

draft, unpublished or preliminary analyses to be inappropriate for inclusion in air quality criteria, and concludes that supplementing the 1996 O₃ criteria is not warranted.

(5) As discussed in more detail in section II.B.2, EPA also determined that it was not in a position to supplement the air quality criteria by developing its own more extensive analysis because information essential to the development of such an analysis (*e.g.*, behavioral patterns related to potential UV-B radiation exposure) is not available at this time.

(6) The EPA has appropriately consulted CASAC by providing for its review and comment the proposed response, as well as the key documents from the record upon which EPA's response is based.²⁰ The CASAC has expressed no concern with this procedure nor indicated that any further CASAC involvement was necessary or appropriate. Indeed, only one member of CASAC chose to comment at all, and that member likewise expressed no concern with the method by which EPA consulted with CASAC on the response to the remand. Finally, the commenters have not provided any reason to believe that additional review by CASAC would have affected the outcome of this action in any way.

In view of the above factors, in particular the quality and type of analyses relied on by commenters and the fact that CASAC had the opportunity to review those analyses as well as other information in the record, EPA believes its approach to this response represents a reasonable exercise of its discretion to decide when to supplement the review process and fulfills the Agency's responsibilities under the Act. The EPA's response fully complies with the direction of the Court that EPA determine ozone's net adverse effect on public health and not "disregard the studies" upon which the petitioners primarily relied in their challenge. *ATA I*, 175 F.3d at 1053. Nothing in the Court's remand purports to require EPA to reopen the air quality criteria, or indeed the entire review process, before concluding this aspect of the 1997 review. For the reasons

discussed above, EPA also believes it would be inappropriate to do so.

Accordingly, the EPA concludes that any further extension of this review, through reopening the rulemaking record or review process, would represent an unwarranted delay in completing this review cycle, which began in 1992 and originally concluded in 1997. Any further extension of this review would also delay Agency and State actions to implement the 8-hour O₃ NAAQS, which EPA believes would be inappropriate and contrary to the purpose of the Act, in that it would impede the important public health protections afforded by the 8-hour ozone NAAQS.

A. Direct Adverse Health Effects From Breathing O₃ in the Ambient Air

This section briefly summarizes information on the direct adverse health effects from breathing O₃ in the ambient air, information as to when those effects become adverse to individuals, and insights gained from human exposure and risk assessments intended to provide a broader perspective for judgments about protecting public health from the risks associated with direct O₃ inhalation exposures.²¹

1. Health Effects Associated With O₃ Inhalation Exposures

Based on information from human clinical, epidemiological, and animal toxicological studies, an array of health effects has been attributed to short-term (1 to 3 hours), prolonged (6 to 8 hours), and long-term (months to years) exposures to O₃. Long-established acute health effects²² induced by short-term exposures to O₃, generally while individuals were engaged in heavy exertion, include transient pulmonary function responses, transient respiratory symptoms, and effects on exercise performance.²³ The 1997 review included substantial new information on similar effects associated with prolonged exposures at concentrations as low as 0.08 ppm and at moderate levels of exertion. Other health effects associated with short-term or prolonged O₃ exposures include increased airway responsiveness, susceptibility to respiratory infection, increased hospital

admissions and emergency room visits, and transient pulmonary inflammation. The 1997 review also included new information on chronic health effects²⁴ associated with long-term exposures. This array of effects is briefly summarized below, followed by considerations as to when these physiological effects could become medically significant such that they should be regarded as adverse to the health of individuals experiencing them.

a. Effects of Short-term and Prolonged O₃ Exposures

(i) Pulmonary function responses. Transient reductions in pulmonary function have been observed in healthy individuals and those with impaired respiratory systems (*e.g.*, asthmatic individuals) as a result of both short-term and prolonged exposures to O₃. The strongest and most quantifiable exposure-response information on such responses has come from controlled human exposure studies, which clearly show that reductions in lung function are enhanced by increased levels of activity involving exertion and by increased O₃ concentrations. Numerous such studies of exercising adults have demonstrated decrements in lung function both for exposures of 1–3 hours at ≥ 0.12 ppm O₃ and for exposures of 6.6 hours at ≥ 0.08 ppm O₃, providing conclusive evidence that O₃ levels commonly monitored in the ambient air induce lung function decrements in exercising adults. Further, numerous summer camp studies provide an extensive and reliable data base on comparable lung function responses to ambient O₃ and other pollutants in children and adolescents. The extent of pulmonary function decrements varies considerably among individuals, pulmonary function generally tends to return to baseline levels shortly after short-term exposure, and effects are typically attenuated upon repeated short-term exposures over several days.

(ii) Respiratory symptoms and effects on exercise performance. Various transient respiratory symptoms, including cough, throat irritation, chest pain on deep inspiration, and shortness of breath, have been induced by O₃ exposures of both healthy individuals and those with impaired respiratory systems. Increasing O₃ exposure durations and levels have been shown to elicit increasingly more severe symptoms that persist for longer periods

²⁰ The EPA's request for comments, together with copies of the proposed response and key documents, was transmitted to CASAC in a letter to Dr. Philip Hopke from Dr. Karen Martin, January 14, 2002, which is available in the docket. The EPA had previously provided an earlier draft of the proposed response, together with copies of key documents, to CASAC members in January 2001, ten months before the proposed response was published. See letter to Dr. Philip Hopke from Dr. Karen Martin, January 22, 2001 (available in the docket).

²¹ See the 1996 proposal and 1997 final rule for more complete summaries and the Criteria Document and Staff Paper for more detailed discussion.

²² "Acute" health effects of O₃ are defined as those effects induced by short-term and prolonged exposures to O₃. Examples of these effects are functional, symptomatic, biochemical, and physiologic changes.

²³ The 1-hour O₃ primary NAAQS set in 1979 was generally based on these acute effects associated with heavy exercise and short-term exposures.

²⁴ "Chronic" health effects of O₃ are defined as those effects induced by long-term exposures to O₃. Examples of these effects are structural damage to lung tissue and accelerated decline in baseline lung function.