternative 8-hour standards for O₃.

An evaluation of the available analyses that have produced estimates of UV-B radiation-related health risks associated with changes in ground-level O₃ and the comments received on them, in section II.B.3 above, identifies major limitations in available information that resulted in the need for the analyses to incorporate broad and unsupportable assumptions. These limitations are particularly important with regard to

information on spatial and temporal patterns of changes in ground-level O₃ (across the entire year and extending vertically up through the tropospheric mixing layer) likely to result from various future emission control strategies, relevant meteorological conditions and atmospheric chemistry leading to a cascade of broader indirect effects, and human demographic and activity patterns (e.g., the degree of shading within outdoor microenvironments, and the prevalence of sun-seeking and sun-avoidance behaviors among sensitive groups) likely to affect UV-B radiation-related exposures of concern. For the reasons discussed above, these limitations are judged to be of central importance in any such analysis. Thus, in light of such limitations, and after careful consideration of the comments received, EPA continues to agree with internal and external reviewers, and some commenters, in concluding that the available scientific and technical information would not permit credible quantitative estimates of these potential beneficial effects.⁶⁸ Thus, EPA concludes that available analyses based on such limited information cannot serve as credible estimates of potential beneficial effects associated with the presence of ground-level O₃ due to man-made emissions of O₃-forming substances.

Beyond the specific technical comments discussed above in section II.B, several commenters expressed the general view that EPA had inappropriately applied a "double standard" in its evaluation of the scientific evidence because it failed to evaluate the protective shielding effects of ground-level O₃ using the same criteria by which it evaluated the adverse respiratory effects. This viewpoint was specifically expressed by one commenter in stating that "EPA has accepted, often without reservation, scientific evidence purporting to establish the adverse effects of ground-level ozone on respiratory effects. At the same time it has often discounted proffered scientific evidence of the potential benefits of ground-level ozone in screening harmful UV-B radiation."

⁶⁸ This conclusion was also reached by the Health and Ecological Effects Subcommittee of the Advisory Council on Clean Air Compliance Analysis, a part of EPA's Science Advisory Board, in conjunction with their review of "The Benefits and Costs of the Clean Air Act 1990 to 2010" (EPA, 1999b), noting that the relevant information "was very weak and more information is required" (EPA, 1999a). As one commenter noted, this SAB Council has more recently recommended that in EPA's next periodic prospective analysis of the Act, the Agency's analysis address this issue (Advisory Council for Clean Air Compliance Analysis, 2001).

(Docket No. A-95-58, VI-C-8, pg. 28) As discussed below, EPA strongly rejects both aspects of this comment. Other commenters expressed the opposite view, finding EPA's approach to be evenhanded in its evaluation of the scientific evidence for potential beneficial and adverse effects, with one commenter noting that EPA "has never concluded that any allegation or "evidence" [of adverse effects], regardless of its preliminary or speculative nature or degree of uncertainty, must be factored into NAAQS decisionmaking." (Docket No. A-95-58, VI-C-6, pg. 2)

First, EPA believes that there is ample evidence in the record of the 1997 review of the O₃ NAAQS to invalidate the notion that the Agency uncritically accepts scientific evidence of adverse respiratory effects of ground-level ozone. For example, in considering evidence of adverse respiratory-related effects such as increases in bronchial responsiveness, decrements in alveolar macrophage function, and O₃-induced markers of inflammation and cell damage (as discussed in the 1996 proposed rule, 61 FR 65720-21), EPA judged that there was not sufficient information on dose-response relationships to develop quantitative risk estimates for these acute effects, even in light of the availability of peer-reviewed human exposure studies demonstrating indicators of these effects in humans at quantified exposure levels over quantified time periods (1997 final rule, 62 FR 38868). Similarly, EPA limited the scope of its quantitative risk assessment of acute respiratory-related hospital admissions of asthmatics to just one city (New York City), despite the availability of peer-reviewed studies showing increased admissions in other cities, because it judged that there was not adequate city-specific concentration-response information from epidemiological studies in other cities, that applying the New York City concentration-response information to other cities would introduce too much uncertainty into any such quantitative estimates, or that adequate ambient O₃ monitoring data were not available for other study areas to produce credible estimates of this risk for those cities (EPA, 1996b, pp. 111-112). Further, EPA did not rely on quantitative estimates of other adverse effects that have been related to hospital admissions of asthmatics in published documents submitted by commenters on the 1996 proposed rule (e.g., the "pyramid of effects" including hospital admissions among the general population, visits to emergency departments and doctors'

⁶⁷ Since the EPA's 1987 risk assessment on stratospheric ozone depletion, numerous changes have been made to the model to reflect the commitments made since 1987 by the United States, under amendments to the Montreal Protocol, for reductions in production of various ozone depleting chemicals and to incorporate more accurately the latest scientific information.