Eastman Chemical Co. and Hoechst Celanese stated that the deletion of acetone will ''improve EPA's TRI program as well as conserve EPA and industry resources.'' Further, Outboard Marine Corp., Hoechst Celanese, and the Savannah River Pulp and Paper Corp. stated that the removal of acetone from the list of EPCRA section 313 toxic chemicals will reduce, in part, the administrative burden on facilities.

As described in the economic analysis, EPA agrees that the deletion of acetone will result in a resource savings by EPA and industry. In addition, EPA agrees that, as a result of this action, there will be a decrease in the administrative burden on facilities who have previously been required to report for acetone under EPCRA section 313.

A number of the commenters who supported the deletion stated that acetone is a substitute for more hazardous air pollutants, and that removing acetone from the list will encourage facilities to use acetone rather than these more hazardous chemicals. Specifically, Eastman Chemical Co. and Hoechst Celanese commented that the proposed rule does not address any of the environmental benefits associated with deleting acetone from the section 313 list. These two commenters pointed to the benefits derived from the use of acetone as a substitute for other regulated chemicals.

Although there might be environmental benefits from using acetone rather than some other chemicals, this has no impact on whether acetone meets the listing criteria of EPCRA section 313(d)(2). EPA agrees that, to the extent that the substances being substituted by acetone are more hazardous to human health or the environment than acetone, such substitution would be beneficial.

These two commenters further brought up several technical points, which they felt should have been included in the proposal. Specifically, they believe that a description of drinking water studies which have been conducted with acetone, as well as information on the recently revised oral reference dose (RfD) for acetone, would be a useful addition to the preamble to this final rule. EPA acknowledges that the drinking water studies have been conducted, but does not feel that a description of them is warranted. These studies support the decision to delist acetone. EPA also acknowledges that the RfD has recently been revised. At the time of publication of the proposed rule, the RfD was 0.1 milligram per kilogram per day (mg/kg/day). EPA has revised this RfD to 0.9 mg/kg/day. This higher value reflects a slightly lower toxicity and, as stated above, supports the delisting decision.

A number of the commenters that oppose the delisting stated that there are substantial data to support a concern for health effects from acetone, and that EPA's review of evidence of toxicity for acetone must address the serious concerns raised by the Agency for Toxic Substances and Disease Registry (ATSDR) in its *Draft Toxicological Profile for Acetone*. In addition, as some commenters have pointed out, there are insufficient data to assess the toxicity of acetone.

As reviewed by the ATSDR, there has been considerable research on the health effects of acetone. However, most of this research has involved acute or subchronic exposure to relatively moderate and high levels of acetone. There is a lack of information with which to firmly characterize the critical effects of low-level exposure to acetone. Under EPCRA section 313, a lack of evidence cannot be used as a basis for listing a chemical. The known toxicity levels for acetone fall in the range which can be considered to be moderately low to low, and the decision must be based on the weight-of-the-evidence available.

EPA has reviewed the ATSDR draft profile as well as other relevant materials and has concluded that there is not sufficient evidence of toxicity to retain acetone on the EPCRA section 313 list. According to the ATSDR, based on a lowest observed adverse effect level (LOAEL) of 1,250 parts per million (ppm) for (transient) neurological effects over a 6-week period, intermediate and chronic inhalation Minimal Risk Levels (MRLs) of 13 ppm were calculated. Furthermore, the ATSDR indicates that levels of acetone which are normally found in outdoor air are generally significantly lower than this, at less than 8 parts per billion (ppb), and also generally lower than the air concentrations of acetone inside homes. At this time, there is insufficient evidence regarding chronic or subchronic exposure to such low levels of acetone to warrant listing (Ref. 1).

Several commenters recommended that EPA require industry to fully test acetone for toxicity under the criteria of section 4 of the Toxic Substances Control Act (TSCA), stating that testing should be performed before acetone is removed from the public's right-to-know. Other commenters, noting that EPA is currently negotiating with industrial users of acetone for neurotoxicity testing of the chemical, claimed that the proposal for delisting is ill-timed and inappropriate.

At this time, the Agency has already entered into an Enforceable Consent Agreement with industry, requiring subchronic testing of acetone for neurotoxicity. At concentrations to which workers may be exposed in the workplace, which are much higher than those in outdoor air, central nervous system (CNS) effects such as narcosis, headache, and changes in operant behavior do appear to be relevant concerns indicative of neurotoxicity. However, the criteria for requiring neurotoxicity testing under TSCA section 4 and the criteria for inclusion in section 313 of EPCRA are very different. At this point in time, the weight-of-the-evidence is not sufficient to show that acetone meets the EPCRA section 313(d)(2) criteria for listing. EPA cannot deny a petition under EPCRA section 313 based on the fact that testing is going to be performed to fill data gaps.

A number of commenters stated that EPA should consider the synergistic effects of acetone together with other chemicals and stated that exposure to acetone is well known to increase the toxicity of many other chemicals. Commenters stated that the increased toxicity of other compounds in combination with exposure to acetone, as detailed in the ATSDR draft profile, justifies maintaining the EPCRA section 313 listing of acetone.

The ATSDR draft profile does provide a detailed review of the interaction of acetone and other chemicals. This report indicates that acetone may alter the effect of other chemicals by either increasing, decreasing, having a mixed effect on or having no effect on their toxicity. For example, carbon tetrachloride, halogenated alkanes, ethanol, and some ketones were more toxic when co-administered with acetone. However, acetone had mixed effects on the toxicity of other chemicals (dichlorobenzene, chlorinated alkanes, possibly halogenated alkanes, nitrosoamine, and acetonitrile) either at varying doses or for different toxicity endpoints. Furthermore, acetone had no reported effect on styrene or methyl ethyl ketone, and actually reduced the toxicities of acetaminophen and semicarbazide (Ref. 1).

As with the toxicity of acetone alone, the doses of acetone required for these interactive effects far exceed the concentrations of acetone which are found in outdoor air. For example, the lowest doses for acetone potentiation of toxicity reported by the ATSDR were found with carbon tetrachloride. Liver toxicity of carbon tetrachloride was shown to be potentiated by co-administration of acetone. However, non-effective doses of acetone were as high as 78 milligrams/kilogram (mg/kg)